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Electrical states in the rabbit brain can be altered by light and electromagnetic fields

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The effect of low-frequency magnetic fields on the rabbit electroencephalograph (EEG) was studied using a quantitative procedure that permitted statistical evaluation of the response of individual animals. The field conditions used were those predicted by various theories to result in field—animal interactions; light and sham exposure were employed as positive .and negative controls, respectively. Sixty-seven percent of the rabbits exhibited changes in the EEG power spectra when light was presented in 2-s epochs; none of the animals responded to sham exposure. When 1 Gauss, 5 Hz, was presented in 2-s epochs, 100% of the animals tested responded to the presence of the field. The rabbits did not respond when the magnetic-field frequency was higher than the physiological range (1—20 Hz) or when it was tuned for resonance of K^+. The results showed that an electrical state function may be operationally defined for the rabbit brain, and used to assess the occurrence of an interaction between an animal and external magnetic fields.

INTRODUCTION

Exposure to subliminal electromagnetic fields (EMFs) has been associated with effects on the hematological[28], immunological[15], cardiovascular[16], and nervous[19] systems of animals, and with both the cure[5] and cause[24] of disease Knowledge of the biological and molecular mechanisms that subserve these observations has not kept pace with their increasing number. Various mechanisms have been proposed[13,26,32], three of which presently seem promising.

EMFs may alter calcium-binding in membrane-bound glycoproteins, thereby affecting receptor-ligand interactions and resulting in transmission of a signal to the cell interior[3]. The supporting evidence includes reports of the effects of EMFs on calcium-binding[7], ligand-receptor interactions[25], and the EEG in monkeys[6,18]. The link with cancer may involve the EMF functioning as a promoter[14]. The glycoprotein-EMF interaction appears to be maximal when the EMF frequency corresponds to a frequency of ongoing physiological activity[2,6,17,18] (PA model).

A second possible mechanism[22] involves movement of ions through protein channels in the cell membrane when the angular frequency of the EMF equals the product of the unhydrated ion's charge-to-mass ratio and the local magnetostatic field (ion resonance (IR) model). Altered ion fluxes could activate second-messenger systems or induce cytoskeletal alterations leading to changes in cell function. Supporting evidence involves resonance-induced changes in Ca^{2+}-dependent motility[29], and Li^+-dependent behavior[23].

Alternatively (or additionally) EMFs may be stressors capable of producing physiological effects without directly causing changes in second-messenger systems or cytoskeletal structures[8] (stressor (SR) model). Excitable cells may contain an electrogenic protein (EGP) whose functional state depends on the presence of weak pencellular EMFs[33]. Activated EGPs could produce subthreshold changes in membrane potential that modify neuronal activity through a variety of specialized junctional structures[1,34]. The modified neuronal activity is an afferent signal that projects to the thalamus and results in efferent signals to the endocrinologic, immune and autonomic nervous systems that presumably serve a beneficial purpose for the organism — perhaps to maintain homeostasis. In this view the link with cancer is indirect, and stems from impaired immune surveillance resulting from chronic stimulation of the neuroendocrine systems. The evidence in support of this theory consists of reports of biological changes in myriad physiological parameters following exposure of subjects to spectrally disparate EMFs[27]. Since many different types of EMFs resulted in biological changes, probably neither the frequency nor the strength of the EMF were crucial factors in determining

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whether an effect would occur.

By using EMFs that conformed to the various theories, we sought to obtain evidence in support of one or more of the theories of EMF action. Our idea was that the EMF detection by the organism would be manifested as a change in the brain's electrical activity as a consequence of transduction of the EMF, and that relative sensitivity to the different fields could be determined from the extent of the changes induced.

Since the locus of transduction might be within the central nervous system itself, we devised a method that did not require resolution of afferent and efferent signals or delineation of anatomical paths of signal propagation. The validity of the concept of the existence of well-defined brain electrical states corresponding to the presence of a specific somatic stimulus was studied using light as the stimulus. The method was then applied using the magnetic fields. The results obtained using rabbits are described here; our results with human subjects are described elsewhere[10-12].

