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Review

The Effects of Low-Frequency Environmental-Strength Electromagnetic Fields on Brain Electrical Activity: A Critical Review of the Literature

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Reports dealing with the stimulus-response relationship between low-level, low-frequency electromagnetic fields (EMFs) and changes in brain electrical activity permit assessment of the hypothesis that EMFs are detected by the body via the process of sensory transduction. These reports, as well as those involving effects on brain activity observed after a fixed time of exposure, are critically reviewed here. A consistent stimulus-response relationship between EMFs and changes in brain activity has been demonstrated in animal and human subjects. The effects, which consisted of onset and offset evoked potentials, were observed under conditions permitting the inference that the fields were transduced like ordinary stimuli such as light and sound. However, unlike the changes in brain activity induced by these stimuli, the changes induced by EMFs were governed by nonlinear laws. The studies involving attempts to determine whether a period of EMF exposure caused a metabolic effect reflected in pre-exposure/post-exposure differences in brain activity were generally inconclusive.

Keywords Electromagnetic field; Brain electrical activity; Nonlinear analysis; Electroencephalogram; Evoked potentials; Recurrence analysis.

Introduction

Concern regarding the impact of environmental-strength electromagnetic fields (EMFs) on the nervous system arose independently from two strikingly different research initiatives. Referring to research he had done from 1965–69 to help explicate the Soviet microwave irradiation of the American embassy in Moscow and the results in several published reports (Gavalas et al., 1970; Bawin et al., 1973), Ross Adey said:

> My colleagues and I have observed the effects of weak electric and electromagnetic fields on the behavior of man and animals, and we have correlated these observations with neurophysiological effects and brain

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> chemistry. The most striking conclusion drawn from these observations is that mammalian central nervous functions can be modified by electrical gradients in cerebral tissue substantially less than those known to occur in postsynaptic excitation, and also substantially smaller than those presumed to occur with inward membrane currents at synaptic terminals in release of transmitter substances. (Adey, 1976)

During the same time period, Robert Becker sought to understand the role of endogenous electrical signals in the control of tissue regeneration; in 1972, after summarizing his work he said:

> I also feel concern for a much broader problem, which is the continuous exposure of the entire North American population to an electromagnetic environment in which is present the possibility of inducing currents or voltages comparable with those now known to exist in biological control systems. (Becker, 1972)

The idea they had in common was that man-made EMFs might interfere with the electrical signals that governed the body's regulatory systems, like sand in the gears of a machine, thereby promoting human disease.

In 1980, Becker and Marino presented a general theory of the link between EMFs and disease, based on a putative electrogenic protein in excitable cells whose functional state was altered by the presence of weak pericellular EMFs (Fig. 1)

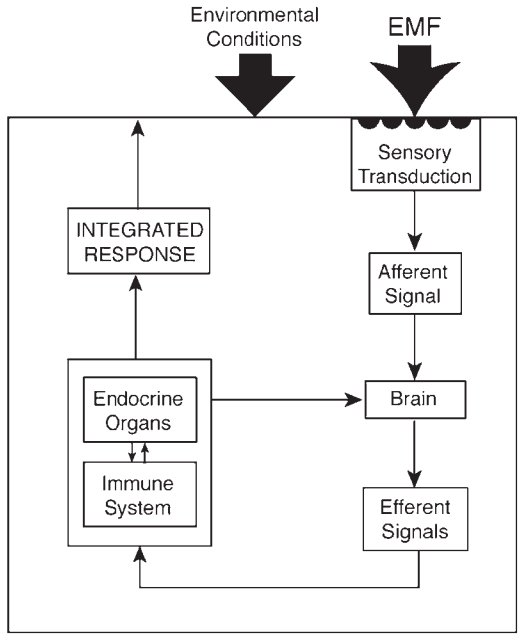

Figure 1. The proposed control system that mediates EMF-induced biological effects. The field is transduced and the resulting signal is cognitively processed thereby permitting the brain to initiate and regulate the appropriate adaptive physiological responses. A key observable in the theory is the stimulus-response relationship formed by onset of the EMF and subsequent deterministic changes in brain electrical activity.

(Becker and Marino, 1982). As theorized, a resulting subthreshold change in membrane potential led to an afferent signal, cognitive processing, and efferent signals to the body's effector systems. In this view, the reported links between EMFs and effects in body tissues (and ultimately human disease) were indirect and stemmed from overtaxing the body's sensory and compensatory mechanisms (excess biological stress).

The theory predicted that the onset of an EMF almost immediately triggered alterations in brain electrical activity. Consequently, evidence of a stimulus-response relationship between presentation of an EMF and changes in brain electrical activity would support the validity of the initial stages of our theory (Fig. 1). Our purpose here is to describe and evaluate the literature pertinent to the existence of such a relationship. Only studies dealing with the effects of low-frequency EMFs on brain electrical activity will be discussed; effects due to high-frequency EMFs (mobile phones) were reviewed elsewhere (Carrubba and Marino, In press).

Methods

We searched electronic databases (PubMed, Science Citation Index) using various combinations of an electrical term (field, electromagnetic, electric, magnetic, ELF, nonionizing, DC, AC), a device (high-voltage powerlines, electrical appliances), and an outcome (electroencephalogram, evoked potentials, brain electrical activity) to identify English-language studies that involved the effects of low-frequency, non thermal EMFs on the brain electrical activity of humans or animals. The inclusion criteria were: (1) a reasonable description of the experimental conditions; (2) use of a control group; and (3) statistical evaluation of the data. The exclusion criteria were: (1) the use of thermal EMFs; and (2) application of electrical energy by means of surface electrodes rather than fields. All other factors including blinding of study participants, counter-balancing of experimental conditions, performance of sham studies, and corrections for multiple comparisons were considered with regard to the weight given to the study rather than to its admissibility as evidence of the ability of EMFs to affect brain activity.

Linear Studies

Animal Studies

Bell et al. (1992b) statistically compared brain electrical activity from rabbits in the presence and absence of fields, using spectral analysis. The EMFs studied were: (1) 1 G, 5 Hz (a prominent frequency in the rabbit brain); (2) 0.64 G, 25 Hz (the ion-resonance conditions for K^+); (3) 1 G, 25 Hz (a field whose suspected physiological significance was that it was a nospecific stressor). Each rabbit was exposed to the three fields, a light stimulus (positive control), and a sham stimulus (negative control) in one experimental session, and each test session was repeated ($\geq$1 day between replications). The fields were uniform throughout the animal's body, thereby permitting an accurate characterization of its strength at the location of the electrogenic protein, wherever it occurred.

