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Main Article

Low frequency electromagnetic radiation and hearing

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Abstract

Objective: To analyse the possible impact of low and extremely low frequency electromagnetic fields on the outer hairs cells of the organ of Corti, in a guinea pig model.

Materials and methods: Electromagnetic fields of 50, 500, 1000, 2000, 4000 and 5000 Hz frequencies and 1.5 μT intensity were generated using a transverse electromagnetic wave guide. Guinea pigs of both sexes, weighing 100–150 g, were used, with no abnormalities on general and otic examination. Total exposure times were: 360 hours for 50, 500 and 1000 Hz; 3300 hours for 2000 Hz; 4820 hours for 4000 Hz; and 6420 hours for 5000 Hz. One control animal was used in each frequency group. The parameters measured by electric response audiometer included: hearing level; waves I–IV latencies; wave I–III interpeak latency; and percentage appearance of waves I–III at 90 and 50 dB sound pressure level intensity.

Results: Values for the above parameters did not differ significantly, comparing the control animal and the rest of each group. In addition, no significant differences were found between our findings and those of previous studies of normal guinea pigs.

Conclusion: Prolonged exposure to electromagnetic fields of 50 Hz to 5 KHz frequencies and 1.5 μT intensity, produced no functional or morphological alteration in the outer hair cells of the guinea pig organ of Corti.

Key words: Inner Ear; Cochlea; Electromagnetic Radiation; Guinea Pig

Introduction

Both sound and electromagnetic waves represent different forms of energy propagation. While they have common characteristics, there are also marked differences. Sound waves are transmitted by the vibrations of molecules without any net displacement. The propagation of electromagnetic waves requires no material support and may therefore take place in a vacuum, which is not possible for sound waves. In its zone of influence, a sound wave produces molecular compression and decompression, which is responsible, for example, for the sonic boom produced when the speed of the source exceeds the speed of sound. Electromagnetic waves, for which we follow the description given by Saravi,[1] are oscillations of both electric and magnetic fields, which are perpendicular to each other and perpendicular to the direction of propagation. The speed of propagation of both sound waves and electromagnetic waves depends on the density of the medium (among other parameters), the relationship being direct for sound waves and inverse for electromagnetic waves. The speed of wave propagation in both cases is the product of the frequency multiplied by the wavelength: $v = f \times \lambda$. For both types of wave, higher frequencies correspond to shorter wavelengths. The speed of sound in air, under certain standard conditions of pressure, temperature and humidity, is 344 m/second (m/s); the speed of all electromagnetic waves, such as light, is 300000 km/s in a vacuum.

The amplitude and frequency of sound waves have upper and lower thresholds outside of which one does not speak of hearing since no such sensation is produced. In particular, sound intensities above the maximum hearing level (130 dB HL) will cause humans an unpleasant or painful sensation, and those below the minimum level (0 dB HL) will produce no auditory stimulus at all. With respect to frequency, sounds below the minimum detection threshold of 16 Hz correspond to infrasounds, while those above the maximum threshold of 20 kHz correspond to ultrasounds.

The energy of electromagnetic waves increases with their frequency. There is a reference frequency ($\geq 10^{15}$ Hz) above which electromagnetic waves can

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strip electrons from atoms, leaving the matter ionised. Ionising radiation is the term given to all electromagnetic radiation with frequencies above this reference value, and non-ionising radiation the term for frequencies below this value.[1] The latter includes, in decreasing order of energy, ultraviolet radiation, visible light, infrared radiation, microwaves and the radio frequencies used in mobile telephony. This last type of radiation has provoked increased interest because of the spectacular growth of cell phone communication. Saravi[1] estimated cell phone sales of nearly 800 million in the year 2005 alone, and also estimated total at the end of this year, a concomitant worldwide increase in transmitting antennae of one and a half million.

Currently, Universal Mobile Telecommunication System mobile telephony uses radio frequencies of around 2 GHz. The possible side effects of these radio waves have been studied epidemiologically for almost all organs and systems of the human body, and experimentally in laboratory animals. No clear and credible direct relationship has been found between such waves and any possible functional or organic alterations. Examples of such investigation include functional studies of the human ear using otoacoustic emission techniques[2–4] and laboratory studies of guinea pigs[5] (which found no morphological alterations in organ of Corti specimens).

