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# Trigeminal neurons detect cellphone radiation: Thermal or nonthermal is not the question

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## ABSTRACT

Cellphone electromagnetic radiation produces temperature alterations in facial skin. We hypothesized that the radiation-induced heat was transduced by warmth-sensing trigeminal neurons, as evidenced by changes in cognitive processing of the afferent signals. Ten human volunteers were exposed on the right side of the face to 1 GHz radiation in the absence of acoustic, tactile, and low-frequency electromagnetic stimuli produced by cellphones. Cognitive processing manifested in the electroencephalogram (EEG) was quantitated by analysis of brain recurrence (a nonlinear technique). The theoretical temperature sensitivity of warmth-sensing neurons was estimated by comparing changes in membrane voltage expected as a result of heat transduction with membrane–voltage variance caused by thermal noise. Each participant underwent sixty 12-s trials. The recurrence variable $r$ ("percent recurrence") was computed second by second for the $\Delta$ band of EEGs from two bilaterally symmetric derivations (decussated and nondecussated). Percent recurrence during radiation exposure (first 4 s of each trial) was reduced in the decussated afferent signal compared with the control (last four seconds of each trial); mean difference, $r = 1.1 \pm 0.5\%$, $p < 0.005$. Mean relative $\Delta$ power did not differ between the exposed and control intervals, as expected. Trigeminal neurons were capable of detecting temperature changes far below skin temperature increases caused by cellphone radiation. Simulated cellphone radiation affected brain electrical activity associated with nonlinear cognitive processing of radiation-induced thermal afferent signals. Radiation standards for cellphones based on a thermal/nonthermal binary distinction do not prevent neurophysiological consequences of cellphone radiation.

## Introduction

Cutaneous warmth-sensing neurons in the trigeminal nerve respond to small changes in temperature above the local baseline level (Hensel, 1973). Electromagnetic radiation from cellphones interacts with facial skin to produce alterations in the baseline temperature (Bernardi et al., 2000, 2001; Hirata et al., 2003; Ibrahiem et al., 2005; Van Leeuwen et al., 1999; Wainright, 2000; Wang and Fujiwara, 1999). Heat production is the only conclusively established biophysical process by which cellphone radiation can interact with the body. Sensory transduction of radiation-induced heat as evidenced by detection of resulting afferent signals in the brain has not been reported at radiation levels emitted by typical cellphones.

Analysis of brain recurrence (ABR) is a method for quantifying changes in brain electrical activity (Frilot II et al., 2014). Employing ABR, subliminal electromagnetic fields (EMFs)—strengths below those that produced conscious awareness—triggered evoked potentials in humans and animals (Carrubba et al., 2007; Frilot II et al., 2009; Marino et al., 2003a), and increased glucose metabolism in the rat hindbrain (Frilot II et al., 2009; Frilot II et al., 2011). Sensory transduction of both low- and high-frequency EMFs occurred in the head (Marino et al., 2003a; Marino et al., 2003b), possibly mediated by a glutamate-dependent pathway (Frilot II et al., 2014).

Detection of low-frequency EMFs can be explained by their ability to directly exert forces on the gates of ion channels (Kolomytkin et al., 2007; Marino et al., 2009), but that process cannot explain transduction at the high frequencies characteristic of cellphone radiation. In this study, we considered the possibility that cellphone radiation is transduced indirectly, by warmth-sensing neurons in the trigeminal nerve, resulting in altered brain electrical activity due to cognitive processing of the afferent signal. The hypothesis was tested and verified in human volunteers by

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employing ABR to quantitate brain electrical activity in the presence and absence of electromagnetic waves that mimicked cellphone radiation.

## Methods

### Subjects

The participants were 10 clinically normal adults (5 male, 5 female), aged 21–74 years (mean 45). They were chosen randomly, informed of the goal, methods, and general design of the investigation, but not told exactly when or for how long during the experimental session the radiation would be applied. Written informed consent was obtained from each participant prior to entering the study. The institutional review board for human research approved all experimental procedures.

### Exposure system

A 1 GHz continuous-wave signal (a characteristic cellphone frequency) (Signal Forge Model 1000, Austin, TX) was amplified (Empower, Inglewood, CA) and fed to an electrically controlled timing switch that sent the signal to a patch antenna (Schmid et al., 2007) located 10 cm from the participant's right cheek or to a floor-level dummy antenna located 5 m away from the participant (Figure 1). The signal energized the patch antenna for 4 s and the dummy antenna for 8 s in successive 12-s trials. During the trials, the participant sat in a comfortable chair flanked by radiation-absorbing material, which was employed to reduce the possibility of radiation reflections. There were no equipment-related visual or auditory stimuli perceptible by the participants, as assessed by interviews after completion of the exposure sessions. The unperturbed electric field measured at the location of the participant's cheek was 60 V/m (Extech, Nashua, NH). To provide an objective basis for follow-up studies, the power density of the incident radiation, which was the independent variable in the study, was characterized empirically (direct measurement) rather than by employing model-dependent dosimetry parameters such as the specific absorption rate (Hirata et al., 2008). At 1 GHz, the far-field approximation for the radiation from our patch (Schmid et al., 2007) was somewhere between 0.5 and 1.2 cm (Yang et al., 2000). Consequently, radiation from any antenna where the far-field approximation is applicable and the unperturbed electric field is 60 V/m would mimic our exposure conditions. At this field strength, the specific absorption rate calculated using conventional structural models and physiological assumptions is about 0.6 W/kg (less than half of the US-approved limit for cellphones). The maximum electric field measured 2 cm from a cellphone is about 400 V/m (Stewart, 2000).

