Effect of acute exposure to radiofrequency electromagnetic fields emitted by a mobile phone (GSM 900 MHz) on electrodermal responsiveness in healthy human

Brahim Selmaoui, Soafara Andrianome, Rania Ghosn, René de Seze

Effect of acute exposure to radiofrequency electromagnetic fields emitted by a mobile phone (GSM 900 MHz) on electrodermal responsiveness in healthy human

[1,2*]Brahim Selmaoui, [1,2]Soafara Andrianome, [1,2]Rania Ghosn and [1,2]René de Seze

[1]Institut National de l'Environnement Industriel et des Risques (INERIS), Department of Experimental Toxicology, Verneuil-en-Halatte, France

[2]Université de Picardie Jules Verne, Peritox-Laboratoire de Périnatalité et Risques Toxiques UMR-I-01 Unité mixte INERIS, Amiens, France

Running head: Electrodermal responsiveness to a GSM 900 MHz signal

*** Corresponding author:** Brahim Selmaoui, PhD, HDR; INERIS, Parc Technologique ALATA, 60550 Verneuil en Halatte, France; tel.: +33 3 44 61 82 68; e-mail: brahim.selmaoui@ineris.fr

Effect of acute exposure to radiofrequency electromagnetic fields emitted by a mobile phone (GSM 900 MHz) on electrodermal responsiveness in healthy human

Abstract

Purpose: The present study aimed to determine the effect of acute exposure to electromagnetic fields (EMF) emitted by a mobile phone on electrodermal activity (EDA) in response to an auditory stimulus. **Materials and methods:** The EDA of 28 young volunteers was recorded following 26 min of exposure to a GSM mobile phone (900 MHz). Palmar sensors enabled repeat recording of 2 min 45 s in the pre-exposure, exposure and post-exposure phases in response to sound stimuli. **Results:** The latency, amplitude of skin conductance responses (SCRs), integral of skin conductance response and number of SCRs in response to the auditory stimuli were not modified by exposure. Skin conductance and tonic activity decomposition of the recorded signal were significantly different between the two sessions ($p < 0.0001$), but the changes could not be attributed to EMF exposure. There was also a tendency toward a fast reduction in the amplitude and number of electrodermal responses after placement of the mobile phone. In response to successive stimuli, there was a significant difference between the first response and subsequent responses for all variables except latency. **Conclusion:** Our results showed a decrease in the number of responses and their amplitude as a result of placement of the mobile device and whether it was turned "on" or "off," but there were no changes associated with exposure to GSM radiofrequency waves in this group of volunteers.

Keywords: *Electrodermal activity (EDA); Galvanic skin response (GSR); Mobile phone; Skin conductance response (SCR)*

Introduction

Mobile phones represent the most commonly used and studied source of electromagnetic fields (EMF) in recent decades. Due to the close proximity of the mobile phone to the head during its use, a particular concern has been raised with regard to the possible effects of EMF exposure on the brain. Exploration of the autonomic nervous system (ANS) remains an interesting way to analyse the reaction of an organism to EMF exposure. Numerous provocation studies assessing the effects of EMF exposure (radiofrequency) on the physiology of organisms have been undertaken, and these typically explore brain electroencephalography (EEG), heart rate, heart rate variability (HRV) or respiration rate. Recording electrodermal activity (EDA), among other techniques, is a non-invasive and easy low-tech solution for measuring ANS changes. Electrodermal activity refers to changes in skin conductance on the surface, reflecting the autonomic innervations of eccrine sweat glands. Although EDA is also known as skin conductance (SC) or galvanic skin response (GSR), EDA is the currently used term. The EDA complex includes both background tonic (skin conductance level, SCL) and rapid phasic components (skin conductance responses, SCRs) that result from sympathetic neuronal activity. As EDA is a sensitive index, it is also widely used in psychological studies (Sequeira et al., 2009; Dawson et al., 2011). An electrodermal response can be elicited by discrete stimuli, inducing rapid changes (phasic) that are in contrast to the slow variations associated with a basic level of EDA (tonic). Neuroimaging studies have shown that the basal measurement, represented by the SCL, varies in line with neural activity within the ventromedial prefrontal cortex and the orbitofrontal cortex, providing a task-independent representation of the autonomic state. On the other hand, several regions of the central nervous system were found to be activated for event-related activity or SCR (Patterson et al., 2002; Nagai et al., 2004).

Very few studies have examined EDA under mobile phone (MP) exposure; however, several studies on the effect of EMF on EEG have shown a consistent effect of MP on the EEG by increasing or decreasing alpha (8–13 Hz) power (Reiser et al., 1995; Croft et al., 2002; Huber et al., 2002; Karamenko et al., 2003; Cook et al., 2004; Curcio et al., 2005; Maby et al., 2006; Perentos et al., 2007;

Regel et al., 2007; Vecchio et al., 2007; Hinrikus et al., 2008; Vecchio et al., 2012; Perentos et al., 2013; Ghosn et al., 2015; Yang et al., 2017).

With regard to EDA, one study showed a shorter latency of EDA under exposure to 900 MHz (Esen and Esen, 2006). In studies involving subjects hypersensitive to EMF, the results showed no effect of EMF exposure on the latency of the amplitude, the number of peaks of SCR (Wilén et al., 2006) or on the level of skin conductance (Eltiti et al., 2009).

While these studies are informative for identifying the effects of EMF on general skin conductance, several techniques have been developed over recent decades to specifically analyse skin conductance (Benedek et al., 2010). Development of mathematical models has enabled better reduction in noise and better estimation of SCR.

Using a mathematical-based model, we aimed to determine whether exposure to 26 min of EMF from a mobile phone with a GSM signal has an effect on numerous parameters of EDA, including slowly varying activity or skin conductance level (SCL) and the short-lasting skin conductance response (SCR), by analysing changes in several parameters (latency, amplitudes, the integral of SCR) in response to repeated auditory stimuli.