Such research assesses two types of propagated energy. The first – sound – has a receptor organ, the ear, which has been progressively refined over the course of evolution, so that hearing and sight are now the two principal means of gathering information and determining relationships with the environment. The second type – radio-frequency electromagnetic radiation – has steadily lost importance as a source of information over the course of the phylogenetic progress by which humans evolved. The corresponding field receptors passed early in our phylogeny from the lateral line organs to the utricular and saccular maculae, but the latter now have no receptor electromagnetic radiation function in humans, in whom, so far as is known, there exists no specific element for the detection of electromagnetic fields.

Most previous studies have focused on the high frequencies used by cell phones. However, in the present study we analysed the influence of electromagnetic radiation of frequencies similar to those detected aurally, and assessed the effect of such radiation on the body in general and on the ear in particular (as the representative, in humans, of the organ on which such radiation once acted). With regard to such investigations, it is relevant to cite the work of Simkó and Mattsson,[6] who applied electromagnetic fields of extremely low frequency (50–60 Hz) to *in vitro* cell preparations and found a possible mechanism of stimulation of the cell's immune response. Other authors[7] have found that similar field frequencies, at intensities of 1.0–1.5 μT and exposure times of 24–72 hours, led to a 30 per cent increase in the *in vitro* cell proliferation rate together with deoxyribonucleic acid damage, and that both these phenomena were inhibited by pretreating the cell preparations with antioxidants. Other investigations[8] have found alterations in the function of murine macrophages *in vitro*, using field parameters similar to those described above. In the second case (Ear in particular), experiments on the ear showed that electrical field stimulation of the intact cochlea produced a sensation of an 8-kHz acoustic tone.[9]

Based on the phenomenon of the piezoelectric effect as the mechanism of contraction of the outer hair cells of the organ of Corti,[10] the aim of the present study was to investigate the possible impact of electromagnetic radiation on these cells, using extremely low frequency electromagnetic fields of 50, 500, 1000, 2000, 4000 and 5000 Hz.

Materials and methods

Electromagnetic fields were generated using a transverse electromagnetic wave guide, in which the propagation was in the transversal mode (mode transverse electromagnetic) so that the electric and magnetic components were maintained in a plane perpendicular to the direction of propagation. This allowed us to simulate far-field conditions.

The cell used was a model CC103S, manufactured by IFI (Ronkonkoma, NY, EEUU). This had a useful height in the rehearsal enclosure of 0.3 m, corresponding to a 3.34 cell factor. The load used was a 50-Ω resistance of 500 W dissipation capacity, with a coaxial connection.

The feed to the cell was by means of a generator (Mod 251 TL; California Instruments, San Diego, CA, EEUU, California, USA) with the capacity to provide sinusoidal signals to a maximum of 250 V, at 10 kHz, with an I_{MAX} in these conditions of 3 A.

For all the groups of animals, sinusoidal signals of 200 V_{MAX} (141.42 V rms) were used. This gave the following values in the rehearsal enclosure: electric field strength $E = (141.42/0.3) = 471.40$ V/m; magnetic field strength $H = (471.13/377) = 1.249$ A/m; and magnetic induction $B = \mu H = 4\pi \cdot 10^{-7} \cdot 1.249 = 1.569$ μT.

We used guinea pigs as experimental animals. No distinction of sex was observed. The animals' weights at the beginning of the experiment were between 100 and 150 g. The experimental protocols were approved by the institutional ethics committee for animal research of the University of Extremadura. All animals underwent general and otic examinations, together with functional hearing tests with electric response audiometry. Those animals with abnormal features were excluded.

Table I lists the electromagnetic field frequency to which guinea pigs were subjected, the numbers exposed, the exposure period, the daily exposure time and the total exposure in hours. The exposure period was 180 days for experimental animals subjected to 50 Hz, 500 Hz and 1000 Hz, 165 days for 2000 Hz, 214 days for 4000 Hz, and 390 days for 5000 Hz. The total exposure time was between 3600 and 6240 hours.

These frequency limits (i.e. frequencies from 50 Hz to 5 kHz) were chosen because the first

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TABLE I

EXPOSURE PARAMETERS

EMF freq (Hz)	Animals (*n*)	Exposure		
		Period (days)	Daily (hr/day)	Total (hr)
50	4	180	20	3600
500	4	180	20	3600
1000	4	180	20	3600
2000	7	165	20	3300
4000	8	214	20	4820
5000	7	390	16	6420

EMF freq = electromagnetic field frequency; hr = hours

corresponded to the frequency of the domestic alternating current by which we are constantly surrounded, while the second corresponded to the effective upper limit of the equipment used.