### Electroencephalograms

Electroencephalograms (EEGs) were obtained from two derivations, using disposable silver/silver-chloride sensors.
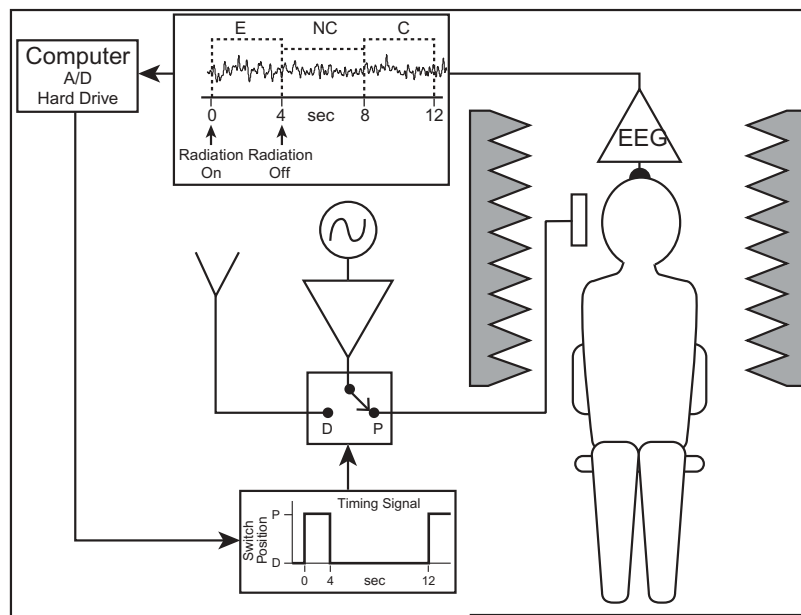


**Figure 1.** Schematic diagram of the exposure and electroencephalogram (EEG) detection systems. D, dummy antenna. P, patch antenna. E, NC, C, respective radiation-exposed, negative-control, and control EEG intervals in a typical trial. A/D, analog-to-digital conversion.

Recording sensors were attached to the forehead on the right (R) and left (L) side of the mid-sagittal plane; each sensor was centered over the eyebrow 1 cm below the hairline (Figure 2). The reference sensor was attached to the left ear (opposite side from the patch antenna) to lessen the possibility of a direct interaction between the sensor and the applied field. Sensor impedances (measured before and after each recording) were <50 kΩ. The EEG voltages were measured continuously, amplified (Nihon Kohden, Irvine, CA, USA), analog-filtered to pass 0.5–35 Hz, sampled at 500 Hz using a 12-bit analog-to-digital converter (National Instruments, Austin, TX, USA), and analyzed offline. The participants underwent 60 trials; the EEG recorded during the time the patch antenna was energized (exposure interval) was compared statistically with the EEG from the last 4 s of the trial (control interval) (Figure 1). As a negative control for the analytical techniques, the negative-control and control intervals (Figure 1) were also compared.

## Hypothesis

Temperature transduction in facial skin occurs at the epidermal–dermal junction, and is mediated by free nerve endings of sensory neurons in the trigeminal nerves, one on each side of the face (Hensel, 1973). The resulting afferent signals (either action potentials or modifications of ongoing afferent signaling) are carried by cells whose bodies are in the trigeminal ganglion and whose axons synapse with secondary neurons in the pons. The secondary neurons decussate in their paths to the thalamus, where they synapse with third-order neurons that pass to the cerebral cortex (Martin, 1989). Cellphone radiation penetrates the skin where its electric vector interacts with the finite electrical conductivity of skin, thereby producing heat. Because the participants were irradiated on the right side of the face and afferent signals from warmth detectors decussate, we predicted that the neurophysiological consequences of the radiation-induced heat would be found in the L-Ref derivation but not necessarily in the R-Ref derivation (Figure 2).

## Theoretical sensitivity limits

To estimate the temperature sensitivity of a warmth-sensing neuron, we compared the change in membrane voltage expected as a result of heat transduction with the noise in the membrane voltage caused by random thermal fluctuations. We assumed that heat transduction was facilitated by thermally sensitive $Na^+$–selective ion channels in the dendritic terminals (Figure 3). The channels have only two states, open and closed, and heat transduction results from changes in their Boltzmann distribution. The number of open channels is $n_1 = N/[1 + e^{-\lambda}]$, where $N$ is the total number of $Na^+$ channels, $\lambda = E/kT$, $E$ is the energy of the open state relative to the closed state, $k$ is the Boltzmann constant, and $T$ is the local temperature. Temperature elevation increases the probability for the channels to be
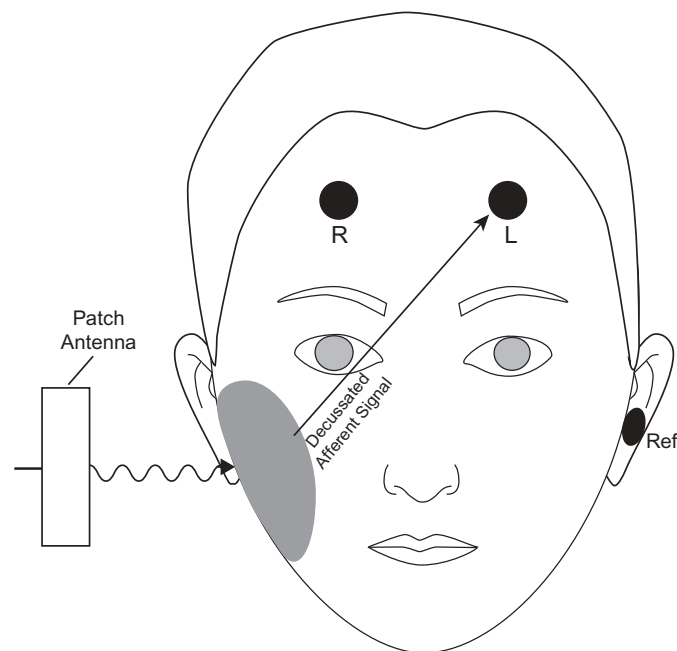