MATERIALS AND METHODS

EMF exposure system

Magnetic fields were chosen for this study because, compared with electric fields, they are easier to control and characterize; the dose of magnetic field experienced by each animal could therefore be rigorously determined. Low-frequency fields were used because they are commonly present in the general environment, and because one of the theories to be tested is based on the frequency similarity between the field and on-going brain electrical activity. Low-frequency magnetic fields were produced using a pair of coils 130 cm in diameter, each consisting of 250 turns of copper wire (0.79 mm in diameter); the coils were maintained parallel and separated by 65 cm (the Helmholtz condition) by a wooden frame. When energized, the coils produced a magnetic field uniform to within 5% in a cylindrical volume having a diameter of 54 cm, located coaxially with the coil axis[9]. Exposure occurred with the animals confined to this region; consequently, all putative sensory transducers simultaneously received the same stimulus. The coil current was obtained from a signal generator (Model 182A, Wavetek, San Diego, CA), and amplifier (Model 7500, KrohnHite, Avon, MA).

Three field conditions were chosen for study based on their relation to the theories regarding biological detection of EMFs. The PA and IR models both place requirements on the spectral characteristics of the applied EMF. Since 5 Hz is prominent in the rabbit EEG, we applied 1 Gauss, 5 Hz to test the hypothesis that stimulation at a frequency of ongoing electrical activity would reinforce such activity (1 Gauss is a field present in the workplace and residential environments, and is not known to produce sensory stimulation). The corresponding magnetic field is designated B(1,5).

Ion-resonance bioeffects involving Ca^{2+}, K^+ and Li^+[22,23,29] have been described; we chose K^+ ions as the target because K^+ channels are ubiquitous in both excitable and non-excitable cells[20]. At 25 Hz, the charge-to-mass ratio of K^+ (0.0246×10^8 C/kg) results in resonance for that ion in static magnetic field of 0.64 Gauss. We therefore applied a static field of 0.64 Gauss and a 25-Hz field of 0.64 Gauss (R.M.S.) (designated B(0.64,25)). The Helmholtz coils were positioned such that their fields were orthogonal to the vertical plane containing the geomagnetic field. This arrangement ensured that the effective static field along the coil axis was 0.64 Gauss, as required for K^+ resonance at 25 Hz.

Under the stressor hypothesis, EMF detection by the organism can occur at all frequencies; 25 Hz was a convenient choice of frequency because there was minimal activity in the rabbit EEG at this frequency, and because it does not correspond to a resonance condition for any biologically significant ion in the geomagnetic field in our laboratory (designation B(1,25)).

Procedure

If *P(f, t)* represents the power in the EEG at frequency *f* and time *t* (averaged over 2-s epochs) as determined by the Fourier transformation of the EEG voltage signal, we hypothesized that *P(f, t)* differed reliably between stimulus-on and stimulus-off epochs, and therefore that the occurrence of such a difference was evidence that stimulation has occurred.

Nine female New Zealand rabbits were used in this study. A trial consisted in the presentation of the magnetic field for 2 s, followed by an interstimulus period having an average duration of 8 s (range, 5—11 s, varied randomly). Each measurement consisted of 250—300 trials, of which the first 200 artifact-free trials were used for analysis of the effect of the stimulus. During each measurement the rabbit was restrained in a wooden box; a plastic collar prevented the rabbit from withdrawing its head into the box. The restraint box was placed within a larger wooden box designed to eliminate the entry of light and restrict the entry of sound and odor. The larger box was placed in the gap of the Helmholtz coils such that the rabbit's rostral-caudal axis was perpendicular to the coil axis, and the rabbit was located within the region where the magnetic field varied by less than 5%. Presentation of the magnetic field commenced S mm after the rabbit was placed in the light-tight box. The first 5 trials were discarded, and the next 200 artifact-free trials were used for EEG analysis.