In each group, one animal was used as a control and the others were exposed to the electromagnetic field. Their feeding and housing conditions were identical. Each animal was tagged with an identification number, which was maintained for all functional and morphological data collection procedures.

The animals' hearing levels were determined by a Bio-Logic TE electric response audiometer (Bio-logic Systems Corporation, San Carlos, CA, USA). The surface electrodes were fixed by sutures, with the active electrode in the right auricle, the reference electrode in the shaved scalp vertex, and the guard electrode in the left auricle. The program used was that corresponding to 'brainstem electrical responses', with presentation of alternating polarity clicks as the stimulus. These averaged a total of 2021 per scan. The filters were set at 3 kHz and 100 Hz, the amplifier gain at 10^5 and the analysis period at 10 ms. The maximum permitted resistance in the electrodes was 5 Ω. In all the auditory experiments, the intensities of the stimuli used for each scan were 90, 70, 50, 40 and 30 dB sound pressure level. The stimuli were presented through a conventional earphone 2 or 3 cm from the right ear, with two scans taken for each intensity.

The parameters measured by electric response audiometry included, in addition to the hearing level, the latencies of waves I–IV, the interpeak latency of waves I–III, and the percentage appearance of waves I–III at 90 and 50 dB sound pressure level intensity.

The anaesthetics used were sodium thiopental administered intraperitoneally at a dose of 35 mg/kg, and mepivacaine hydrochloride applied subcutaneously at the sites of electrode implantation.

All the electric response audiometry investigations (except the initial examination of the animal) were carried out at two hours after the last of electromagnetic field exposure. The functional status of hearing was monitored weekly by assessing Preyer's reflex using pure tone stimuli of 8, 6 and 4 kHz supplied by a conventional Maico audiometer, and monthly by electric response audiometry.

One animal per group was left to survive for one month after the last exposure, and was then sacrificed in the same way as the others. The other animals were sacrificed prior to morphological study, following the last hearing level measurement, while they were still sedated. The corresponding control animal was sacrificed on that same day. Samples of the organ of Corti were obtained with the surface specimen technique, and processed following the usual procedure for scanning electron microscopy.

The possible existence of other, non-otic changes caused by the electromagnetic field exposure was checked by general examination and autopsy, focusing primarily on the brain, liver and lungs.

Results

Both the control animals and those exposed to the electromagnetic field exhibited positive Preyer reflexes at 90 or 100 dB HL immediately prior to the last electric response audiometry examination and subsequent sacrifice. Analysis of the electric response audiometry scans of all the animals

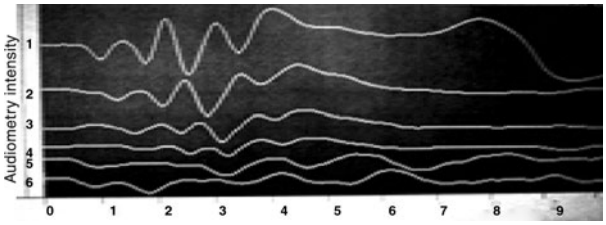


FIG. 1

Electric response audiometry potentials for animal 17 after 3600 hours' exposure to a 2000 Hz electromagnetic field. 1 = 90 dB SPL; 2 = 70 dB SPL; 3 = 50 dB SPL; 4 = 40 dB SPL; 5 & 6 = 30 dB SPL.

TABLE II

WAVE I–IV LATENCIES AND WAVE I–III INTERPEAK LATENCY: 50 HZ ELECTROMAGNETIC FIELD

Latency	90 dB	70 dB	50 dB	40 dB	30 dB
*Control**					
I	1.12	1.36	1.60	1.76	–
II	1.98	2.30	2.62	2.80	–
III	2.46	3.20	3.70	–	–
IV	2.88	–	–	–	–
I–III	1.34				
Animal 2					
I	1.12	1.28	1.58	1.70	–
II	2.04	2.20	3.12	3.22	–
III	2.60	3.86	–	–	–
IV	3.78	–	–	–	–
I–III	1.48				
Animal 3					
I	1.38	1.76	2.00	2.08	2.30
II	2.14	2.88	–	–	–
III	3.04	3.32	–	–	–
IV	3.76	4.02	–	–	–
I–III	1.66				
Animal 4					
I	1.14	1.42	1.88	1.92	–
II	1.94	2.26	2.74	2.84	–
III	–	3.54	3.66	3.76	–
IV	–	4.26	–	–	–
I–III	–				

*Control = Animal #1; I, II, III and IV: Latencies (in m.s.) corresponding to intensities analyzed (in dB); I–III: interpeak latency (in m.s.)