**Figure 2.** Thermal consequences of simulated cellphone radiation on brain electrical activity. Radiation-induced heat is detected by warmth-sensing neurons in the trigeminal nerve. The afferent signals decussate and project to higher brain centers for processing, the effect of which is detected by the left (L) sensor on the forehead. R, right sensor. Ref, reference sensor.
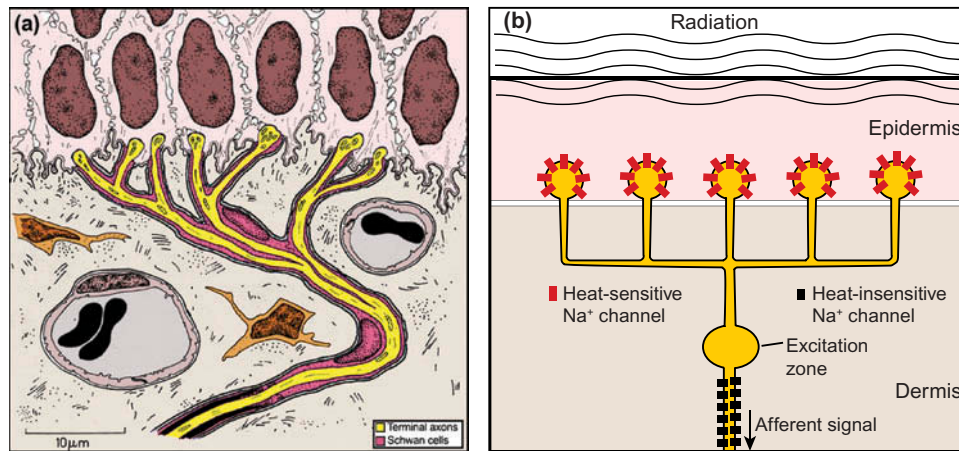
**Figure 3.** Structural and functional representation for thermal transduction of heat deposited in tissue by cellphone radiation. (a) Diagram of nerve endings in the skin (redrawn from (Hensel, 1973)). (b) Model for thermoreception. Dendritic terminals containing the thermally active Na$^+$ channels in the basal epidermis.

in the open state, during which time they pass only Na$^+$. The membrane potential is decreased as a consequence of the increased inward Na$^+$ current, leading to an afferent signal. K$^+$-selective channels, which are located everywhere in the cell membrane, were assumed for simplicity to be temperature insensitive. For simplicity, we assumed that only K$^+$ and Na$^+$ currents determined the membrane potential according to the Goldman equation.

Johnson–Nyquist voltage noise resulting from fluctuations of ion current across the membrane due to thermal motion was estimated as $\sqrt{KT/C}$ (DeFelice, 2012), where $C$ is the membrane-specific capacitance (1 μF/cm$^2$). The actual capacitance of the sensory cell was calculated assuming the dimensions of a typical neuron: cell-body diameter, 20 μm; unmyelinated axon and dendrite, 10 cm and 1 cm, respectively, each with a diameter 1.5 μm in the vicinity of the cell body and 0.2 μm at the terminal.

### Analysis of brain recurrence

Each second of EEG was sent through four Fourier digital filters respectively designed to preserve only the Δ (0.5–4 Hz), θ (>4–8 Hz), α (>8–12 Hz), or β (>12–35 Hz) frequency bands. The aim was to maximize the possibility of observing an effect of the radiation by removing mathematical frequencies in the Fourier representation of the EEG that did not contribute to discrimination between the radiation-exposed and control intervals. The band-specific EEGs were characterized using ABR, a phase-space-based computational method for characterizing the deterministic (law-governed) activity present in the EEG during any given time interval (Frilot II, Kim et al., 2014). ABR is

based on the conjecture that brain function, including cognitive processing of sensory signals, is mediated by electrical activity that occurs in localized neuronal networks as influenced by their dynamic electrical synchronization (Bola and Sabel, 2015; Snyder et al., 2015). In this perspective, an EEG from any location on the head is regarded as a delocalized measure of the instantaneous state of regional brain electrical activity. ABR permits quantification of the extent of deterministic (as opposed to random) activity in the EEG resulting from regional cognitive processing.