Materials and methods

Participants

Thirty participants were recruited for this study. Due to technical problems, data from two participants were excluded from the final analysis. Fourteen healthy males and 14 healthy females aged between 19 and 31 years (The mean age of participants was 24 ± 3 years) were enrolled in this study. All participants were healthy without disease. The average (mean ± SD) body mass index (BMI) of participants was 21.96 ± 2.1 kg/m², and systolic and diastolic blood pressure measures were 118 ± 8 and 72.8 ± 6.7 mmHg, respectively. There was no significant difference between male and female participants in terms of BMI ($F = 2.074$, not significant [ns]), blood pressure ($F = 0.39$, ns; $F = 1.129$, ns) and age ($F = 3.089$, ns). Participants were recruited through a website advertisement and were

selected after a routine clinical examination. The inclusion criteria were regular sleep habits, the absence of medication, chronic disease, disability or acute illness, being a non-smoker, and the absence of neurological or psychiatric illness. Volunteers were asked to avoid eating or drinking alcohol and coffee, or any other stimulants 24 h prior to and during the experimental sessions. They were also instructed not to use a mobile phone for at least 2 h before the start of the experiment. Participants received compensation, and the study was approved by the local ethics committee (CPP ref: N°ID-RCB: 2011-A01455-36).

Study design

Electrodermal activity, including SCR, was simultaneously recorded with electrocardiogram (ECG) and EEG parameters (results of the EEG were previously presented in Ghosn et al., 2015) over two sessions in a randomised, double-blinded, counterbalanced study design. Neither the participant nor the researchers were aware of the exposure. Each subject was randomly assigned to receive two sessions of real or sham exposure. Each session included pre-exposure (run 1 and run 2), exposure (run 3, run 4, run 5) and post-exposure (run 6, run 7) periods. The total session duration was 61 min, including 26 min 15 s of real or sham exposure. Each session was separated by at least one week, and the order of exposure was counterbalanced. Each participant attended the two experimental sessions at the same time of the day to avoid any confounding effects related to circadian rhythm.

Exposure system

A commercial, dual-band, GSM mobile phone (Nokia 6650) was used for RF exposure. For the "real" exposure, participants were exposed to GSM-modulated exposure with the full power of the mobile phone (2W peak, 250 mW average, pulse modulated with 1/8 duty cycle) at 900 MHz for 26 min. Maximum SAR, averaged for 10 g tissue, 1 g tissue or the peak value, was 0.49, 0.70 and 0.93 W/kg, respectively. The SARs during the "sham" exposure were <0.0001 W/kg. Before the exposure session, the mobile phone was placed on the left ear of the participants and secured using a tubular bandage (for more details see Ghosn et al., 2012 and Ghosn et al., 2015). Skin temperature measured during the

experiment in the right side (with no handset) did not significantly change in the cheek and under the ear. It was stable during sham and real exposure (about 24.5 °C).

Physiological measurements

The EDA and SCR were recorded using a BIOPAC MP 150 (Biopac systems, Goleta, CA, USA). Two Ag/AgCl electrodes were placed at the end of the second and third fingers of the participants' left hand (GSR 100C). The BIOPAC MP 150 was connected to a Dell laptop for acquisitions using Acqknowledge software. For all sessions, instructions were given through Omnistim at the beginning of the recordings. Omnistim also delivers a burst for SCR through loudspeakers, with signal tones of 60 dB and 1000 Hz lasting for 0.3 s. The interstimulus interval was 15 s. Each EDA recording had a duration of 2 min 45 s, and the inter-run duration was 6 min. Each stimulus delivered was recorded as a visual signal so that it could be synchronised with data for analysis. In order to obtain data of sufficient quality, participants were requested to take some sharp and deep breaths so that the quality of the response could be checked (positive control). The protocol for the EDA recording is shown in Figure 1.

Data analysis

The frequency of the sample was corrected and resampled from 10 to 1000 Hz. Artifacts were visually inspected, and a continuous decomposition analysis (CDA) was performed using Ledalab 3.3.4 (Benedek and Kaernbach, 2010) for component analysis. In parallel, the software could also provide parameters using the trough to peak analysis method (TTP) and global measurement calculations. Skin conductance was first decomposed into tonic activity and phasic activity. The phasic and tonic amplitudes of each session for each run were recorded and analysed. The parameters of responses were then linked to an auditory stimulus, and the SCR studied was extracted using the CDA and TTP methods. This included a number of SCRs above the threshold, the latency of the response, SCR sum of amplitude and the integral of skin conductance response (ISCR). We defined the following parameters for SCR: a response window of 1 to 4 s, and a SCR threshold amplitude of 0.02

microSiemens (µS). The global measurement parameters were the global mean and global max deflection.

Statistical analysis

Statistical analysis was performed using R software and SPSS. After extraction of tonic and phasic activities, runs and sessions were analysed as factors and mean values for tonic and phasic activity during each run as the variables. For SCR-related parameters, the effect of habituation was analysed considering the event number as a factor. Then, two-way ANOVA was performed for each parameter, with the number of SCRs, latency, SCR, ISCR and global mean and global max deflection considered as variables, and runs and sessions considered as factors. A p-value <0.05 was considered statistically significant.

Results

Skin conductance (SC) and tonic (TA) and phasic activity (PA)

Tonic activity, extracted from skin conductance by CDA, was significantly different between the two sessions ($F = 4.79$, $p < 0.0001$), but was not significantly different between runs ($F = 0.44$, $p = 0.9401$; Figure 2a). Participants submitted to the "real" session of RF exposure had higher mean SC and TA during the session compared to those who received the "sham" session.

There was no difference between runs or sessions for PA, which was also extracted from the CDA, reflecting rapid changes in EDA (run: $F = 1.04$, $p = 0.6756$; session: $F = 0.17$, $p = 0.4246$; Figure 2b).

Proportion of significant responses (continuous decomposition method)

The number of significant responses or electrodermal responses, considering a peak detection threshold of 0.02 µS and a time window of 1 to 4 s, was determined for both sessions. A comparison of the proportion of responses to the auditory stimulus between sessions showed no difference

between sessions ($chi^2 = 2.283$, $p = 0.065$), but there was a significant difference between runs ($chi^2 = 35.144$, $p < 0.0001$), independent of session (data not shown).