We avoided the use of ANOVAs to obviate the possibility that averaging the results across the subjects might obscure a real effect if the subjects reacted differently

from one another. Instead, we acquired multiple independent trials of brain activity, each containing a stimulus (or sham stimulus) and control epoch, and the effect of the stimulus was evaluated in each animal. The stimulus was applied for 2-s epochs, with a variable inter-stimulus period (5–11-s, varied randomly). The first 30 ms of each field-exposure epoch was removed to eliminate the field-onset spike in the electroencephalogram (EEG) and the remaining signal was filtered at 0.3–35 Hz; these precautions were taken in all subsequent studies involving the effects of fields on brain electrical activity (Bell et al., 1991, 1992a, 1994a,b, 1996; Marino et al., 1996, 2002, 2003, 2004; Carrubba et al., 2006, 2007a,b, 2008). Fourier transforms were performed on all 2-s stimulus epochs and their corresponding control epochs (the 2-s period immediately preceding the stimulus) (N = 200). The Fourier analysis of each epoch yielded 39 dependent variables consisting of the power at 1–20 Hz (units of μV^2) in increments of 0.5 Hz, each of which was compared between the stimulus and control epochs under conditions such that the family-wise error rate for the decision that a rabbit detected the field was $p < 0.05$.

Sixty-seven percent of the rabbits detected the light stimulus and none detected the sham stimulus. All of the rabbits tested detected the 1-G, 5-Hz magnetic field, but not the other two fields. The rapidity and circumstances of the effect (observed during 2 s of stimulus presentation in the context of multiple independent trials on the same animal) suggested that the effects were a result of sensory transduction (because other known forms of signal detection would have been too slow). When the EEG measurements were repeated after the rabbits had been killed, the results showed that the 5-Hz effect could not be attributed to an induction artifact.

A major shortcoming of the study was the assumption that any real effect in any specific animal was necessarily consistent. The assumption was made tacitly when we chose to use the *t* test to compare the average spectral power between groups. Mathematically, the assumption was that any change in the Fourier coefficient at a particular frequency would be more or less identical from trial to trial. The problem generated by this assumption was ultimately appreciated and eliminated (see the Nonlinear Studies section below).

Normal Human Subjects

Our initial human studies were performed to determine whether frequency-specific responses also occurred in the human brain during EMF exposure (Bell et al., 1992a). We measured the electroencephalogram (EEG) from C3, C4, P3, P4, O1, and O2 (International 10–20 System, referenced to linked ears) in 19 subjects; this electrode configuration was used in all subsequent human studies (Bell et al., 1991, 1994a,b; Marino et al., 1996, 2004; Carrubba et al., 2007a,b, 2008). As with the rabbits, each subject served as his own control, the spike artifact in the EEG due to field onset was eliminated, and the results were protected against family-wise error. Using 0.2 and 0.4 G at 1.5 and 10 Hz, we found altered brain activity at the stimulation frequency during exposure in each subject. The effect was more likely at 10 Hz compared with 1.5 Hz, and more likely at 0.4 G compared with 0.2 G (Bell et al., 1992a).

To study the effect of a field whose frequency was not significantly present in the EEG, we exposed subjects to 250–500 mG, 35–40 Hz for 2-s epochs (inter-stimulus period 5–11 s), and compared the spectral power measured during exposure with that measured during the inter-stimulus period in 50 independent trials for each subject (Bell et al., 1991). The control for each field epoch was the immediately preceding 2-s

period. The criterion for concluding that a subject had detected the field was that it produced at least 2 bilateral successes (statistically significant difference between exposed and control epochs) in at least one pair of electrodes, provided that those changes were in the same direction (family-wise error $p < 0.02$). We found that 7 of 14 subjects responded to the EMF, as evidenced by statistically reliable changes in the spectral power at specific frequencies. No false-positive results were seen when the entire procedure was repeated using sham exposure.

The 50% detection rate for a field that had no physiological significance generally supported our theory (Fig. 1), but raised the question of why half the subjects had apparently not responded to the field. We therefore performed replicate studies but using 60 Hz, which we reasoned might yield a higher detection rate because the population has been preconditioned to fields at this frequency since the development of commercial power systems (Bell et al., 1994a). We employed 0.78 G, 60 Hz, in the presence and absence of 0.78 G, DC. Each of 20 subjects underwent a block of trials involving exposure to the DC field (B_{DC}), the 60-Hz field (B_{AC}), combined fields (B_{DC+AC}), and a sham field. A trial consisted in the presentation of the field for 2 s, followed by a 5-s field-off interval; the control epoch for each field epoch was the immediately preceding 2-s interval.

The Fourier coefficients at 1–18 Hz were analyzed; the criterion for accepting an effect due to the presentation of the field was that it resulted in at least 2 bilateral successes in at least one pair of electrodes (family-wise error rate, $p = 0.04$). B_{DC}, B_{AC}, and B_{DC+AC} were detected by 7, 15, and 13 subjects, respectively. Overall, 19 of the 20 subjects tested responded to at least one of the fields studied. Both increases and decreases in field-induced activity were observed, depending on the Fourier frequency. No effects occurred with the sham field.

A major question raised by the two previous studies (Bell et al., 1991, 1994a) involved the interpretation of the negative results that occurred in 35% ([7 + 5]/[14 + 20]) of the subjects studied. One possibility was that the non responders were inherently insensitive to the field (true negatives), thereby suggesting that sensitivity to EMFs was not a general human trait. In our next study, therefore, we measured the false-negative rate of our method for detecting stimulus-induced effects in the EEG, using the reaction to light as the gold standard (Marino et al., 1996).