The basic signal-processing techniques and their unique applicability to model-independent analyses of nonstationary signals like EEG were previously described (Frilot II et al., 2014; Zbilut and Webber Jr., 2006). Briefly, five-component vectors (points in a five-dimensional mathematical hyperspace) were formed, each consisting of the EEG amplitude at time $t$ and four earlier times identified by successive delays of five points (10 ms) in the digitally sampled EEG. The hyperspace sequence of all such vectors obtainable from 1 s of the EEG (480 vectors, given our choices of sampling rate, vector dimension, and delay time) was interpreted as a path that resulted from law-governed (nonrandom) activity of the brain. The amount of deterministic activity was quantified using the variable $r$ ("percent recurrence"), which measures the number of vectors that are near other vectors (Kantz and Schreiber, 1997), expressed as a percent of the 480 vectors in the space. The Euclidean norm was used for measuring distance in the space; vectors were identified as "near" if they were within 15% of the largest distance between any two vectors in the space. All numerical choices of the ABR parameters were made empirically based on our previous studies (Frilot II et al., 2014).

## Statistics

In preliminary studies involving the first three participants, we found that the intrasubject variance in $r$ was at a minimum when $r$ was computed from the EEG that had been filtered to pass only the $\Delta$ band, and that the variance in $r$ increased progressively when it was computed from the $\theta$, $\alpha$, and $\beta$ bands in the EEG. Correspondingly, consistent differences in average $r$ between the exposed and control intervals were found only in $\Delta$. To increase the likelihood of detecting an effect, we therefore based our statistical hypothesis on an expected difference in $r$ computed from the filtered EEG in which only the $\Delta$ band of frequencies was retained.

Percent recurrence in the $\Delta$ band of the EEG was computed second by second, resulting in 4 s × 60 trials = 240 values per participant from the radiation-exposed (E) intervals and an equal number from the negative-control (NC) and control (C) intervals for both the L–Ref and R–Ref derivations. The values in the respective intervals were averaged and the resulting mean differences were interpreted as a measure of the effect of the radiation on the deterministic activity in the EEG. As a negative control for the possibility that an apparent difference between the exposed (E) and control (C) intervals resulted from the mathematical processing of the data, the negative-control (NC) and control intervals were compared (Figure 1). As a control for the use of a nonlinear analytical method, the relative $\Delta$ power was computed second by second for both derivations and evaluated as described for $r$. Spectral analysis, a linear analytical method, is suboptimal for capturing the nonlinear determinism in the EEG. Group-level statistical significance was assessed for both the ABR and spectral analyses using the paired $t$-test at $p < 0.05$. Tolerances were ±SE unless noted otherwise.

## Results

### Detection of radiation

Variance in frequency-band-specific percent recurrence ($r$) was a minimum in the $\Delta$ band (Table 1), as expected based on the preliminary studies.

When the participants were exposed to simulated cellphone radiation, $r$ computed from the $\Delta$ band of the EEG associated with the decussated afferent signal (Figure 2) was decreased in nine participants (one tie). The mean difference between the exposed and control intervals was 1.1 ± 0.5% ($p < 0.005$) (Figure 4a). Percent recurrence did not differ between the negative-control and control intervals.

In the nondecussated signal, no consistent reduction in $r$ was observed (Figure 4b), and mean $r$ did not differ between the exposed and control intervals ($r = 0.5 \pm 0.5\%$, $p > 0.05$).
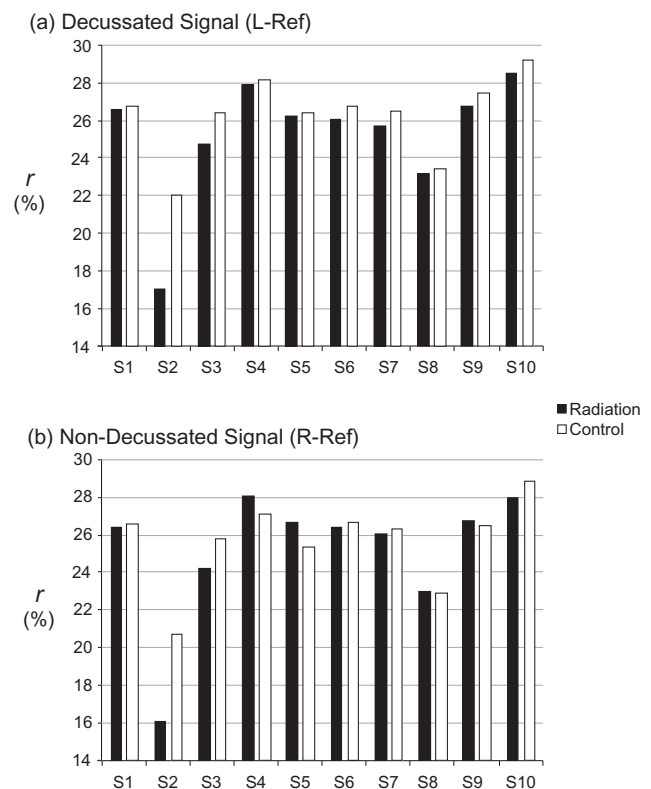


Figure 4. Effect of simulated cellphone radiation on percent recurrence ($r$) computed from the $\Delta$ band of the EEG. Average $r$ during 4-s exposure intervals compared with the corresponding value obtained from 4-s control intervals (N = 60 trials) in 10 subjects (S1–S10). (a) Decussated afferent signal; grand mean of radiation intervals significantly less than the controls ($p < 0.005$). (b) Nondecussated afferent signal; grand means not significantly different.