Habituation effect

Ten successive auditory stimuli were presented to the participants. All parameters were tested for a habituation effect using a block-test method considering the number of events as a factor. The majority of parameters were found to be influenced by the event number, except for latency which remained unchanged (Figures 3 and 4).

Electromagnetic field effect

Parameters characterising SCR were analysed by two-way ANOVA with sessions and runs considered to be factors. The ANOVA for parameters determined by the three different methods is presented in Table 1. Differences between sessions were found using the TTP method (nSCR, latency and AmpSum), although this result was not confirmed by the CDA method (no effect of session; Figures 5 and 6).

Discussion

The present study aimed to evaluate EDA, including SCR parameters, in response to brief auditory stimuli under exposure to RF EMFs. Electrodermal activity is a potential noninvasive marker of sympathetic activity as it can be used to evaluate changes in the function of the ANS. Most commonly, SCR is used as a peripheral index of a central state of sympathetic arousal (Boucsein, 2012; Bach, 2012). Electrodermal acquisition is based on the innervations of the sweat glands by the sympathetic nervous system. Furthermore, sudomotor activity plays an important role in thermoregulation (Wenger, 2003)

The method applied in this study, which involved the use of a model to analyse SCRs, allowed us to obtain less noisy results compared to conventional methods. Conventional analysis of SCR data consists of extraction of indices from an electrodermal recording using a method such as the peak

detection method. These conventional methods are limited by the need for better temporal resolution, the need to reduce noise that cannot be attributed to sympathetic arousal, and the need for greater precision. In this model-based analysis, we used a mathematical model that considers the mechanism leading to a SCR, which results from sympathetic arousal and subsequent stimulation of the sudomotor nerve. A major problem with the conventional scoring technique is the difficult distinction between overlapping SCRs when there are short intervals between stimuli or between SCRs in quick succession. The decomposition procedure first proposed by Alexander et al. (2005) was applied in this study. In order to remove between-subject variation, a z transformation is often advised. However, recent studies have shown that this transformation does not significantly improve the predictive validity of the data (Bach, 2014). More recently, newer model-based methods, similar to the model used in the present study, have shown higher sensitivity for distinguishing differences in sympathetic arousal between two different arousal responses under experimental conditions. This is due to the integrated removal of between-subject variance and methodological differences (Bach, 2014).

Our results showed no effect of RF EMF exposure for 26 min on EDA in healthy young people. However, the tonic activity extracted from skin conductance measures was significantly different between the two sessions, but not different between runs. Participants had higher average SC and tonic activity during the real exposure session than the control session. These differences were seen before the exposure period as well as during the post-exposure period, meaning that these differences cannot be attributed to RF EMF exposure. Instead, they can be considered intraindividual variations from session to session. This difference is not due to diurnal variability (Hot et al., 1999), as all experiments were performed at the same time of day.

With regard to latency, our results are not in accordance with those previously reported in a study by Esen and Esen, in which latency was found to be lengthened by approximately 200 ms under exposure to the EMFs of a mobile phone (Esen and Esen, 2006). It should be noted that there are differences in the methodological approach between our experiment and that of Esen and Esen. For example, the external stimulus in the present study consisted of an auditory stimulus, whereas Esen and Esen used a mechanical stimulus in the form of a “tap” on the patellar tendon. Furthermore, effects on

electrodermal responses or SCRs are known to vary according to age, gender, ethnicity and skin physiology, and also depend on the nature of the stimuli and the environment of the experiment (Boucsein, 2012).

Another study by Johansson et al. on people with atopic dermatitis (Johansson et al., 2008) reported no effect of exposure to a mobile phone-like RF in terms of the number of peaks per minute for the EDA. In studies involving subjects who were hypersensitive to EMF, the results failed to show an effect of EMF exposure on average skin conductance (Wilén et al., 2006; Andrianome et al., 2017) or on EDA measured by the number of peaks per minute (Eltiti et al., 2009). Moreover, a study on the effect of short-term exposure to magnetic fields did not show any effect on skin conductance, expressed as z-scores, and further analyses showed that subjects showed different behavioural responses during exposure, ranging from reduced SC, increased SC or no change (Stevens, 2001).

In our study, we found that the number of SCRs was lower after placement of the mobile phone, regardless of whether exposure was “real” or “sham”. This observation indicates that there is no specific activation or increase in sympathetic activity in response to exposure to the GSM signal or in response to the mobile phone. Furthermore, in response to successive identical stimuli, there was a significant difference between the first response and subsequent responses for all variables except for latency. Our data also showed a tendency toward a faster reduction in amplitude, which is in accordance with data in the literature (Isen et al., 2012). This is due, in part, to the habituation of subjects to the stimulus. Subjects become less responsive to an auditory stimulus under both conditions, as well as after the second presentation of the auditory stimulus. To analyse the overall responsiveness, the authors suggest that the number of SCRs in response to a series of stimuli, as shown above, are more reliable than studies of habituation (Dawson and Schell, 2002).

Conclusions

The lack of effect of RF exposure on elicited SCRs and EDA indicates that sympathetic nervous activity, one of the main branches of the autonomic nervous system, was not affected by RF exposure, at least under the conditions of the present study.

The results of our study do not support the hypothesis that exposure to MP-like RF fields can affect the EDA system in healthy volunteers.

Acknowledgements

This study was fully funded by Institut National de l'Environnement Industriel et des Risques (INERIS) and the program 190 'post-Grenelle' for the 'pôle applicatif en Toxicologie et Ecotoxicologie' of the French Ministry of Ecology and Sustainable Development. We would also like to thank Jean-François Lambert for his precious advice.

Declaration of interest statement

The authors report no conflicts of interest.

REFERENCES

Alexander DM, Trengove C, Johnston P, Cooper T, August JP, Gordon E. 2005. Separating individual skin conductance responses in a short interstimulus-interval paradigm. J. Neurosci. Methods. 146:116–123.