We chose a light stimulus as a positive control to determine whether the method of EEG analysis could reveal changes in on-going activity caused by a specific sensory stimulus. A weak red light from a light-emitting diode was used as the visual stimulus; the diode was mounted inside the light-tight box 10 cm from the rabbit. A procedure similar to that used with the magnetic field was followed for the trials involving presentation of the light, with the light substituted for the field. Each rabbit received the light stimulus B(1,5), B(0.64,25), B(1,25) and a sham stimulus that consisted of 300 trials during which neither a magnetic field nor light was presented (the sham stimulus served as the negative control). Each rabbit was measured no more than once in a 24-h period (1 stimulus/day), and each measurement was replicated not less than 24 h after the first measurement. All animals received all stimuli, except that only 5 rabbits received B(1 5) (because the equipment that produced the DC field was not available when the first 4 rabbits were studied).

Each rabbit received a complete ophthalmological examination. In all animals, the corneas were clear, and about 15 mm in diameter. The pupils all reacted to light, and the lens and vitreous were clear. The optic nerves were pink and the optic-nerve and choroidal blood vessels appeared normal. The retina was clear, and the retinal blood vessels were normal. Following the EEG measurements, the rabbits were killed (Beuthanasia-D, Schering Co., Kenilworth, NJ), and measurements were made on the passive electrical properties of the field interaction with the electrodes.

EEG analysis

Surface electrodes were used for all EEG determinations. The recording electrode was placed over the occipital region of the cerebrum which, in the rabbit, lies under the easily-palpable suture of the parietal and interparietal cranial bones. The indifferent electrode was placed 2.5 cm rostral along the midline, and the ground electrode was placed 2.5 cm caudal to the recording electrode (also along the midline). The electrodes (gold-plated, 0.5 cm in diameter, Grass Instrument Co., Quincy, MA) were attached to the shaved scalp using conducting paste (EC2, Grass Instrument Co., Quincy, MA). Electrode impedances were 1—3 kg; they were measured before and after each experiment.

The EEG signal was filtered to pass 0.3—35 Hz, amplified, and simultaneously recorded on an electroencephalograph (Model 6, Grass Instrument Co., Quincy, MA) and sampled at 200 Hz and stored on a 40 megabyte hard-drive. Following the measurement (about 30—60 mm in duration), the data was transferred to a mainframe computer for analysis. Trials containing obvious movement artifacts were identified on the written record, and the corresponding digitized data was deleted. Fourier transforms were performed on 200 consecutive 2-s artifact-free stimulus epochs and their corresponding control epochs (using Spectra, SAS Institute, Inc., Austin, TX); the control epoch for each stimulus epoch was the 2-s period immediately preceding the stimulus. 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. The power coefficients were not normally distributed, either as obtained from the Fourier transform calculation, or after a variety of mathematical transformations (including $\log(x/(1—x))$. Consequently, the data was analyzed using the non-parametric Wilcoxen signed rank test to test the hypothesis that the EEG during the stimulus epochs did not differ from that measured during the corresponding control epochs[4,31].

For each Fourier frequency, we adopted the criterion of $P < 0.05$ to conclude that the coefficient at that frequency (in that experiment) differed between the control and stimulus epochs. Thus, the probability that the spectral power at any Fourier frequency would differ by chance in both a measurement and its replicate was 0.05 x 0.05 = 0.0025. The probability that any such differences would be in the same direction was 0.0025 x 0.5 = 0.00125. If the probabilities are viewed as independent (which is appropriate because we are considering the possibility that power at particular frequencies will differ solely by chance), then the overall level of confidence in 39 tests, each at the level of 0.99875 is $0.99875^{39} = 0.9524$. Thus it is unlikely ($P < 0.05$) that a replicable and consistent change in power would occur by chance at even one frequency. Consequently, our criterion for decision to reject the null hypothesis was that the stimulus was considered to have caused a change in the EEG if a consistent and a replicable change occurred at one or more of the 39 Fourier coefficients of the spectral power of the EEG within 1—20 Hz.