Table 1. Percent recurrence (mean percent ± SD) in the study group as a function of the frequency band in the EEG from L-Ref and R-Ref derivations. Values averaged over all trials and participants. Fractional standard deviation is shown in parentheses. $\Delta$, 0.5–4 Hz; $\theta$, > 4–8 Hz; $\alpha$, > 8–12 Hz; $\beta$, >12–35 Hz.

|  | $\Delta$ | $\theta$ | $\alpha$ | $\beta$ |
|---|---|---|---|---|
| L-Ref | 27.4 ± 1.8 (0.06) | 19.5 ± 2.7 (0.14) | 13.6 ± 3.2 (0.24) | 16.2 ± 12.9 (0.80) |
| R-Ref | 27.2 ± 2.0 (0.07) | 19.8 ± 3.2 (0.16) | 14.0 ± 3.4 (0.24) | 18.6 ± 14.5 (0.78) |

The mean differences in relative $\Delta$ power were $10.4 \pm 4.5\%$ (seven increases, three decreases) and $5.8 \pm 4.0\%$ (six increases, four decreases) in the decussated and nondecussated signals, respectively; neither mean difference was statistically significant.

### Sensitivity limit

From the Goldman equation, $dV/dT = V_{rest}/T + (k/e_0)(\lambda/(1 + e^{-\lambda}))$, where $e_0$ is the electronic charge. For a typical trigeminal neuron, the Johnson–Nyquist voltage noise ($dV$) is $\sqrt{KT/C} \approx 1.2 \, \mu V$. The corresponding temperature sensitivity of a warmth-sensing neuron ($dT$) computed from the Goldman equation is shown in Figure 5 as a function of $\lambda$, assuming $V_{rest} = -20$ mV. At $\lambda = 3$ (typical for $Na^+$ channels), the temperature sensitivity is at least 5 mK.

### Discussion

Cellphones produce acoustic, electromagnetic, tactile, and thermal sensory stimuli, and unsurprisingly they alter brain metabolism (Volkow et al., 2011). The electromagnetic stimuli include low- and high-frequency electric and magnetic fields, high-frequency radiation, and static magnetic fields. High-frequency radiation interacts with tissue to produce heat, a process that occurs irrespective of whether the tissue is alive or dead and by direct sensory transduction in the visual system, a process that occurs only in living organisms. We were interested in the possibility of an indirect physiological response to cellphone radiation based on transduction of the heat it generated. Our objective was to demonstrate a statistical association between the
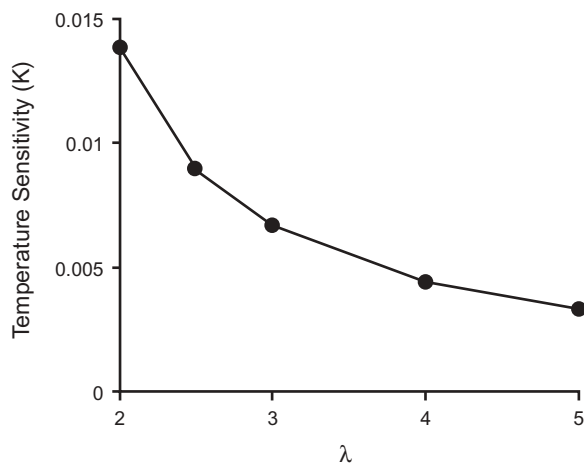


**Figure 5.** The minimum temperature change that can be distinguished from Johnson–Nyquist noise by a warmth-sensing neuron. $V_{rest} = 20$ mV.

presence of the radiation and the occurrence of changes in brain electrical activity.

When percent recurrence in the EEG (a measure of deterministic activity in the brain) was compared in the presence and absence of simulated cellphone radiation, a difference in activity between the two conditions was seen ($p < 0.005$) (Figure 4a). Several considerations indicated that the association of the presence of the radiation and the change in brain electrical activity was truly causal. First, a decrease in $r$ occurred in each participant, except for one case where no change was observed. The probability of nine changes in the same direction due to chance is <0.01. Second, the direction of the effect of the cellphone radiation on $r$ (more complexity, less machine-like behavior) was the same as that previously observed in rabbits due to exposure to cellphone radiation (Marino et al., 2003b). Third, theoretical considerations indicated that trigeminal neurons were capable of detecting temperature changes at least as small as 5 mK (Figure 5), which is far smaller than the skin temperature increase caused by cellphone radiation (Bernardi et al., 2000, 2001; Hirata et al., 2003; Ibrahiem et al., 2005; Van Leeuwen et al., 1999; Wainright, 2000; Wang and Fujiwara, 1999). Fourth, the effect occurred in the decussated signal, consistent with the hypothesis that transduction originated in warmth detectors in the right trigeminal nerve, which was directly exposed to the radiation (Figure 2). Fifth, cognitive processing attendant signal transduction is a nonlinear dynamical phenomenon (Bola and Sabel, 2015; Snyder et al., 2015). Transduction of cellphone-induced heat was detected by ABR, a method specifically developed to quantify nonlinear dynamical activity (Frilot II et al., 2014), but was not detected by linear analysis. The relative effectiveness of ABR indicated that the radiation detection process involved cognitive processing by localized neuronal networks governed by nonlinear dynamical laws, as predicted. Taken together, these five considerations indicated that the simulated cellphone radiation affected brain electrical activity associated with cognitive processing of radiation-induced thermal stimuli. We recognize that this conclusion is at variance with classical expectations. Nevertheless, whether it was correct or was based on inconspicuous technical errors can be assessed only by means of future studies.