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TABLE III

WAVE I–IV LATENCIES AND WAVE I–III INTERPEAK LATENCY: 500 HZ ELECTROMAGNETIC FIELD

Latency	90 dB	70 dB	50 dB	40 dB	30 dB
*Control**					
I	0.94	1.26	1.52	1.94	–
II	1.74	2.10	2.32	2.48	–
III	2.54	2.82	3.06	3.20	3.38
IV	3.36	3.60	3.88	4.02	–
I–III	1.60				
Animal 6					
I	1.06	1.32	1.64	–	–
II	1.84	2.08	2.50	2.82	–
III	2.54	2.78	3.26	3.40	–
IV	3.54	–	–	–	–
I–III	1.48				
Animal 7					
I	1.04	1.24	1.52	–	–
II	1.84	2.02	2.46	–	–
III	2.62	2.70	3.18	3.24	–
IV	3.50	–	–	–	–
I–III	1.58				
Animal 8					
I	1.00	1.36	1.70	–	–
II	1.80	2.08	2.68	–	–
III	2.52	2.76	–	–	–
IV	3.42	3.74	–	–	–
I–III	1.52				

*Control = Animal #5; I, II, III and IV: Latencies (in m.s.) corresponding to intensities analyzed (in dB); I–III: interpeak latency (in m.s.)

(including controls) taken after their last exposure showed no significant morphological differences. An example is shown in Figure 1, corresponding to the electric response audiometry potentials of guinea pig number 17, after 3600 hours' exposure to a 2-kHz electromagnetic field.

TABLE IV

WAVE I–IV LATENCIES AND WAVE I–III INTERPEAK LATENCY: 1000 HZ ELECTROMAGNETIC FIELD

Latency	90 dB	70 dB	50 dB	40 dB	30 dB
*Control**					
I	1.00	1.20	1.54	1.64	1.70
II	1.80	2.10	2.14	–	–
III	2.64	2.68	2.90	2.98	3.02
IV	3.26	3.50	3.90	3.98	4.20
I–III	1.64				
Animal 10					
I	1.36	1.60	–	–	–
II	2.14	2.34	2.50	–	–
III	2.70	2.88	3.08	3.20	–
IV	3.62	3.84	–	–	–
I–III	1.34				
Animal 11					
I	1.12	1.64	1.82	–	–
II	1.94	–	2.54	–	–
III	2.56	3.02	3.16	3.26	–
IV	3.52	–	–	–	–
I–III	1.44				
Animal 12					
I	1.32	1.94	–	–	–
II	1.88	2.66	–	–	–
III	2.50	3.24	–	3.86	–
IV	3.34	4.26	–	–	–
I–III	1.18				

*Control = Animal #9; I, II, III and IV: Latencies (in m.s.) corresponding to intensities analyzed (in dB); I–III: interpeak latency (in m.s.)