Andrianome S, Gobert J, Hugueville L, Stéphan-Blanchard E, Telliez F, Selmaoui B. 2017. An assessment of the autonomic nervous system in the electrohypersensitive population: a heart rate variability and skin conductance study. J. Appl. Physiol. (1985). 123(5):1055–1062.

Bach DR. 2014. A head-to-head comparison of SCRalyze and Ledalab, two model-based methods for skin conductance analysis. Biol. Psychol. 103:63–68.

Bach DR. 2014. Sympathetic nerve activity can be estimated from skin conductance responses - a comment on Henderson et al. (2012). NeuroImage. 84:122–123.

Benedek M, Kaernbach C. 2010. A continuous measure of phasic electrodermal activity. J. Neurosci. Methods. 190:80–91.

Boucsein W. 2012. Electrodermal Activity [Internet]. Boston, MA: Springer US [cited 9 May 2016]. Available from: http://link.springer.com/10.1007/978-1-4614-1126-0

Cook CM, Thomas AW, Prato FS. 2004. Resting EEG is affected by exposure to a pulsed ELF magnetic field. Bioelectromagnetics. 25:196–203.

Croft RJ, Chandler JS, Burgess AP, Barry RJ, Williams JD, Clarke AR. 2002. Acute mobile phone operation affects neural function in humans. Clin. Neurophysiol. 113:1623–1632.

Curcio G, Ferrara M, Moroni F, D'Inzeo G, Bertini M, De Gennaro L. 2005. Is the brain influenced by a phone call? An EEG study of resting wakefulness. Neurosci. Res. 53:265–270.

Dawson ME, Schell AM, Courtney CG. 2011. The skin conductance response, anticipation, and decision-making. J. Neurosci. Psychol. Econ. 4:111–116.

Dawson ME, Schell AM. 2002. What does electrodermal activity tell us about prognosis in the schizophrenia spectrum? Schizophr. Res. 54:87–93.

Eltiti S, Wallace D, Ridgewell A, Zougkou K, Russo R, Sepulveda F, et al. 2009. Short-term exposure to mobile phone base station signals does not affect cognitive functioning or physiological measures in individuals who report sensitivity to electromagnetic fields and controls. Bioelectromagnetics. 30:556–563.

Esen F, Esen H. 2006. Effect of electromagnetic fields emitted by cellular phones on the latency of evoked electrodermal activity. Int. J. Neurosci. 116:321–329.

Ghosn R, Thuróczy G, Loos N, Brenet-Dufour V, Liabeuf S, de Seze R, Selmaoui B. 2012. Effects of GSM 900 MHz on middle cerebral artery blood flow assessed by transcranial Doppler sonography. Radiat Res. 178(6):543–550.

Ghosn R, Yahia-Cherif L, Hugueville L, Ducorps A, Lemaréchal JD, Thuróczy G, de Seze R, Selmaoui B. 2015. Radiofrequency signal affects alpha band in resting electroencephalogram. J. Neurophysiol. 113(7):2753–2759.

Hinrikus H, Bachmann M, Lass J, Tomson R, Tuulik V. 2008. Effect of 7, 14 and 21 Hz modulated 450 MHz microwave radiation on human electroencephalographic rhythms. Int. J. Radiat. Biol. 84:69–79.

Hot P, Naveteur J, Leconte P, Sequeira H. 1999. Diurnal variations of tonic electrodermal activity. Int. J. Psychophysiol. 33:223–230.

Huber R, Treyer V, Borbély AA, Schuderer J, Gottselig JM, Landolt H-P, et al. 2002. Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG. J. Sleep Res. 11:289–295.

Isen JD, Iacono WG, Malone SM, McGue M. 2012. Examining electrodermal hyporeactivity as a marker of externalizing psychopathology: a twin study. Psychophysiology. 49:1039–1048.

Johansson A, Forsgren S, Stenberg B, Wilén J, Kalezic N, Sandström M. 2008. No effect of mobile phone-like RF exposure on patients with atopic dermatitis. Bioelectromagnetics. 29:353–362.

Kramarenko AV, Tan U. 2003. Effects of high-frequency electromagnetic fields on human EEG: a brain mapping study. Int. J. Neurosci. 113:1007–1019.

Maby E, Le Bouquin Jeannes R, Faucon G. 2006. Short-term effects of GSM mobiles phones on spectral components of the human electroencephalogram. Conf. Proc. IEEE Eng. Med. Biol. Soc. 1:3751–3754.

Nagai Y, Critchley HD, Featherstone E, Trimble MR, Dolan RJ. Activity in ventromedial prefrontal cortex covaries with sympathetic skin conductance level: a physiological account of a “default mode” of brain function. NeuroImage. 22:243–251.

Patterson JC, Ungerleider LG, Bandettini PA. 2002. Task-independent functional brain activity correlation with skin conductance changes: an fMRI study. NeuroImage. 17:1797–1806.

Perentos N, Croft RJ, McKenzie RJ, Cosic I. 2013. The alpha band of the resting electroencephalogram under pulsed and continuous radio frequency exposures. IEEE Trans. Biomed. Eng. 60:1702–1710.

Perentos N, Croft RJ, McKenzie RJ, Cvetkovic D, Cosic I. 2007. Comparison of the effects of continuous and pulsed mobile phone like RF exposure on the human EEG. Australas. Phys. Eng. Sci. Med. 30:274–280.

Regel SJ, Gottselig JM, Schuderer J, Tinguely G, Rétey JV, Kuster N, et al. 2007. Pulsed radio frequency radiation affects cognitive performance and the waking electroencephalogram. Neuroreport. 18:803–807.

Reiser H, Dimpfel W, Schober F. 1995. The influence of electromagnetic fields on human brain activity. Eur. J. Med. Res. 1:27–32.

Sequeira H, Hot P, Silvert L, Delplanque S. 2009. Electrical autonomic correlates of emotion. Int. J. Psychophysiol. 71:50–56.