Each subject underwent a block of trials that included stimulus (either light or magnetic fields) and control epochs. A trial consisted of the presentation of a stimulus (or a sham) for 2 s, followed by a 5-s stimulus-free interval. Only 11 of 28 subjects detected the light ($p < 0.05$ for each subject), whereas all the subjects reported that they had seen the light, which necessarily implied that brain electrical activity had been altered. Thus, the results indicated that the false-negative rate of the method when used to detect light-induced changes in the EEG was 61% (Marino et al., 1996). In 19 other subjects, 11 detected 0.8 G (either 1.5 or 10 Hz) corresponding to a non response rate of 42%. Overall, these results indicated that the true detection rate for low-frequency EMFs was probably higher than the 50–75% that we had observed in our studies.

Several additional reports involved the sensory response of the human brain to EMFs (Heusser et al., 1997; Lyskov et al., 2001; Stevens, 2007). A composite EMF stimulus (17 mG, 8–12 Hz, and 25 mG DC) decreased the global field power (a measure roughly equivalent to the spatial standard deviation of the EEG from 12 scalp locations) during field exposure in a study group of 20 subjects ($P = 0.06$, Wilcoxon signed rank test) (Stevens, 2007).

In a second study, the relative spectral power measured in 62 subjects during exposure to 350 mG (rms), 3 Hz, was compared with pre-exposure levels (Heusser et al., 1997). The induction artifact associated with application of the field was minimized by slowly ramping the field, which was applied for 20 min. Comparisons were made between the pre-exposure levels, each of four 5-min successive intervals during exposure, and the 5-min period following cessation of exposure. Of the 30 planned comparisons (5 E-C conditions $\times$ 2 electrode locations (left and right side of the head) $\times$ 3 frequency bands (theta, alpha, beta)), 4 were statistically significant. The probability of 4 successes due to chance (pair-wise significance $p < 0.05$) in 30 tests is $p = 0.06$. Consequently, as the authors recognized, the results may or may not indicate the true occurrence of a field-induced change in the EEG. Moreover, since most of the significant tests were associated with $\geq$20 minutes' exposure, they were probably not relevant to sensory (as opposed to general metabolic) changes in the EEG.

The study (Heusser et al., 1997) illustrated the quandary faced by an investigator who does not have a hypothesis regarding the nature of the effects of EMFs on the EEG. In such cases the experimental plan invariably calls for the performance of numerous statistical tests involving multiple dependent parameters that characterize brain activity. Usually, some tests are pair-wise significant but their meaning is dubious because of a lack of protection against family-wise error (the alternative explanation that the pair-wise significant effects were all due to chance). Even worse, if one regards the family-wise error rate as sufficient for indicating the occurrence of an effect, it is not possible to identify specific effects. In the present study, for example, the significances occurred in theta on the left side after 5-min exposure, in beta on the right and left side after 20-min exposure, and in beta on the right side following exposure. Even if one could validly conclude that brain electrical activity was affected, there would be no way to decide exactly what was affected or when.

In a third study, the relative spectral power in the resting EEG was unaltered during exposure to 100 mG, 60 Hz in normal subjects and in self-selected electrically hypersensitive subjects (Lyskov et al., 2001); the results were also negative when the exposures were repeated while the subjects performed an arithmetic task. The small applied field, non stationarity of the EEG, inter-subject variations, and the use of a 3-way ANOVA, individually and in combination are reasonable explanations for the consistent negative results.

As part of the power industry's assessment of potential health risks due to the electric and magnetic fields of high-voltage powerlines, investigators studied the effect of these fields on evoked potentials in subjects who were simultaneously exposed to visual or auditory stimuli (Graham et al., 1987; Cook et al., 1992; Graham et al., 1994). In one study, 9 kV/m and 200 mG were applied together while the subjects were presented with visual or auditory stimuli in the context of target-detection tasks (the oddball paradigm) (Cook et al., 1992). The visual stimulus was light from a red/green light-emitting diode, and the auditory stimulus consisted of high- and low-pitched tones; the stimuli (50 ms in duration) were presented 140 times, and the EEG from C_Z (10–20 System) was averaged to characterize the auditory (AEP) and visual (VEP) evoked potentials. The infrequent target stimuli (20% high tones or red lights) were randomly interspersed among the non target stimuli (80% low tones or green lights), and the amplitude and latency of the P300 wave of the evoked potential were computed for each sensory modality for target and non target stimuli, before, during, and after both field exposure and sham exposure. The field altered the non target AEP amplitude during and after exposure, but had no effect on latency. There was no

effect on VEP amplitude either during or after field exposure, but there was a reduction in latency during field exposure (Cook et al., 1992).

In another study, three groups of subjects (18 per group) were exposed to 6 kV/m and 100 mG, 9 kV/m and 200 mG, 12 kV/m and 300 mG, respectively (Graham et al., 1994). Significant alterations in the latency and amplitude of the AEP were found in the low- and medium-strength fields, but not in the high-strength field. However, the effects occurred at N200-P300 in the evoked potential, not at P300 as in the earlier study (Cook et al., 1992).

Counting their initial unpublished results (Graham et al., 1987), the investigators reported some kind of field-induced effect on brain potentials evoked by light or sound in three studies. However, several factors undercut the reliability of their observations. First, the data was embedded in a highly complex set of screening studies involving numerous neurophysiological parameters, and it is difficult to have confidence that their post-hoc data-mining approach yielded anything other than chance associations. On the other hand, roughly parallel changes were observed in three separate experiments, and the work was performed under contract to industry-related groups (which would be predisposed in favor of negative data). Consequently, the experiments probably furnish modest support for the proposition that field exposure affected cognitive brain processing as reflected in changes in brain potentials evoked by other stimuli.

Patients with Epilepsy

If brain electrical activity is altered in the presence of an EMF, it is reasonable to suspect that the effect would occur in subjects with epilepsy because their brain electrical activity is labile and vulnerable to changes caused by imperceptibly subtle internal and external factors. This possibility was investigated in a series of studies involving exposure of subjects with mesial temporal lobe epilepsy (MTLE) who were stimulated with 1–40 G (Fuller et al., 1995, 2003; Dobson et al., 2000a,b). The fields were applied for a fixed interval in multiple independent trials, and the distributions of the anomalous spikes characteristic of epilepsy that occurred during the 10-s intervals before and after the exposure interval were compared (Fig. 2a). In the first study, 6 of 7 subjects showed significantly higher levels of epileptiform activity following field exposure (Fig. 2b) (Fuller et al., 1995). Similar results were claimed in a second study, but the experimental protocols were complex (many combinations of field strengths, durations of field exposure, and times between independent trials), and only 3 subjects were studied (Dobson et al., 2000a). In a third study, 5 of 10 subjects suffering from MTLE exhibited a significant ($p < 0.05$) response to the field (Dobson et al., 2000b). When these results were averaged over all the subjects, no field effect was found. In a fourth study, an increase in epileptic activity following field exposure was observed in 1 of 3 subjects, and a progressive increase in epileptic activity may have occurred during what had previously been assumed to be independent trials (Fuller et al., 2003).