RESULTS

The effect of the light stimulus on the EEG differed among the animals tested. In some animals (Fig. 1) (#1, #2, #4, #6, and #8, Table I) the light resulted in an increase in power in one or more frequencies in the 1—7 Hz range. Detection also occurred in one animal at 18 Hz (#5). Two animals (#3, #9) were consistently unresponsive to the light (Fig. 2). In one animal (#7) (Fig. 3, Table I) differing responses occurred in the 2 measurements: an elevated

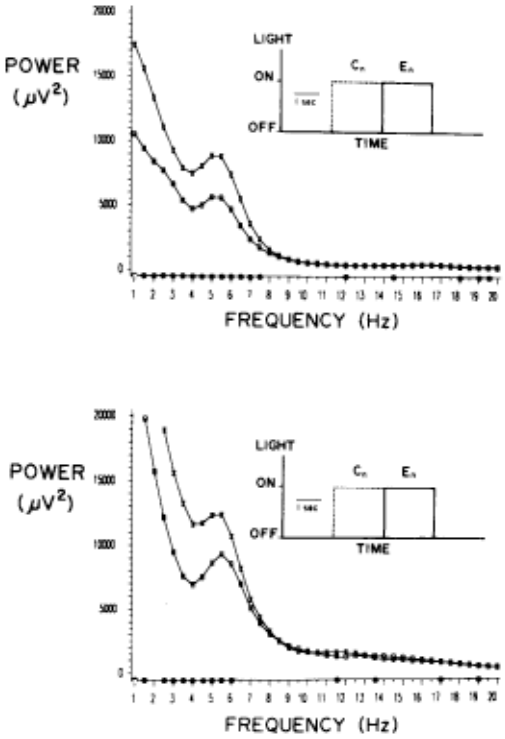


Fig. 1. Average periodograms of rabbit no. 6 showing a response to the presentation of the light stimulus. The average responses (200 two-second epochs) during the light stimulus and control epochs are indicated by X and , respectively. The inset depicts the temporal relation between the nth control (C_n) and exposed (E_n) epoch (n = 200). The average intertrial time was 8 s. C_n, E_n were compared at each frequency using the Wilcoxin signed rank test. Frequencies that differed significantly ($P < 0.05$) are indicated by a filled circle on the abscissa. The spectra were subjected to Bartlett (5-point triangular) smoothing. The initial (upper) and replicate (lower) measurements are shown.

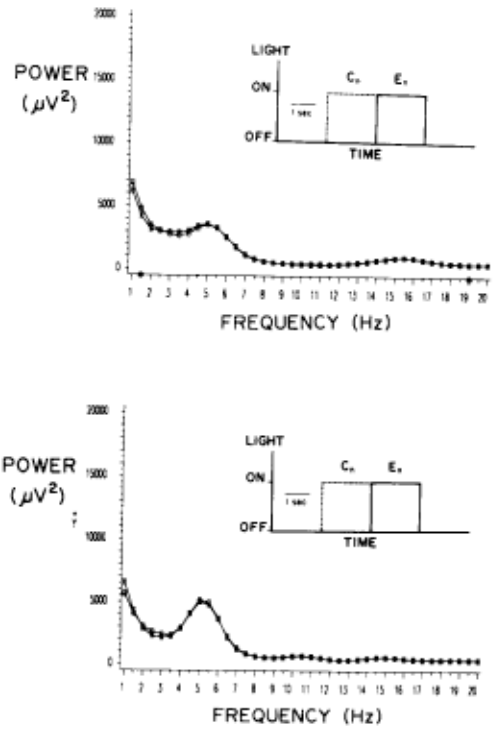


Fig. 2. Average periodograms of rabbit no. 3 showing no response to the presentation of the light stimulus. The average responses (200 two-second epochs) during presentation of the light stimulus and control epochs are indicated by X and , respectively. The inset depicts the temporal relation between the nth control (C_n) and exposed (E_n) epoch (n = 200). The average intertrial time was 8 s. C_n, E_n were compared at each frequency using the Wilcoxin signed rank test. Frequencies that differed significantly ($P < 0.05$) are indicated by a filled circle on the abscissa. The spectra were subjected to Bartlett (5-point triangular) smoothing. The initial (upper) and replicate (lower) measurements are shown.

TABLE I

EEG frequencies at which significant differences were observed following the first (—1) and second (—2) presentation of the indicated stimuli

FORE, frequencies of repeated effects; NM, not measured.