TABLE V

WAVE I–IV LATENCIES AND WAVE I–III INTERPEAK LATENCY: 2000 HZ ELECTROMAGNETIC FIELD

Latency	90 dB	70 dB	50 dB	40 dB	30 dB
*Control**					
I	1.80	2.16	2.46	–	–
II	2.50	3.42	3.80	–	–
III	3.04	4.24	3.80	–	–
IV	3.78	–	–	–	–
I–III	1.24				
Animal 14					
I	1.22	1.58	1.96	–	–
II	2.08	2.26	2.70	–	–
III	2.62	3.02	3.32	3.54	–
IV	3.58	–	–	–	–
I–III	1.40				
Animal 15					
I	1.16	1.46	1.70	–	–
II	2.04	2.30	2.54	2.70	–
III	2.92	3.16	3.36	3.54	–
IV	3.72	4.24	4.38	–	–
I–III	1.76				
Animal 16					
I	1.02	1.20	1.58	1.74	–
II	1.80	2.16	2.32	2.50	–
III	2.54	2.84	3.08	3.32	–
IV	3.48	–	–	–	–
I–III	1.52				
Animal 17					
I	1.06	1.32	1.60	1.78	–
II	1.82	2.10	2.40	2.60	–
III	2.58	2.92	3.16	3.26	–
IV	3.44	3.82	4.08	–	–
I–III	1.52				
Animal 18					
I	1.12	1.36	1.74	1.92	–
II	2.02	2.32	2.72	2.88	–
III	2.72	3.06	3.38	3.52	–
IV	3.66	–	4.32	–	–
I–III	1.60				
Animal 19					
I	1.10	1.54	1.70	1.98	–
II	1.94	2.20	2.54	–	–
III	2.58	3.02	–	3.32	3.66
IV	3.48	–	–	–	–
I–III	1.48				

*Control = Animal #13; I, II, III and IV: Latencies (in m.s.) corresponding to intensities analyzed (in dB); I–III: interpeak latency (in m.s.)

Tables II to VII show values for wave I–IV latencies and the wave I–III interpeak latency. We observed no significant differences between the controls and the study animals in any group.

Most of the animals' hearing levels increased by 10–20 dB SPL compared with values at the beginning of the experiment. However, this was also the case with the controls, except those in the 500 and 1000 Hz groups (animals five and nine).

Waves I and III at 90 dB SPL appeared in all the animals, both controls and study animals, with the exception of animal number four. Wave I at 50 dB SPL was identified in all the controls, but in the study group it had disappeared in animals 10, 12, 23 and 30. Wave III at 50 dB was present in all the controls, and in all but seven of the study animals (these being animals two, three, eight, 12, 19, 24 and 31). Of these seven, wave III was again identified in the

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TABLE VI

WAVE I–IV LATENCIES AND WAVE I–III INTERPEAK LATENCY: 4000 HZ ELECTROMAGNETIC FIELD

Latency	90 dB	70 dB	50 dB	40 dB	30 dB
*Control**					
I	0.92	1.24	1.56	–	–
II	1.72	2.10	2.36	2.46	–
III	2.52	2.86	3.16	3.24	–
IV	3.34	3.62	4.06	4.06	–
I–III	1.60				
Animal 21					
I	1.14	1.40	1.54	–	–
II	1.94	2.36	2.44	2.60	–
III	2.72	3.14	3.20	3.32	–
IV	3.64	3.88	4.18	–	–
I–III	1.58				
Animal 22					
I	1.14	1.52	1.82	–	–
II	1.92	2.26	2.56	3.22	–
III	2.60	3.24	3.20	4.20	–
IV	3.48	–	–	–	–
I–III	1.46				
Animal 23					
I	0.98	1.14	–	–	–
II	1.74	1.94	2.46	3.04	–
III	2.52	2.74	3.10	–	–
IV	3.40	3.60	–	–	–
I–III	1.54				
Animal 24					
I	0.94	1.22	1.54	–	–
II	1.68	2.10	2.36	2.54	–
III	2.40	2.84	–	–	–
IV	3.30	3.70	–	–	–
I–III	1.46				
Animal 25					
I	0.98	1.18	1.44	1.64	–
II	1.78	2.04	2.24	2.40	–
III	2.50	2.76	2.94	3.10	3.26
IV	3.36	3.60	3.84	–	–
I–III	1.52				
Animal 26					
I	1.02	1.16	1.58	–	–
II	1.78	1.90	2.22	–	–
III	2.54	2.64	3.20	–	–
IV	3.42	3.54	3.94	4.14	–
I–III	1.48				
Animal 27					
I	1.82	2.22	2.50	2.72	–
II	2.52	2.96	3.22	3.44	–
III	3.44	3.80	4.12	4.36	–
IV	–	–	–	–	–
I–III	1.62				

*Control = Animal #20; I, II, III and IV: Latencies (in m.s.) corresponding to intensities analyzed (in dB); I–III: interpeak latency (in m.s.)