Stevens P. 2001. Effects of 5 s exposures to a 50 microT, 20 Hz magnetic field on skin conductance and ratings of affect and arousal. Bioelectromagnetics. 22:219–223.

Vecchio F, Babiloni C, Ferreri F, Curcio G, Fini R, Del Percio C, et al. 2007. Mobile phone emission modulates interhemispheric functional coupling of EEG alpha rhythms. Eur. J. Neurosci. 25:1908–1913.

Vecchio F, Buffo P, Sergio S, Iacoviello D, Rossini PM, Babiloni C. 2012. Mobile phone emission modulates event-related desynchronization of α rhythms and cognitive-motor performance in healthy humans. Clin. Neurophysiol. 123:121–128.

Wenger. 2003. Thermoregulation. In: Freedberg IM, Fitzpatrick TB (eds) Fitzpatrick's dermatology in general medicine. 5th ed. New York: McGraw-Hill, Health Professions Division.

Wilén J, Johansson A, Kalezic N, Lyskov E, Sandström M. 2006. Psychophysiological tests and provocation of subjects with mobile phone related symptoms. Bioelectromagnetics. 27:204–214.

Yang L, Chen Q, Lv B, Wu T. 2017. Long-term evolution electromagnetic fields exposure modulates the resting state EEG on alpha and beta bands. Clin. EEG Neurosci. 48(3):168–175.

FIGURE LEGENDS

Figure 1. Study protocol for physiological measurements during the pre-exposure, exposure and post-exposure periods. Each electrodermal activity (EDA) recording period (2 min 45 s) is indicated in the square with the stimulation period.

Figure 2. (a) Tonic activity (TA) during the sham and real sessions. Significant differences were observed between the sham and real sessions ($F = 4.79$, $p < 0.0001$). **(b)** Phasic activity (PA) during the sham and real sessions. No significant differences were detected when the sham and real sessions were compared. Data were expressed as mean ± SEM.

Figure 3. Sum of amplitude of skin conductance responses (SCRs) for 10 consecutive auditory stimuli in sham and exposure sessions. Significant differences were observed between runs. Data were expressed as mean ± SEM.

Figure 4. Latency of skin conductance responses (SCRs) during 10 successive auditory stimuli in sham and exposure sessions. No significant differences were observed between runs and sessions. Data were expressed as mean ± SEM.

Figure 5: Sum of amplitude of skin conductance responses (SCRs) for each run (from run 1 to run 7) in the sham and real sessions. No significant differences were observed between sessions and runs. Data were expressed as mean ± SEM.

Figure 6: Variations in latency of skin conductance responses (SCRs) for each run (from run 1 to run 7) in the sham and real sessions. No significant differences were observed between runs and sessions. Data were expressed as mean ± SEM.

Table 1: Summary of analysis of variance
Factors: Session and runs (run 1, run 2, run 3, run 4, run 5, run 6, run 7); variables: continuous decomposition analysis (CDA) method: nSCR, AmpSum, Latency, SCR, ISCR, trough to peak (TTP) method: n SCR; latency, AmpSum; global measurements: global mean, global max deflection. *nSCR: number of significant skin conductance response, AmpSum: Sum of amplitudes of significant skin conductance response; SCR: Skin conductance response, ISCR: Integral of skin conductance response*

	Session		**Run**		**Interaction**	
Continuous decomposition analysis	**p value**	**F**	**p value**	**F**	**p value**	**F**
nSCR	0.901	0.015	< 0.001***	5.554	0.5097	0.8784
Latency	0.427	0.6311	0.0339*	2.276	0.0388*	2.216
AmpSum	0.597	0.2791	0.006**	3.018	0.0764	1.904
SCR	0.553	0.3525	0.119	1.684	0.027*	2.378
ISCR	0.553	0.3525	0.119	1.684	0.027*	2.378
Through to peak	**p value**	**F**	**p value**	**F**	**p value**	**F**
nSCR	0.029*	4.77	0.0007***	3.918	0.0195*	2.52
Latency	0.0031**	8.738	0.0003***	4.17	< 0.0001***	5.202
AmpSum	0.0378*	4.315	0.019	2.532	0.0407*	2.195
Global measurements	**p value**	**F**	**p value**	**F**	**p value**	**F**
Global mean	< 0.0001***	93.99	0.0006***	3.943	0.3735	1.077
Global max deflection	0.3598	0.8387	0.0029***	3.316	0.0229*	2.451

Figure 1.

Abbreviations: EDA: Electrodermal activity; EEG: Electroencephalogram; ECG: Electrocardiogram