Rabbit	*Light-1*	*Light-2*	*Light FORE*	*Sham-1*	*Sham-2*	*Sham FORE*	*B(1,25) -1*	*B(1,25) -2*	*B(1,25) FORE*	*B(0.64,25) -1*	*B(0.64,25) -2*	*B(0.64,25) FORE*	*B(1,5) -1*	*B(1,5) -2*	*B(1,5) FORE*
	1.0-2.5, 5.5-7.5, 14.0	1.0-3.5, 6.0,6.5, 7.5,11.0	1.0,1.5, 2.0,2.5, 6.0,6.5	3.0,7.0, 10.5	6.5, 12.5, 16.5	None	12.5, 19.5	—	None	3.5,5.5, 13.5, 15.0	5.0,13.0	None	NM		—
2	1.0-5.0, 8.0,9.0, 15.0, 15.5, 19.5	2.0-4.5, 5.5	2.0,2.5, 3.0,3.5, 4.0,4.5,	2.5,8.0	1.0	None	12.0	10.0, 19.5	None	1.5,7.5	19.5	None	NM		—
3	1.5,19.0	—	None	1.0,4.0, 7.5,8.0, 11.5, 14.5, 19.0	4.0,11.0	None	4.0	3.0,4.0, 15.5	4.0	15.5	17.5	None	NM		—
4	2.0,3.5, 12.0	2.0,3.5, 7.0,8.5	2.0,3.5	7.5,11.5, 16.5	5.0,12.5, 18.5, 20.0	None	8.0,13.0, 13.5	4.5, 17.5, 19.5	None	1.0,3.5, 8.0,19.0		None	NM		—
5	6.5, 15.5, 16.0, 18.0	3.5, 4.0, 6.0,6.5, 7.5,8.0, 10.5, 16.5, 17.5, 18.0, 19.5	18.0	10.5, 16.0, 17.0	4.0	None	8.0,10.5, 13.5	3.5	None	2.0,3.0, 7.0	—	None	5	5	5
6	1.0-7.5, 12.0, 14.5, 18.0, 19.0, 19.5	1.0,1.5, 2.5-6.0, 11.5, 13.5, 17.0, 19.0	1.0,1.5, 2.5,3.0, 3.5,4.0, 4.5,5.0, 5.5,6.0, 19.0	11.0	15.0	None	19.5	8.5,11.0, 12.0, 18.0	None	1.0,5.5, 7.0,8.0, 9.0,10.5	4.5,20.0	None	5	5	5
7	2.0-3.0, 5.0,6.5, 8.0,9.0 9.5, 10.0, 17.5	1.5,18.5	None	7.5,15.5	4.0,8.0, 17.0	None	9.5,18.0	5.5,15.0	None	9.0,12.0, 17.0	6.5,10.5, 13.0	None	5	5	5
8	7.0	1.0,1.5, 2.5-4.5, 6.0-8.0, 9.0,17.5	7.0	—	—	None	9.0,12.0, 15.0	5.0,18.0, 20.0	None	5.5	—	None	5	5	5
9	3.0	1.0,5.0	None	6.0	5.5	None	4.5,7.5	10.5, 12.0, 15.5	None	10.5	13.0	None	5	5	5

low-frequency power was observed in one case, but not in the replicate measurement. A similar result was seen in #5 (although in this case, the a priori condition for acceptance of an effect was satisfied).

B(1 ,5) resulted in a significant increase in each of the 5 animals tested (Fig. 4, Table I). Neither B(0.64,25) nor sham exposure yielded any significant differences (Table I). An effect due to the B(1,25) stimulus was observed at one frequency in one animal (one of the 2 rabbits that were unresponsive to the light stimulus (Fig. 4, Table I)).

The data regarding B(1 ,5) (Table I) were consistent with the hypothesis regarding an interaction with ongoing electrical activity, but another possible explanation was that the peak at 5 Hz resulted from an inductive artifact superimposed on the EEG. We conducted 2 further studies to help resolve the question whether the observed changes were due solely to induction.