TABLE VII

WAVE I–IV LATENCIES AND WAVE I–III INTERPEAK LATENCY: 5000 HZ ELECTROMAGNETIC FIELD

Latency	90 dB	70 dB	50 dB	40 dB	30 dB
*Control**					
I	1.02	1.36	1.54	1.58	–
II	1.88	2.18	2.30	2.42	–
III	2.58	2.82	2.98	3.14	–
IV	3.44	3.72	3.88	4.04	–
I–III	1.56				
Animal 29					
I	1.60	1.84	2.14	1.82	–
II	2.34	2.60	–	–	–
III	3.04	3.22	3.54	–	–
IV	3.98	4.18	–	–	–
I–III	1.44				
Animal 30					
I	1.24	1.78	–	–	–
II	2.30	2.44	–	–	–
III	3.42	3.96	4.24	4.34	–
IV	4.66	–	–	–	–
I–III	2.18				
Animal 31					
I	1.04	1.22	1.42	1.78	–
II	1.90	2.12	2.74	2.84	–
III	2.70	2.80	–	–	–
IV	3.52	3.80	–	–	–
I–III	1.66				
Animal 32					
I	1.02	1.24	1.60	1.66	–
II	1.82	2.08	2.42	2.44	–
III	2.62	2.80	3.18	3.20	–
IV	3.52	3.78	4.02	4.08	–
I–III	1.60				
Animal 33					
I	1.08	–	1.62	1.72	–
II	1.82	2.04	–	–	–
III	2.60	3.12	3.14	3.18	–
IV	3.40	–	–	–	–
I–III	1.52				
Animal 34					
I	1.10	1.48	1.76	1.80	–
II	1.88	2.22	2.38	2.54	–
III	2.52	2.98	3.04	3.16	–
IV	3.44	–	4.00	4.16	–
I–III	1.42				

*Control = Animal #28; I, II, III and IV: Latencies (in m.s.) corresponding to intensities analyzed (in dB); I–III: interpeak latency (in m.s.)

40 dB SPL intensity scan for animal 12, and in the 40 and 30 dB SPL scan for animal 19, while the remaining five animals presented no further wave for these lower intensities.

Scanning electron microscopy of organ of Corti specimens from each animal showed no alteration in any cochlear turn. An example of the specimen from animal 17 is shown in Figure 2.

Discussion

It has long been suspected that the physiological processes governing the function of the organ of Corti differ from those proposed by Békésy in 1960.[11] These processes have been termed the 'second filter'. It has been demonstrated[12] that the inner hair cells have a frequency spectrum analogous to that of the acoustic or VIIIth cranial nerve, evidence for the role of a 'second filter' at the peripheral level.

It has been proposed[13] that there must exist some active mechanism in the inner ear responsible for the production of sounds (otoacoustic emissions) that can be registered at the level of the external auditory canal, and which appeared in response to short auditory stimulations of that same ear.

Previous results[14] have shown that, as well as adrenergic innervation, the organ of Corti receives sensory input via afferent and efferent nerve fibres within the central nervous system. The efferent nerve fibres are distributed mainly among the outer hair cells, with which they make synaptic contacts by means of the nerve endings identified by Engström[15] (the morphological aspects of which have since been extensively studied).[16] Functionally,