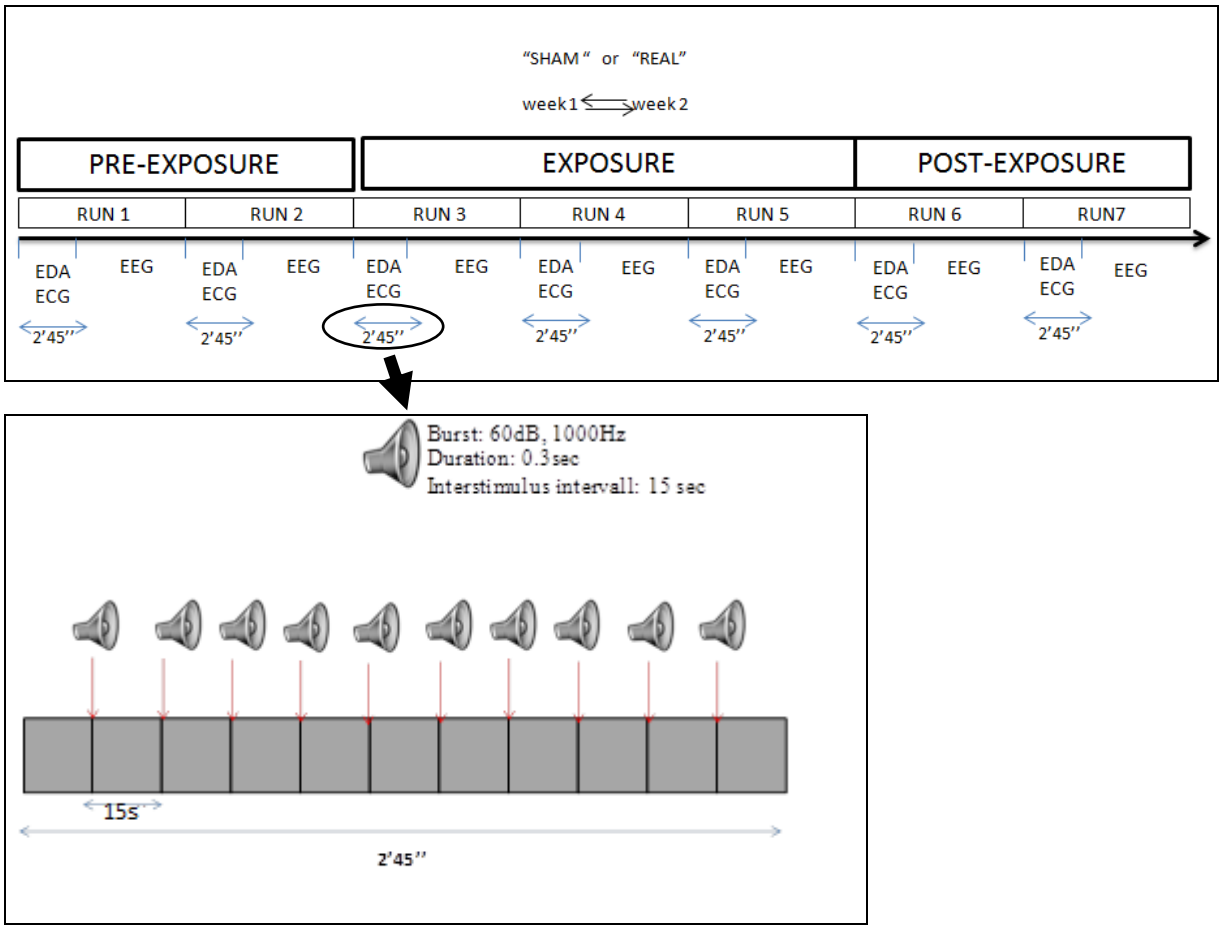
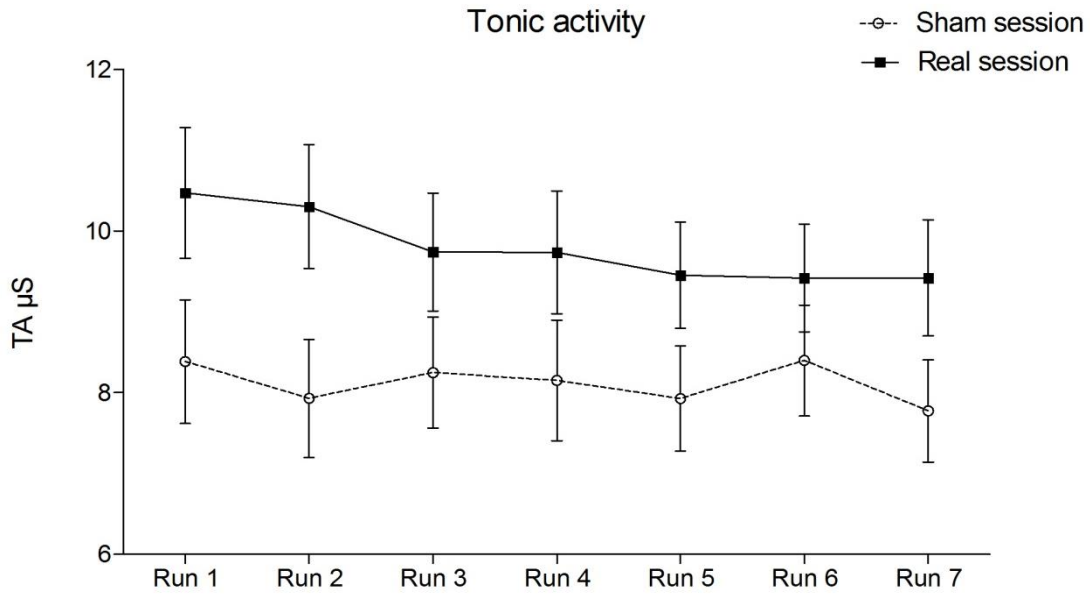


Figure 2

a)


Tonic activity
Sham session
Real session
TA µS
12
10
8
6
Run 1
Run 2
Run 3
Run 4
Run 5
Run 6
Run 7

b)