An inductive interaction yields a voltage that is proportional to the rate of change of the magnetic flux[21]; for a sinusoidal magnetic field, the induced voltage is pro-

portional to the product of the frequency and the field strength[21]. Consequently, if the apparent effect at 5 Hz (Table I) resulted from an inductive artifact, the Fourier coefficient at the stimulation frequency, which has units of μV^2, should be proportional to the product of the squares of the frequency and field strength. If P_O is defined as the difference in average power between the exposed and control epochs at 5 Hz, 1 Gauss, then the difference in average power should change in the manner illustrated in Fig. 6 for the other considered conditions of stimulation. The assumption of an induction artifact was not sufficient to explain the apparent effect associated with stimulation using B(1 ,5) when the animals were alive (Fig. 6A), but it was sufficient when the animals were dead (Fig. 6B).

In a further study, baseline BEG was recorded (average of 25, 2-s epochs), and then the rabbits were exposed continuously for 30 mm to *5* Hz, 1 Gauss. The magnetic field was removed, and the first 25 artifact-free epochs were averaged. The control procedure consisted of sham exposure (identical conditions in all respects, except that the magnetic field was not turned on). All measurements were replicated (on different days), and the results were averaged. A significant difference in 5 Hz power was observed in the trials involving magnetic-field exposure (Table II).

DISCUSSION

The experimental design in which each subject serves as its own control[4,31] is well-suited to determinations of the effect of light or electromagnetic fields because (i) it permits decisions regarding detection to be made concerning each subject, and (ii) it does not require a priori specification of specific sensitive frequencies (thereby allowing for the possibility that different animals may respond at different frequencies). Six rabbits detected the light stimulus, as judged by the criterion of a replicable statistical difference at one or more frequencies (Table I), 5 of which (#1, #2, #4, #6, #8) consistently exhibited elevated power in one or more frequencies in the 1-7 Hz range. Fig. 7 displays the frequency distribution

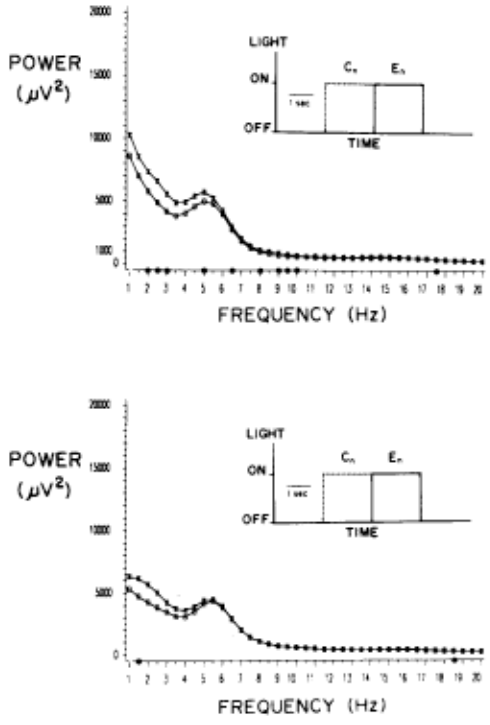


Fig. 3. Average periodograms of rabbit no. 7 showing an inconsistent response to the presentation of the light stimulus. The average responses (200 two-second epochs) during presentation of the light stimulus and control epochs are indicated by X and , respectively. The inset depicts the temporal relation between the nth control (C_n) and exposed (E_n) epoch *(n* = 200). The average intertrial time was 8 s. C_n, E_n were compared at each frequency using the Wilcoxin signed rank test. Frequencies that differed significantly *(P* < 0.05) are indicated by a filled circle on the abscissa. The spectra were subjected to Bartlett (5-point triangular) smoothing. The initial (upper) and replicate (lower) measurements are shown.