TRPV cation channels, which sense temperatures around and above body temperature (Clapham and Miller, 2011), are probably the primary molecular basis for detecting the small changes in skin temperature induced by cellphone radiation. The channels are strongly expressed in sensory neurons and keratinocytes (Nilius and Flockerzi, 2014), and have a functional thermal sensitivity that depends on their anatomical arrangement and functional integration (Moon, 2011). There are additional possible TRPV-related mechanisms that

could account for an effect of cellphone radiation on brain electrical activity. For example, central neurons contain TRPV channels (Menigoz and Boudes, 2011), which raises the possibility that transduction of cellphone radiation may also occur deep in the brain, where radiation-induced temperature increases are known to occur (Bernardi et al., 2000, 2001; Hirata et al., 2003; Ibrahiem et al., 2005; Van Leeuwen et al., 1999; Wainright, 2000; Wang and Fujiwara, 1999). Such a mechanism could explain epidemiological reports of a link in heavy cellphone users between the anatomical location of brain disease and the location of the cellphone during use (Hardell and Carlberg, 2015). We did not address these possibilities.

Slight change in airflow in the experiment room might have caused changes in facial-skin temperature that exceeded those caused by the radiation. Even if so, empirical and experimental-design considerations militate against the possibility that the effect could account for the statistical association between the presence of the radiation and the observed changes in brain electrical activity. No association was found in the negative-control analysis where the radiation-off intervals ($t = 5$–$8$ s in the trial) and control intervals ($t = 9$–$12$ s) were compared. Further, by virtue of the experimental design, any consequences of random stimuli were averaged away, and the possibility that they could have accounted for the observed effect was nil. The only statistically permissible consequence of random fluctuations in air temperature would have been to increase the variance in $r$, thereby lowering the likelihood that an effect of the radiation would be seen.

Putting the observed effect into a broad context, the integrated intensity of solar radiation at the Earth's surface in the range 0.2–10 GHz is about $10^{-8}$ mW/cm$^2$, compared with $10^2$ mW/cm$^2$ attributable to the entire solar spectrum (Pfafflin and Ziegler, 1992), indicating that the power density of the radiation from a typical cellphone is about $10^7$ times greater than the natural background. Considering the solar processes that generate microwave radiation (Tapping, 1987), it is likely that the solar microwave power density at the Earth's surface has not changed during the entire period of human evolution. Thus, cellphones developed instantaneously (in evolutionary terms) and produced a great increase in microwave exposure for almost the entire adult population (Pew Research Center, 2015) without causing any obvious biopathological consequences. Nevertheless, the possibility of nonobvious consequences exists, mostly unstudied by independent investigators. One possibility is that any such consequences are mediated by a specific biophysical mechanism that was not activated during evolution (because the radiation was not present), but that became possible because of evolution (a vulnerability in a conditioned physiological process).

The public-health significance of chronic and subacute exposure to cellphone radiation is under scrutiny (Chu et al., 2011; Coureau et al., 2014; Hardell et al., 2013; Szykjowska et al., 2014), and marked disagreements exist among the stakeholders as regards the public-health risks. Our work is directly pertinent to one aspect of the contentiousness, the assumption that there exists a binary distinction between so-called thermal and nonthermal biological effects associated with cellphone radiation. In that perspective, cellphone radiation is regarded as inherently nonthermal and consequently unable to cause any biological effects, health related or otherwise. The results reported here indicated that a standard cellphone radiating at a level well within approved emission limits will necessarily produce a physiological thermal effect triggered by heat deposited in the user's facial skin. Consequently, cellphone safety cannot validly be predicated on the absence of thermal effects because they are never absent.

A brief description of the historical setting for the work described here seems appropriate. In 1981, we concluded that the biological effects of environmental-strength electromagnetic fields (EEFs) on animals and humans were real (Becker and Marino, 1982), rather than the result of inconspicuous errors in experimental design, execution, or data analysis (Michaelson, 1982; Schwan, 1971), and we hypothesized that the prototypical effect was an emergent property of the organism's stress-response system that followed EEF transduction, rather than a direct cellular or subcellular phenomenon (Bawin and Adey, 1976; Blank and Goodman, 2009). The hypothesis entailed the idea that EEF bioeffects were energetically possible, not forbidden by physical law (Adair, 1991; Weaver et al., 1998). We demonstrated that low- and high-frequency EMFs altered the stress-response system (Bell et al., 1991; Marino et al., 1977; Marino et al., 1979; Marino et al., 1980; Marino et al., 2001; Marino et al., 2000), that transduction occurred in the head (Marino et al., 2003a, 2003b), and that low-frequency transduction could be understood as a direct Coulomb force on aggregates of sugar molecules attached to the gates of ion channels in sensory neurons (Kolomytkin et al., 2007). But that transduction mechanism was precluded at cellphone frequencies by the mechanical inertia of the responding aggregates. Here we showed that an indirect process in which Coulomb forces on atomic charges generated heat that altered the open time of ion-channel gates in sensory neurons could explain transduction of cellphone radiation. Taken as a whole, the various experimental studies support the original hypothesis that the prototypical process by which EEFs (including but not limited to cellphone radiation) are linked to human

disease consists of sensory transduction and a chronic stress response resulting from neuroendocrine–immune interactions.