The investigators concluded that they had demonstrated field-induced changes in the EEG from MTLE patients, and that may indeed have been the case. However, they did not discuss the limitations of their conclusion. First, although they used scalp electrodes, it appears their most quantitatively reliable data was obtained from implanted electrodes that monitored the hippocampus. The possibility that the observed effects arose from current induced in the electrodes and delivered deep in the

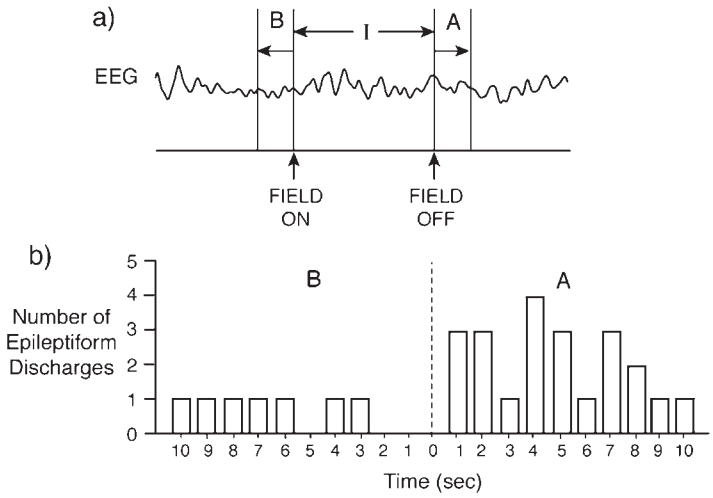

Figure 2. Exposure of a subject with mesial temporal lobe epilepsy to DC magnetic fields. (a) Experimental design. The subject was exposed to the field for a predetermined interval (I) and the epileptiform activity that occurred during the 10-s intervals before (B) and after (A) exposure were compared in multiple independent trials in each subject. (b) Typical result, showing the number of epileptiform discharges in A and B in subject WB (Fuller et al., 1995); control activity is shown for the 10-s interval prior to field application (cumulative results for 27 trials).

brain was not discussed. The authors manually switched on and off the magnetic field for the express purpose of minimizing consequences of Faraday induction but they neither evaluated the effectiveness of this precaution nor applied it in all experiments.

The investigators used terms "increased" and "decreased" to refer to statistical decisions ($p < 0.05$) and also to quantitative data that had not been evaluated statistically, which sometimes makes it difficult to ascertain which inferences were justified statistically. Also, they employed complex exposure protocols involving different field strengths but did not explain why they thought that the results could be combined for analysis.

The authors favored the view that the effect on epileptiform activity arose not from the presence of the field but from the fact that it was switched on and off. However, given their experimental design (Fig. 2a), it is equally possible that the effect could have been due (wholly or partly) to the presence of the field. In the study in which both individual and group analyses were performed (Dobson et al., 2000b), they found that the individual effects were averaged away when the subjects were analyzed as a group. This result is consistent with an inference that the effects were nonlinear, but it could also be explained by assuming that the effects were linear but weak, and hence were not averaged away but simply diluted by the 50% of the subjects who did not show statistically significant results.

Nonlinear Studies

Initial Reports

The principal shortcoming in the studies described above was the assumption by the investigator, almost always made tacitly, that any real effect associated with

presentation of an EMF stimulus would be consistent from trial to trial. When we recognized that this assumption was unwarranted and probably incorrect, we began analyzing the effects of EMFs on brain electrical activity using mathematical tools that had been developed for studying nonlinear systems. These methods, phase-space embedding followed by calculation and quantitation of the corresponding recurrence plot, permitted us to capture the deterministic activity in the EEG caused by the stimulus (regardless of whether it was an increase or a decrease) prior to comparing means in a statistical analysis. The basic mathematical techniques and the tailoring necessary to apply them to the analysis of the EEG are described in detail elsewhere (Carrubba et al., 2006).

Rabbits were exposed to 2.5 G, 60 Hz, using a set of coils that ensured the field was uniform throughout the animal's environment (Marino et al., 2002). The field was applied for 2 s (E epoch) followed by a field-free period of 5 s (minimum of 60 trials). The procedure was repeated using light as the stimulus (positive control). The signal from the last 2 s of each trial was used as the control (C) epoch for the corresponding E epoch. The signal from the 2 s preceding the C epoch was defined as the sham (S) epoch and was analyzed statistically relative to C to evaluate the possibility that any positive results might be attributable solely to our analytical method.

The induction artifacts (approximately 30 ms at onset and offset of the field) and trials containing movement artifacts were removed from the recorded voltage. The remaining trials were embedded in a 5-dimensional space with a time delay of 1 point. Recurrence plots were then produced for the E, S, and C epochs in each trial, and the plots were quantitated using percent recurrence (%R) and percent determinism (%D), which, respectively, represent the number of recurrence points in the plot and the percentage of the recurrent points that fell along diagonal lines (Webber and Zbilut, 1994).

We first evaluated the data from one rabbit, using a discriminant procedure to optimize our ability to detect an effect. Corresponding segments of the stimulus and control epochs of the EEG (E and C, respectively) were systematically compared using the Wilcoxon signed rank test to identify the portion of the signal that was most responsive to the field (window). In this manner we localized the effect of the field to a 250-ms segment centered at 250 ms after commencement of the field. In a similar manner, we determined that the response to the light stimulus occurred within a 266-ms interval centered at 175 ms after the light was applied.

The windows in the E epoch thus identified were studied prospectively in nine additional rabbits to analyze the effect of application of each of the stimuli. In each case the nonlinear quantifiers (%R and %D) were significantly greater (family-wise $p < 0.05$) in the E epoch segment, and there were no cases of false-positive results (assessed by comparing the sham and control segments) (Marino et al., 2002). The entire experiment was repeated three times for each rabbit and the results were identical.