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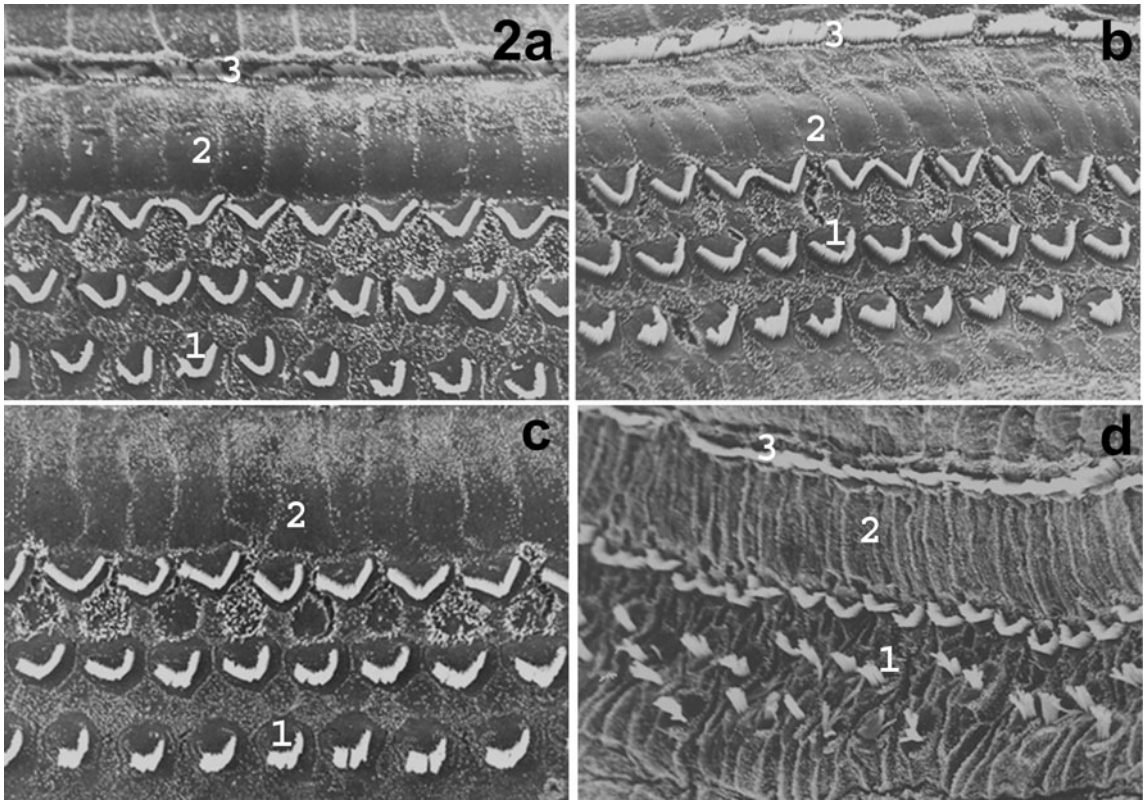


FIG. 2
Scanning electron photomicrograph of the organ of Corti of animal 17. (a) Basal turn; (b) second turn; (c) third turn; (d) apical turn. 1 = outer hair cells; 2 = pillar cells; 3 = inner hair cells (×450)

the medial efferent fascicle carries signals capable of influencing the sensitivity of the peripheral auditory system, specifically with respect to frequency, through its action on the dynamics of the basilar membrane (analysed by means of *in vivo* studies).[17]

Isolated outer hair cells respond to electrical stimuli with contractions, presumably due to the presence of subsurface cisternae on their lateral faces.[18] This electromotility, together with the peculiar distribution of the synaptic contacts and other morpho-functional features of the outer hair cells, forms part of the morphological substrate of what has come to be known as cochlear micromechanics. This is considered to be responsible for an active process of cochlear amplification, and is thought to participate in enhancing the organ of Corti's auditory sensitivity and capacity for frequency analysis – in other words, to act as a 'second filter'.

Given this situation, studies of cochlear micromechanics from the 1990s onwards have focused on possible mechanisms of action and regulation. Different investigative techniques have been used, but all have focused primarily on isolated outer hair cells. Mammano and Ashmore[19] recorded the electrical activity of outer hair cells in the four turns of the intact guinea pig cochlea, and also evaluated the effectiveness of their procedures for predicting *in vivo* function. Nevertheless, we can find no reliable basis on which to compare these authors' *in vitro* results with our own *in vivo* results obtained from animals exposed to electromagnetic fields.

Other investigators have recorded the movements associated with outer hair cell membrane motility, [20] and have established that electromagnetic fields act on the cell via the actin filaments of the microvilli.[21]

The force exerted by outer hair cells closely follows their membrane potential up to 60 kHz, indicating that 10–13 kHz may be the limits for outer hair cell electromotility in mammals.[22] Except for those of the basal turn, the outer hair cells need no additional mechanism to trigger electromotility. Outer hair cells of the basal turn may have their electromotility frequency limited by ion channels. Such data on the lower limits of electromagnetic field frequencies capable of simulating the organ of Corti are consistent with our own results. It has been previously associated[23] the regulation of the electromotility of the OHC with the reorganization of the cytoskeleton, without relation to changes in the execution of the membrane's molecular motors.