## Declaration of Interest

The authors report no declarations of interest.

## References

Adair, R. K. (1991). Constraints on biological effects of weak extremely-low-frequency electromagnetic fields. *Phys. Rev. A*, 43:1039–1048.

Bawin, S. M., Adey, W. R. (1976). Sensitivity of calcium binding in cerebral tissue to weak environmental electric fields oscillating at low frequency. *Proc. Natl. Acad. Sci. USA*. 73:1999–2003.

Becker, R. O., Marino, A. A. (1982). *Electromagnetism & Life*. Albany: State University of New York Press.

Bell, G. B., Marino, A. A., Chesson, A. L., Struve, F. (1991). Human sensitivity to weak magnetic fields. *Lancet*. 338:1521–1522.

Bernardi, P., Cavagnaro, M., Pisa, S., Piuzzi, E. (2000). Specific absorption rate and temperature increases in the head of a cellular-phone user. *IEEE Trans. Microwave Theory Tech*. 48:1118–1126.

Bernardi, P., Cavagnaro, M., Pisa, S., Piuzzi, E. (2001). Power absorption and temperature elevations induced in the human head by a dual-band monopole-helix antenna phone. *IEEE Trans. Microwave Theory Tech*. 49:2539–2546.

Blank, M., Goodman, R. (2009). Electromagnetic fields stress living cells. *Pathophysiology*. 16(203):71–78.

Bola, M., Sabel, B. A. (2015). Dynamic reorganization of brain functional networks during cognition. *Neuroimage*. 114:398–413.

Carrubba, S., Frilot, C., Chesson Jr., A. L., Marino, A. A. (2007). Evidence of a nonlinear human magnetic sense. *Neuroscience*. 144: 356–367.

Chu, M. K., Song, H. G., Kim, C., Lee, B. C. (2011). Clinical features of headache associated with mobile phone use: a cross-sectional study in university students. *BMC Neurol*. 11:115.

Clapham, D. E., Miller, C. (2011). A thermodynamic framework for understanding temperature sensing by transient receptor potential (TRP) channels. *PNAS*. 108:19492–19497.

Coureau, G., Bouvier, G., Lebailly, P., et al. (2014). Mobile phone use and brain tumours in the CERENAT case-control study. *Occup. Environ. Med*. 71:514–522.

DeFelice, L. J. (2012). *Introduction to Membrane Noise*. Dordrecht, Netherlands: Springer Science & Business Media.

Frilot II, C., Carrubba, S., Marino, A. A. (2009). Magnetosensory function in rats: localization using positron emission tomography. *Synapse*. 63:421–428.

Frilot II, C., Carrubba, S., Marino, A. A. (2011). Transient and steady-state magnetic fields induce increased fluorodeoxyglucose uptake in the rat hindbrain. *Synapse*. 65:617–623.

Frilot II, C., Carrubba, S., Marino, A. A. (2014). Sensory transduction of weak electromagnetic fields: role of glutamate neurotransmission mediated by NMDA receptors. *Neuroscience*. 258:184–191.

Frilot II, C., Kim, P. Y., Carrubba, S., et al. (2014). Analysis of brain recurrence. In C. Webber & N. Marwan (Eds.), *Recurrence Quantification Analysis: Understanding Complex Systems* (pp. 213–251). Dordrecht, Switzerland: Springer.

Hardell, L., Carlberg, M. (2015). Mobile phone and cordless phone use and the risk for glioma - analysis of pooled case-control studies in Sweden, 1997–2003 and 2007–2009. *Pathophysiology*. 22:1–13.

Hardell, L., Carlberg, M., Hansson Mild, K. (2013). Use of mobile phones and cordless phones is associated with increased risk for glioma and acoustic neuroma. *Pathophysiology*. 20:85–110.

Hensel, H. (1973). Cutaneous thermoreceptors. In A. Iggo (Ed.), *Handbook of Sensory Physiology, Vol. 2: Somatosensory System*. New York: Springer-Verlag.

Hirata, A., Morita, M., Shiozawa, T. (2003). Temperature increase in the human head due to a dipole antenna at microwave frequencies. *IEEE Trans. Electromag. Compatibility*, 45:109–116.

Hirata, A., Shirai, K., Fujiwara, O. (2008). On averaging mass of SAR correlating with temperature elevation due to a dipole antenna. *Prog. Electromagnetics Res*. 84:221–237.

Ibrahiem, A., Dale, C., Tabbara, W., Wiart, J. (2005). Analysis of the temperature increase linked to the power induced by RF source. *Prog. Electromagnetics Res. PIER* 52:23–46.

Kantz, H., Schreiber, T. (1997). *Nonlinear Time Series Analysis*. Cambridge, UK: Cambridge University Press.

Kolomytkin, O. V., Dunn, S., Hart, F. X., et al. (2007). Glycoproteins bound to ion channels mediate detection of electric fields: a proposed mechanism and supporting evidence. *Bioelectromagnetics*. 28:379–385.