To study the effect of the level of consciousness on the ability of the stimuli to affect brain electrical activity, we repeated the experiments following induction of anesthesia. The previously observed effect of the field on the EEG was absent in all rabbits; in contrast, anesthesia had no effect on the EEG changes caused by light. After the animals were killed the field experiments were repeated. The input signals to the EEG amplifier were analyzed as previously, and we found that %R and %D were essentially zero, independent of the presence of the field.

The reproducibility and consistency of the results far exceeded those of any previously reported study involving the biological effects of electromagnetic fields.

We attributed this consistency to the use of nonlinear analysis because we found that linear analytical methods were not capable of evidencing field-induced effects in the EEG.

The coil arrangement used in the study (Marino et al., 2002) produced fields that varied by less than 5% throughout the region occupied by the rabbit; we therefore knew that the field at the location of the electrogenic protein was 2.5 G, $\pm$ 5%, even though we did not know its anatomical location. One possibility was that electroreception occurred throughout the body as, for example, in somatosensory transduction. Alternatively, electroreception might have been localized, such as for the special senses. To help choose between the two possibilities we modified the coil arrangement so that the average magnetic fields in the cranial and caudal half of the rabbit were maximally different (Marino et al., 2003). Exposure of the cranial half of the animal resulted in effects on %R and %D in each case as previously (with one exception), with no false-positive results. When the experiment was repeated with the cranial half in the low-field region and the caudal half in the high-field region, no effect on the EEG was observed. When the field was localized to the head, the effects on determinism in the EEG described above were again seen. When the field was further localized to the eye, the effects did not occur. Taken together, the results can be interpreted to indicate that EMF transduction occurred somewhere in the head, probably the brain.

Employing conditions of analysis similar to those described in connection with the rabbit studies (Marino et al., 2002), we measured the response rate of normal human subjects to a low-strength, low-frequency magnetic field (Marino et al., 2004). Eight subjects were exposed to a series of trials consisting of the application of 1 G, 60 Hz, for 2 s, followed by a field-free period of 5 s, and the EEG was analyzed statistically using phase-space methods to assess whether the subject detected the field. As we had done with the rabbits, we used a discriminant procedure in the first subject to locate the epoch-segment windows that maximized the effect of the stimulus, and then applied those windows prospectively to compare E vs. C and S vs. C in the remaining subjects. The criterion for accepting the conclusion that a stimulus-related change in brain activity actually occurred was that the field resulted in at least 2 significant differences from among the 6 EEG derivations (family-wise error $p < 0.05$). As in all our previous studies, we removed the 30-ms portion of each trial after field onset and offset, and deleted trials that contained movement artifacts ($<$5% of all trials).

We found that a 190-ms window centered at 215 ms after commencement of the field yielded the lowest p value for E vs. C (C segment centered at 5.215 s, width of 190 ms) when p was not significant for S (3.215 s, width of 190 ms) vs. C. When the 190-ms window was shifted 30 ms earlier or later, the E vs. C comparison was not significant, indicating that the subject's response started at about 100 ms. The window width and location thus determined were then applied prospectively to 7 additional subjects in 7 independent experiments to ascertain the effect of exposure, and significant ($p < 0.05$) differences in %R and %D were found in each experiment (Marino et al., 2004). Light was also detected by all the subjects (190 and 175 ms for width and center location, respectively). No false-positive comparisons were found when the same mathematical procedures were used to compare sham-exposed and control segments.

The 100% response rate to EMFs manifested by the human subjects (Marino et al., 2004) was similar to the results found with the rabbits (Bell et al., 1992b),

suggesting that the ability to detect low-strength EMFs is a common property of the mammalian nervous system.

Recent Reports

Although the nonlinear method successfully showed that essentially all rabbit and human subjects exhibited altered brain activity when an EMF was presented (Marino et al., 2002, 2004), the way we implemented the nonlinear approach superimposed on each study subject a specific latency and duration of response that could be observed. We therefore modified the method so that the latency or duration of the response were not fixed in advance of the application of the field, but rather were determined for each subject with the requisite degree of statistical reliability; the details are given elsewhere (Carrubba et al., 2006).

Employing the improved procedure, we found that evoked potentials caused by onset or offset of the field (2 G, 60 Hz) occurred 109–454 ms after the stimulus application, depending on the subject; the evoked potentials were detected in the occipital electrodes in 16 of 17 subjects (family-wise error rate, <0.05 in each case) (Carrubba et al., 2007a). The potentials, which consisted of statistically significant increases or decreases in the nonlinear quantifiers, could not be detected when the EEG was analyzed by time averaging, indicating that occurrence of the potentials was nonlinearly related to presentation and removal of the field.

Several considerations led to the conclusion that the observed effects were true post-transduction changes in brain electrical activity triggered by the magnetic stimulus, that is, magnetosensory evoked potentials (MEPs): (1) The alternative explanation (that the effects resulted from interaction between the field and the scalp electrodes) was ruled out because the observed MEPs occurred several hundred ms after the stimulus (typical latency for evoked potentials); (2) sensory evoked potentials are typically produced by both onset and offset of a stimulus, and both responses were observed with EMFs; and (3) inter-subject variation in latency (within a well-defined range) was seen, as is the case with all known types of evoked potentials. It followed for all these reasons that the observed changes in brain electrical activity were true MEPs.

Nonlinear systems do not follow the law of superposition, and therefore their reaction to change in external conditions cannot be precisely predicted. If the MEPs (Carrubba et al., 2007a) were nonlinear, the brain electrical response exhibited by human subjects would be expected to differ even when the experimental conditions were replicated. We tested this hypothesis by comparing a subject's response to a weak magnetic stimulus at two times, separated by at least one week (Carrubba et al., 2007b). Eight clinically normal subjects were exposed to 1 G, 60 Hz applied for 2 s, with a 5-s inter-stimulus period, and EEGs were recorded from O1 and O2 (International 10–20 System) and analyzed as described previously (Carrubba et al., 2007a) to detect the onset MEP. Using nonlinear analysis, MEPs were detected in all subjects in the initial series of studies, and in all but one subject in the replicate studies (Fig. 3) (Carrubba et al., 2007b); no MEPs were detected using linear analysis. With one exception (Fig. 3, S6), the MEPs observed in the initial studies were also observed in the replicates. However, the relation of the determinism in the replicate (the law-governed dynamical activity reflected in the recurrence plot and characterized by the quantifier %R) to that in the original MEP differed significantly from subject to subject. The replicate MEP was manifested as a consistent increase in %R in S1 and