Furthermore, studies based on isolated outer hair cells stimulated with currents that were longitudinal or transversal relative to the cells' major axes[24] have observed resonant frequencies of approximately 13 kHz and 1 MHz, respectively, demonstrating a piezoelectric effect in these cells and confirming other authors' conclusions.[10] Another possible mechanism of electromotility regulation may involve modification of the 'plasma membrane molecule motors'; investigators have observed alterations of intracellular pressure and chloride concentration,[25] and differences in outer hair cell electromotility at

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TABLE VIII

ACCEPTED REFERENCE VALUES FOR GUINEA PIG AUDITORY WAVE LATENCIES

Wave	M	LLN	ULN	I–III
90 dB				
I	1.41	0.9	2.23	1.83
II	2.23	1.35	3.10	
III	3.28	2.45	4.11	
IV	4.40	3.31	5.48	
70 dB				
I	1.50	0.60	2.40	1.99
II	2.39	1.58	3.20	
III	3.46	2.59	4.34	
IV	4.53	3.54	5.53	
50 dB				
I	1.60	0.80	2.40	2.25
II	2.55	1.76	3.33	
III	3.73	2.74	4.41	
IV	4.54	3.54	5.52	
30 dB				
I	1.71	0.67	2.76	2.08
II	2.68	1.64	3.72	
III	3.82	2.76	4.48	
IV	4.58	3.74	5.42	

M = arithmetic mean latency; LLN = latency lower limit of normality; ULN = latency upper limit of normality; I–III = wave I–III interpeak latency

the basal and apical cochlear turns.[26] The former showed rapid activation of ion currents, which was absent in the latter. Moreover, it has been reported that the electromotility of the outer hair cells makes them capable of generating a force for each frequency, with a repercussion on the basilar membrane of similar magnitude.[27]

The maximum electromagnetic field intensity used in the current study, as measured by magnetic induction, was an order of magnitude below the limits set in 1999 by the European Economic Community as dangerous (i.e. 16 μT for frequencies 0–50 Hz and 10 μT for 50–1000 Hz). This level was chosen because the aim of the study was to detect the minimum intensities which were capable of stimulating (and thus, hypothetically, damaging) certain groups of hair cells, when the ear was subjected to prolonged exposure to those intensities. Accepted reference values for the auditory response are listed in Table VIII.

- **This study investigated the effects on the outer and inner hair cells of the organ of Corti of electromagnetic radiation of very low or low frequency and intensity**
- **The study used a guinea pig model to investigate the potential effect of electromagnetic fields of frequencies ranging from 50 to 5000 Hz**
- **Prolonged electromagnetic field exposure for periods roughly equivalent to a third of the average human lifespan produced no functional or morphological alteration in the hair cells of the guinea pig organ of Corti**

When our results for wave I–IV latencies in the different frequency groups (and their respective control animals) were compared with the reference values listed in Table VIII, it was clear that our results were all within normal limits. Our wave I–III interpeak latency values were also within the normal range, which at 90 dB SPL is 0.84–2.82 ms with a mean of 1.83 ms (G. Barrantes, unpublished data).

With respect to hearing levels, practically all our animals showed a loss of 10 dB SPL compared with values at the beginning of the experiment. This small loss was also found in the control animals, indicating that it was not an effect of electromagnetic field exposure.

In reference guinea pigs (G. Barrantes, unpublished data), waves I and III are recognised at 90 and 50 dB SPL in 100 per cent of cases. This was also the case in all our control animals. However, seven of our animals exposed to electromagnetic fields did not present these waves. Five of these had a wave III response cut-off at 70 dB SPL. These cases represent significant hearing loss which could have been related to electromagnetic field exposure. Nevertheless, this finding showed no specificity for electromagnetic field frequency level, as it was observed in at least one animal in all the frequency groups, and in two animals in the 50 Hz group. It could be hypothesised that this possible but inconstant influence of electromagnetic fields on auditory response could have resulted from differences in individual animals' sensitivities. The opposing argument would regard such an effect as unlikely, given the lack of any proportional relationship with total exposure times, especially as these covered a fairly wide range.

On scanning electron microscopy of organ of Corti specimens, we noted marked irregularity in the arrangement of the outer hair cell cilia bundles in the apical turn, compared with the rest of the turns; however, this appearance was compatible with normality.

Conclusion

In the current study, prolonged electromagnetic field exposure for periods roughly equivalent to one-third of the average human lifespan, using frequencies from 50 Hz to 5 kHz and field intensities of 1.5 μT, produced no functional or morphological alteration in the outer hair cells of the guinea pig organ of Corti.

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