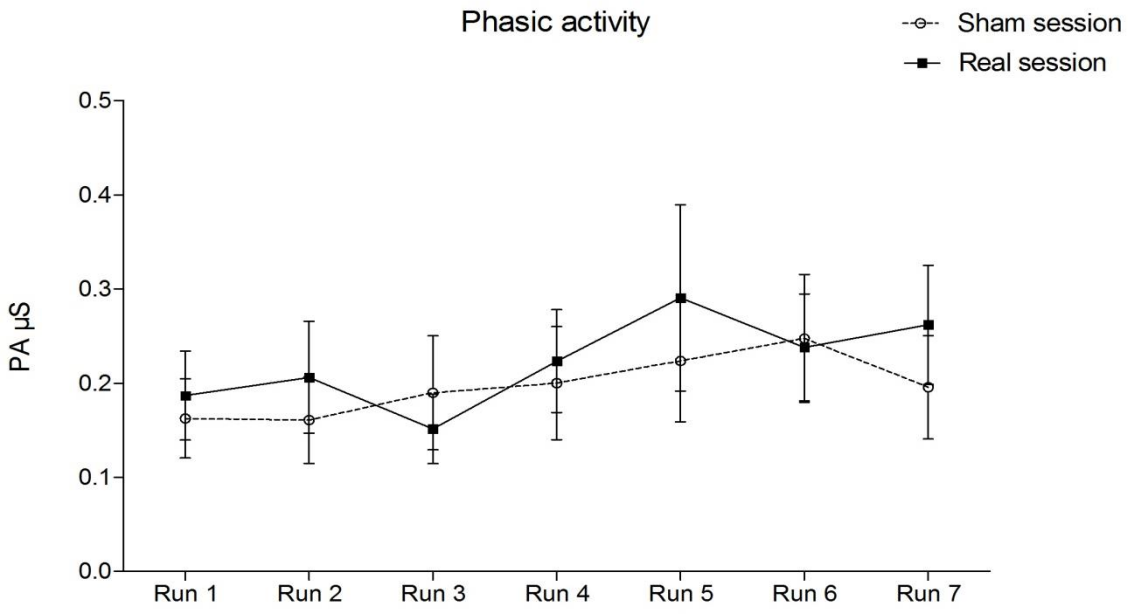

Phasic activity
Sham session
Real session
PA µS
0.5
0.4
0.3
0.2
0.1
0.0
Run 1
Run 2
Run 3
Run 4
Run 5
Run 6
Run 7

Figure 3.

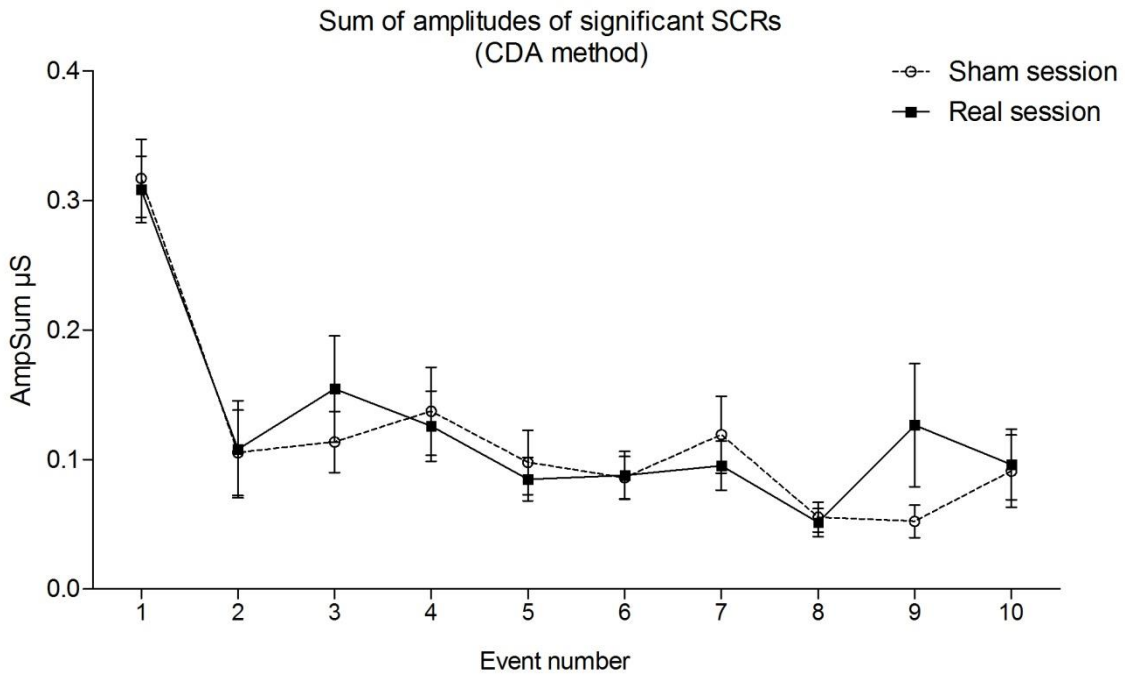

Sum of amplitudes of significant SCRs
(CDA method)
Sham session
Real session
AmpSum µS
0.4
0.3
0.2
0.1
0.0
1
2
3
4
5
6
7
8
9
10
Event number

Figure 4.

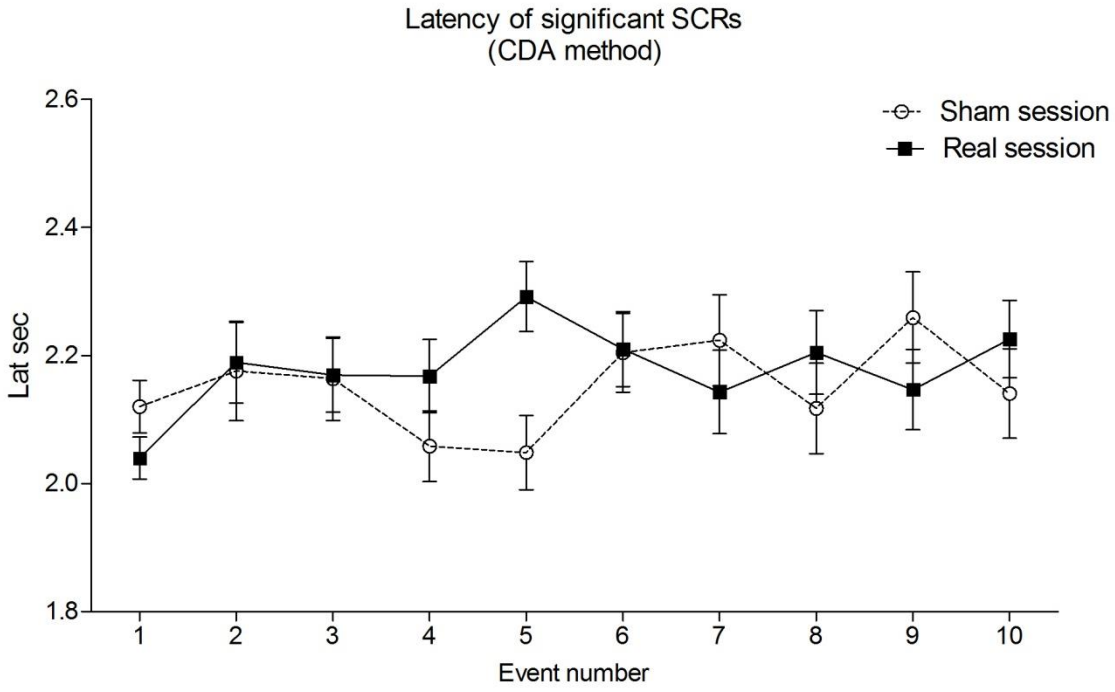

Latency of significant SCRs
(CDA method)
Sham session
Real session
Lat sec
2.6
2.4
2.2
2.0
1.8
1
2
3
4
5
6
7
8
9
10
Event number