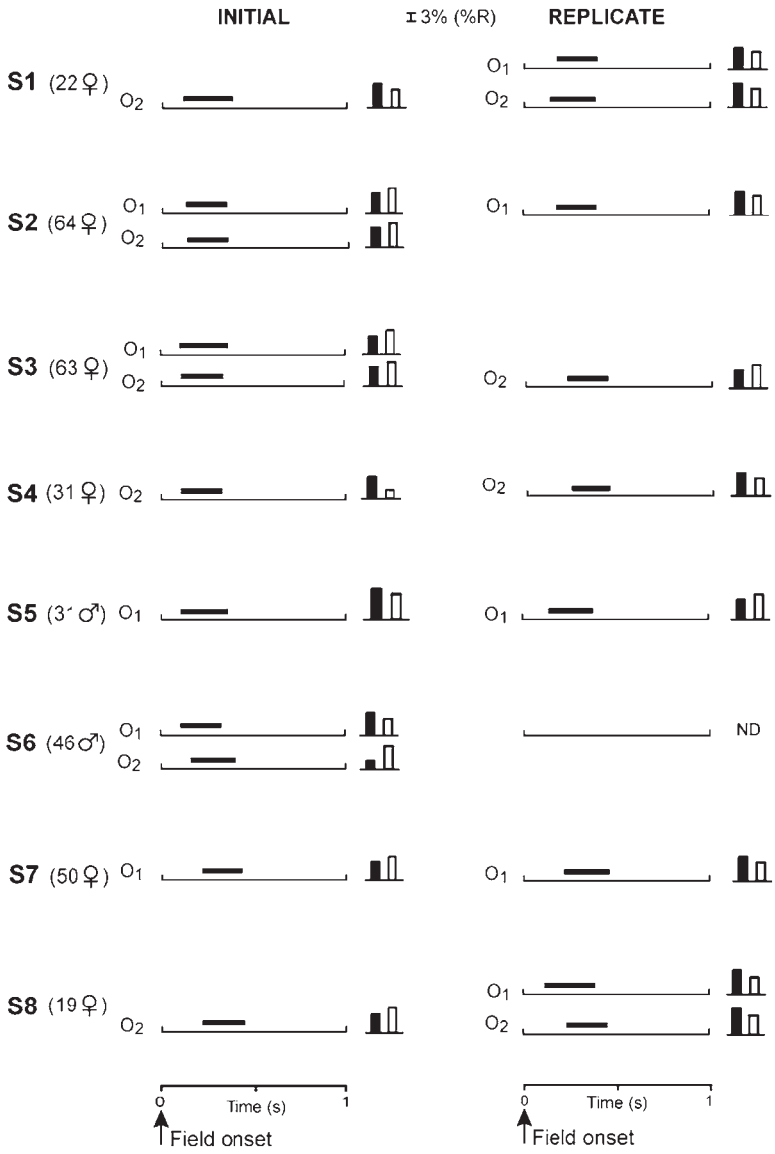

Figure 3. Detection of magnetosensory evoked potentials (MEP) in initial and replicate studies, using recurrence analysis. Latency and duration in each subject are indicated on the time axis. Bar graphs indicate the mean of the MEP (average of the significant points in the %R time series); black and white bars correspond to onset and control epochs, respectively (SD not resolved at scale presented). S1–8, subjects 1–8. ND, not detected (Carrubba et al., 2007b).

S4, a consistent decrease in S3, and as inconsistent differences in the other subjects which included 3 subjects who first exhibited a decrease and then an increase (S2, S7, S8) and one subject who responded oppositely (S5). Thus, the MEPs detected in this study were inconsistent, as predicted. Only a system governed by nonlinear laws can exhibit such a pattern of response.

Given that the effects of EMFs on brain electrical activity were nonlinear in origin (Carrubba et al., 2007a,b), it became necessary to reevaluate how the scientific requirement of reproducibility should be formulated because, in distinction to linear systems, consistency in the magnitude and direction of a stimulus-response relationship are not general properties of nonlinear systems. We therefore developed a procedure for demonstrating the consistent occurrence of changes in magnetosensory evoked potentials (MEPs) in individual subjects exposed to a magnetic field (Carrubba et al., 2008). In these studies, the magnetic field was applied for only 50 ms, and the MEPs were recorded during the interstimulus period. After all conditions that affected the analysis of the EEG in association with the presentation of a stimulus were specified in advance, we detected MEPs in all 15 subjects ($p < 0.05$ in each experiment) (Carrubba et al., 2008). The MEPs occurred within the predicted latency interval, were independent of the frequency and direction of the field, and were not detected using the traditional linear method of analysis, time averaging. When the results obtained within subjects were averaged across subjects, the evoked potentials could not be detected, indicating how nonlinear phenomena can be averaged away when incorrect analytical procedures are used.

Metabolic Studies

The studies discussed above were designed to test the hypothesis that EMFs were transduced by the sensory system (Fig. 4a) or were at least pertinent to that hypothesis (Fig. 4b). Another group of studies involved an attempt to determine whether field exposure resulted in a generalized metabolic effect that was reflected in brain electrical activity (Fig. 4c). For example, we compared the 10-Hz power in the occipital EEG one minute after 10 min exposure to 1 G, 10 Hz, with the pre-exposure 10-Hz power (Bell et al., 1994b) and found that the power was significantly reduced. Thus, after 10 min exposure, brain electrical activity was reduced immediately following the exposure—for whatever that means.

When the average relative spectral power in 20 subjects before and after exposure for 1 h to 12.6 G, 45 Hz was compared, changes in various frequency bands were seen, depending on the electrode derivation (Lyskov et al., 1993); the effects occurred when the field was applied intermittently (1 s on and 1 s off), but not when it was applied continuously. This result was consistent with the idea that the body recognized the onset and/or offset of the field (as opposed to its presence); however, the data was not protected against the possibility of family-wise error.