Marino, A. A., Berger, T. J., Austin, B. P., et al. (1977). In vivo bioelectrochemical changes associated with exposure to ELF electric fields. *Physiol. Chem. Phys*., 9:433–441.

Marino, A. A., Carrubba, S., Frilot, C., Chesson Jr., A. L. (2009). Evidence that transduction of electromagnetic field is mediated by a force receptor. *Neurosci. Lett*., 452:119–123.

Marino, A. A., Cullen, J. M., Reichmanis, M., Becker, R. O. (1979). Fracture healing in rats exposed to extremely low frequency electric fields. *Clin. Orthop*. 145:239–244.

Marino, A. A., Cullen, J. M., Reichmanis, M., et al. (1980). Sensitivity to change in electrical environment: a new bioelectric effect. *Am. J. Physiol*. 239:R424–427.

Marino, A. A., Nilsen, E., Frilot, C. (2003a). Localization of electroreceptive function in rabbits. *Physiol. Behav*., 79:803–810.

Marino, A. A., Nilsen, E., Frilot, C. (2003b). Nonlinear changes in brain electrical activity due to cell-phone radiation. *Bioelectromagnetics*. 24:339–346.

Marino, A. A., Wolcott, R. M., Chervenak, R., et al. (2001). Coincident nonlinear changes in the endocrine and immune systems due to low-frequency magnetic fields. *NeuroImmunoModulation*. 9:65–77.

Marino, A. A., Wolcott, R. M., Chervenak, R., et al. (2000). Nonlinear response of the immune system to power-frequency magnetic fields. *Am. J. Physiol. Regulatory Integrative Comp. Physiol*. 279:R761–R768.

Martin, J. H. (1989). *Neuroanatomy*. New York: Elsevier.

Menigoz, A., Boudes, M. (2011). The expression pattern of TRPV1 in brain. *J. Neurosci.* 31:13025–13027.

Michaelson, S. M. (1982). Health implications of exposure to radiofrequency/microwave energies. *Br. J. Indust. Med.* 39:105–119.

Moon, C. (2011). Infrared-sensitive pit organ and trigeminal ganglion in the crotaline snakes. *Anat. Cell. Biol.* 44:8–13.

Nilius, B., Flockerzi, V. (Eds.). (2014). *Mammalian Transient Receptor Potential (TRP) Cation Channels, Vol. II*: Springer.

Pew Research Center. (2015). Mobile Technology Fact Sheet:. 2016, from http://www.pewinternet.org/fact-sheets/mobile-technology-fact-sheet/

Pfafflin, J. R., Ziegler, E. N. (1992). *Encyclopedia of Environmental Science and Engineering, Edition 3*. New York: Gordon & Breach Publishing Group.

Schmid, G., Cecil, S., Goger, C., et al. (2007). New head exposure system for use in human provocation studies with EEG recording during GSM900- and UMTS-like exposure. *Bioelectromagnetics*. 28:636–647.

Schwan, H. (1971). Interaction of microwave and radio frequency radiation with biologic systems. *Microwave Theory Tech.*, MTT-19:146–152.

Snyder, A. C., Morais, M. J., Willis, C. M., Smith, M. A. (2015). Global network influences on local functional connectivity. *Nat. Neurosci.*, 18:736–743.

Stewart, W. (2000). Radiofrequency Fields from Mobile Phone Technology. *In* Mobile Phones and Health. from http://webarchive.nationalarchives.gov.uk/20101011032547/http://www.iegmp.org.uk/report/text.htm

Szykjowska, A., Gadzicka, E., Szymczak, W., Bortkiewicz, A. (2014). The risk of subjective symptoms in mobile phone users in Poland—an epidemiological study. *Int. J. Occup. Med. Environ. Health*, 27:293–303.

Tapping, K. F. (1987). Recent solar radio astronomy at centimeter wavelength: the temporal variability of the 10.7-cm flux. *J. Geophys. Res.* 92:829–838.

Van Leeuwen, G. M. J., Lagendijk, J. J. W., Van Leersum, B. J. A. M., et al. (1999). Calculation of change in brain temperatures due to exposure to a mobile phone. *Phys. Med. Biol.* 44:2367–2379.

Volkow, N. D., Tomasi, T., Wang, G. J., et al. (2011). Effects of cell phone radiofrequency signal exposure on brain glucose metabolism. *JAMA*. 305:808–813.

Wainright, P. (2000). Thermal effects of radiation from cellular telephones. *Phys. Med. Biol.* 45:2363–2372.

Wang, J., Fujiwara, O. (1999). FDTD computation of temperature rise in the human head for portable telephones. *IEEE Trans. Microwave Theory Tech.*, 47:1528–1534.

Weaver, J. C., Vaughan, T. E., Adair, R. K., Astumian, R. D. (1998). Theoretical limits on the threshold for the response of long cells to weak extremely low frequency electric fields due to ionic and molecular flux rectification. *Biophys. J.* 75:2251–2254.

Yang, K., David, G., Yook, J.-G., et al. (2000). Electrooptic mapping and finite-element modeling of the near-field pattern of a microstrip patch antenna. *IEEE Trans. Microwave Theory Tech.*, 48:288–294.

Zbilut, J. P., Webber Jr., C. L. (2006). Recurrence quantification analysis. In M. Akay (Ed.), *Wiley Encyclopedia of Biomedical Engineering* (pp. 2979–2986). Hoboken: John Wiley & Sons.