Figure 5:

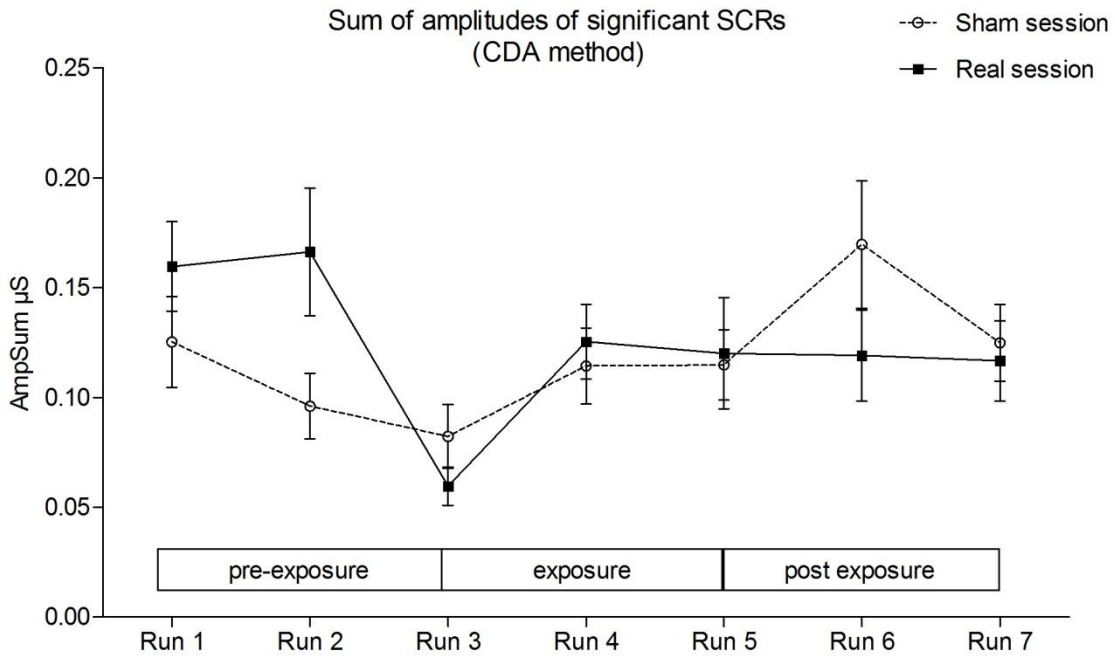
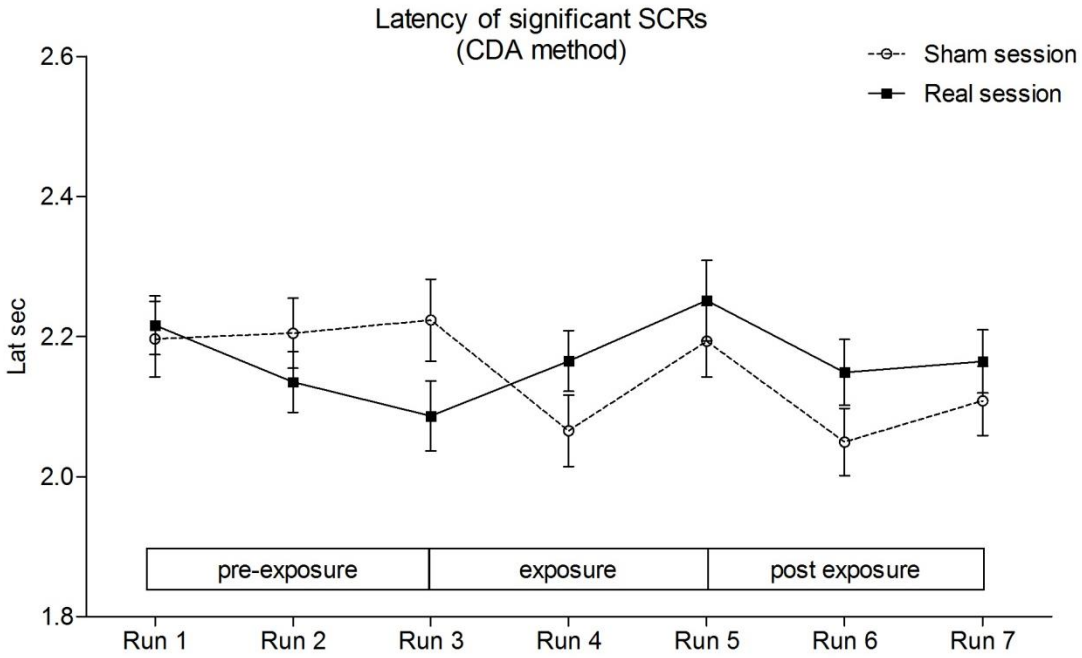

Sum of amplitudes of significant SCRs
(CDA method)
Sham session
Real session
AmpSum µS
0.25
0.20
0.15
0.10
0.05
0.00
pre-exposure
exposure
post exposure
Run 1
Run 2
Run 3
Run 4
Run 5
Run 6
Run 7

Figure 6:


Latency of significant SCRs
(CDA method)
Sham session
Real session
Lat sec
2.6
2.4
2.2
2.0
1.8
pre-exposure
exposure
post exposure
Run 1
Run 2
Run 3
Run 4
Run 5
Run 6
Run 7