Investigators exposed subjects to complex, therapeutically motivated pulses whose salient features were an amplitude of 1.4 G (rms), a width of 853 ms, and a variable inter-stimulus period (110–1200 ms) (Cook et al., 2004, 2005). Occipital alpha power was increased after 15 min exposure (Cook et al., 2004). The investigators were unable to replicate their observation, but did report a decrease in alpha power in the context of a complicated exposure procedure (Cook et al., 2005).

The major limitation of these studies (Cook et al., 2004, 2005) was the absence of consideration of the family-wise error in the statistical analysis. In each experiment, several hundred complex ANOVAs were performed; consequently, the several statistically significant results found could reasonably be attributed to chance. On the other hand, it seems statistically improbable for chance results to occur in two independent experiments in the same electrodes (occipital) at the same Fourier frequencies (alpha).

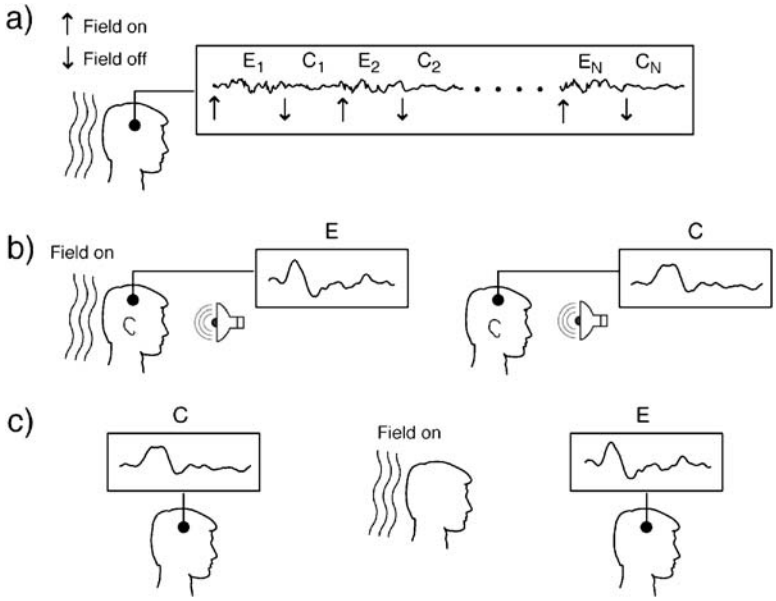

Figure 4. Distinction between sensory and metabolic studies. (a) A comparison between exposed (E) and control (C) epochs permits statistical evaluation of the hypothesis that the onset (or offset) of the field causes evoked potentials. (b) Comparison of auditory (or visual or somatosensory) evoked potentials in the presence and absence of a field permits statistical evaluation of the hypothesis that fields alter ongoing sensory processing. (c) Comparison of brain activity before and after a period of EMF treatment can establish that the field causes an effect on body metabolism but is not directly probative with regard to the responsible biological processes.

When subjects were exposed for 90 min to 50 Hz, 280 and 560 mG (rms), alpha activity from O_Z was significantly increased (176%, $p < 0.05$) at the higher field in 15 subjects but was unaffected in 10 subjects exposed to the lower field (Ghione et al., 2005). The difference in absolute alpha might have been real, but several pertinent considerations suggested otherwise. First, the authors did not present the results for relative alpha power; although it was not a planned comparison, it could have helped in the interpretation of the results. Second, the results were inconsistent with other dependent variables measured by the investigators (for example, an effect on hyperanalgesia was observed at the lower field strength but not at the higher field strength).

Several studies involved the effect of EMF exposure on brain activity during sleep. Sleep is divided into stages defined principally by the frequency content and pattern of the EEG. The deepest sleep levels (stages 3 and 4) are characterized by the presence of prominent delta waves (slow-wave sleep). Exposure of 18 subjects to 50 Hz, 10 mG, significantly reduced the duration of slow-wave sleep (fraction of the sleep period during which the subjects were in stages 3 and 4) (Akerstedt et al., 1999). In another study, however, exposure to 60 Hz, 283 mG had no effect on slow-wave sleep (Graham and Cook, 1999). Not surprisingly, there were numerous differences between the two studies that could have accounted for the differing results.

The metabolic effects of EMF treatment on the response to visual and auditory stimuli has been evaluated for several different purposes. In studies undertaken as part of a health assessment of power-frequency magnetic fields, investigators assayed

many electrophysiological variables in subjects exposed to 1 G, 50 Hz (Crasson et al., 1999; Crasson and Legros, 2005). In the first study (Crasson et al., 1999), the investigators reported that the amplitude of the N1 wave in a specialized AEP paradigm (dichotic listening task) and the P300 latency in a visual discrimination task were altered after magnetic-field exposure. However a large number of 2-way ANOVAs were performed, only a few of which were statistically significant. The second study (Crasson and Legros, 2005) was conducted specifically to test the hypothesis that the original observations were real, however none of the expected effects were found.

The possible explanations for the generally negative results of the first were listed (Crasson, 2003): (1) differences in the functional state of the nervous system; (2) differences in individual sensitivity; (3) the possibility that the effect was simply small and was lost in the noise. However, the most reasonable explanation was that the relationship between the applied field and the neurophysiological response was nonlinear, and consequently was unlikely to be detected using linear analysis.

In another biohazard study (Lyskov et al., 2001), exposure to 100 mG, 60 Hz, for 10 min did not alter the fundamental frequency in the Fourier representation of the visual evoked potential (flickering video display, refresh frequency, 60 Hz), on average, in either normal subjects (8.1 ± 4.5 Hz and 7.9 ± 4.1 Hz before and after exposure, respectively) or in 20 self-selected electrically hypersensitive subjects (9.4 ± 8.1 Hz and 9.1 ± 6.9 Hz) (Lyskov et al., 2001). The experimental design was based on a 3-way ANOVA, which may have been insufficiently sensitive for detecting changes in the VEP.

Pain-related somatosensory evoked potentials obtained before and after 2-h exposure to 0.7 G, 0.03–0.07 Hz were compared (Sartucci et al., 1997); the field-generating apparatus had been designed to study the effect of earth-strength magnetic fields on the homing ability of pigeons. The amplitudes of the P150 and P250 waves were reduced after exposure, but the waveform latencies were unaffected. The difficulty with the results involved the statistical analysis. For example, the reported amplitudes ($\pm$ SEM) of the P150 waves (in μV) were 6.3 ± 1.2 and 4.8 ± 0.8 before and after exposure, respectively. The investigators claimed that these means differed at $p < 0.05$; however, the two-tailed p value for this data is $p = 0.31$. A similar problem occurred for all of the reported evoked-potential data.

Event-related potentials (visual oddball task, 9 EEG channels) were measured before and after 20-min exposure to 20 G, either 5 Hz or 20 Hz (Wei et al., 1997). A reduction in P300 latency was reported after 5-Hz but not 20-Hz stimulation, in some of the electrodes. However, the results were not protected against family-wise error.

Discussion

The seminal question regarding the effects of low-frequency electromagnetic fields is whether their presence is detected by the body. If so, then the diverse biological effects attributed to EMFs might all be deterministically explainable within the broad biological theory of stress. If not, EMF-induced bioeffects are not logically possible. We theorized that the detection process was sensory transduction. Whenever stimulus-induced changes in brain activity are observed, cognitive processing of stimulus-related information, hence transduction of the stimulus, can reliably be inferred. It was with an intent to argue in this manner that we performed a series of studies on animal and human subjects regarding the effects of EMFs on brain

electrical activity (Bell et al., 1991, 1992a,b, 1994a,b; Marino et al., 1996, 2002, 2003, 2004; Carrubba et al., 2006, 2007a,b, 2008). Other investigators conducted similar studies for their own purposes, and those studies were included in this review.

From a dynamical perspective the changes in brain activity triggered by EMFs could have been linear or nonlinear; but like poor Oedipus who did not know who he was, we did not know which model was correct. In our initial studies we assumed a linear model and found a stimulus-response relation between EMFs and brain electrical activity; but we could demonstrate this relation in only some subjects (Bell et al., 1991, 1992a,b, 1994a,b; Marino et al., 1996). Other investigators who also assumed a linear model found results that were generally in accord with ours, namely some subjects responded, some did not, and at least some of the non-responders could probably be explained by a lack of sensitivity of the analytical method (Fuller et al., 1995, 2003; Dobson et al., 2000a,b). Although a linear model was incorrectly assumed in both groups of experiments, the further error of averaging the data over all subjects was avoided. In six other studies where the data was averaged across the subjects, a recognizable pattern regarding the meaning of the results did not emerge (Graham et al., 1987, 1992, 1994; Cook et al., 1992; Heusser et al., 1997; Lyskov et al., 2001; Stevens, 2007); such inconsistency is normal in all areas of EMF biology whenever the data is averaged in that manner.

When the effects of EMFs on brain electrical activity were analyzed using mathematical tools that had been developed for studying nonlinear systems, it became possible to capture the deterministic activity in the EEG caused by the stimulus (regardless of whether it was an increase or a decrease) prior to comparing means in a statistical analysis. Capturing the effect of the stimulus prior rather than subsequent to averaging the data was the key step that enabled us to overcome the problem that we identified, and allowed us to show that EMFs were consistently transduced by essentially all the animal and human subjects (Marino et al., 2002; Carrubba et al., 2007a). We showed that a fundamental effect of an EMF stimulus is the triggering of onset and offset evoked potentials in the brain (Carrubba et al., 2007a), and we described a procedure by which their presence can be demonstrated consistently, in every subject, with the requisite statistical reliability (Carrubba et al., 2008).

The various meanings of "nonlinear" are discussed elsewhere (Marino and Frilot, 2003). As used here, the term refers to the nature of the law that governs brain electrical activity when the brain is cognitively processing EMF-stimulus-related information. If a process is "nonlinear," some counter-intuitive (at least to some investigators) phenomena can properly fall within the realm of science (law-governed activity), for example phenomena that are "inconsistent" with regard to various pertinent characteristics (Fig. 3). It is crucial to recognize that the scientific requirement of reproducibility applies with full force to nonlinear EMF phenomena. Properly applied, "reproducibility" simply means that the EMF stimulus affected brain activity—there is no further condition regarding, as examples, magnitude, or direction of the change.

After the first concerns that man-made electromagnetic fields in the environment might be a hazard to public health were raised almost 40 years ago (Becker, 1972; Adey, 1976), the main counter-argument was that the reported EMF-induced bioeffects were inconsistent, thereby indicating only the existence of inconspicuous experimental errors, not real biological processes. There never was any reliable evidence that the argument was true. Now, there is clear evidence the argument is false; magnetosensory evoked potentials elicited by EMFs can be detected in essentially every subject examined when the proper form of analysis is used (Carrubba et al., 2008).

The results of the metabolic EMF studies do not materially advance our understanding of EMF biology. Perhaps EMFs can alter spectral power (Lyskov et al., 1993; Bell et al., 1994b; Cook et al., 2004, 2005; Ghione et al., 2005), sensory evoked potentials (Sartucci et al., 1997; Wei et al., 1997; Lyskov et al., 2001), or brain activity during sleep (Akerstedt et al., 1999; Graham and Cook, 1999). Focused, hypothesis-driven studies having appropriate statistical designs are needed to verify and establish the validity of these ideas.

In closing, we think it appropriate to speculate on how and why human subjects respond nonlinearly to EMFs. Electric and magnetic receptors that facilitate finding food, avoiding predators, and navigating in the environment occur in lower life forms (Wachtel and Szamier, 1969; Manger and Pettigrew, 1996; Walker et al., 1997). We previously described a biophysical process that could explain how EMF transduction occurs in these species (Kolomytkin et al., 2007); vestiges of this detection system might still exist in human beings. Evolutionary considerations also suggest a reason that the MEPs were nonlinear. The processes responsible for the linear correspondence between stimuli such as sound or light and the cognitive responses they induce resulted from evolution by natural selection, leading progressively to physiological linear sensory systems because consistency conferred a selective advantage. Conversely, in the absence of natural selection there is no process by which the phenomenon of consistency in response to a stimulus can come about. Compared with their present-day levels, EMFs were negligible throughout the period of evolution of life on earth, and consequently, a physical mechanism capable of producing a linear response did not develop. In this view, the existence of a nonlinear human magnetic sense could be a vulnerability in the molecular machinery chosen by evolution to mediate other sensory modalities because any physical realization of a sensory system for one kind of stimulus is unlikely to be completely immune to all other kinds of inputs (Nesse and Williams, 1998).

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