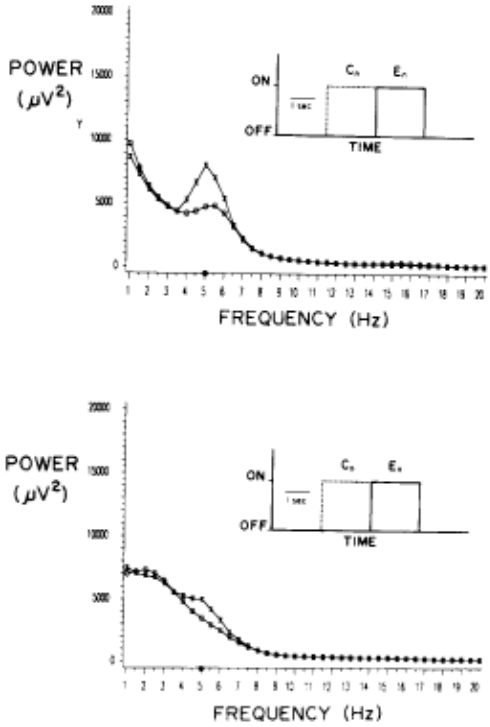


Fig. 4. Average periodograms of rabbit no. *5* showing a response to the presentation of B(1,5). The average responses (200 two-second epochs) during presentation of the light stimulus and control epochs are indicated by X and , respectively. The inset depicts the temporal relation between the nth control (C_n) and exposed (E_n) epoch *(n* = 200). The average intertrial time was 8 s. C_n, E_n were compared at each frequency using the Wilcoxin signed rank test. Frequencies that differed significantly *(P* < 0.05) are indicated by a filled circle on the abscissa. The spectra were subjected to Bartlett (5-point triangular) smoothing. The initial (upper) and replicate (lower) measurements are shown.

TABLE II

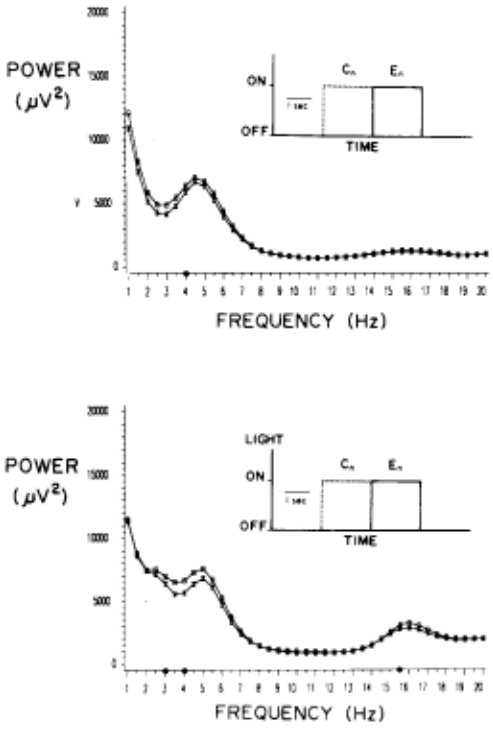
Effect of magnetic field (1 Gauss, 5 Hz) on EEG power at 5 Hz

P_A, P_B is the average 5-Hz power (averaged over 25 two-second epochs) before and after treatment (magnetic field or sham exposure), respectively. The absolute values of the differences were averaged over 2 measurements of the response to each treatment (all measurements taken on different days). The paired t-test was used.

Animal number	$\overline{\lvert P_A - P_B \rvert}$ (μV^2)	
	Magnetic field	*Sham exposure*
5	5022	2687
6	4153	1502
7	5593	2662
8	5067	556
9	2197	1813
Mean ± SD	4406 ± 1339	1844 ± 888
		$P < 0.05$

of observed significant differences (data from Table I). Although most differences occurred within 1—7 Hz, the average power in this frequency range did not differ

Fig. 5. Average periodograms of rabbit no. 3 showing the response to the



presentation of B(1,25). The average responses (200 two-second epochs) during presentation of the light stimulus and control epochs are indicated by X and , respectively. The inset depicts the temporal relation between the nth control (C_n) and exposed (E_n) epoch (n = 200). The average intertrial time was 8 s. C_n, E_n were compared at each frequency using the Wilcoxin signed rank test. Frequencies that differed significantly ($P < 0.05$) are indicated by a filled circle on the abscissa. The spectra were subjected to Bartlett (5-point triangular) smoothing. The initial (upper) and replicate (lower) measurements are shown.

significantly from the control (Table III). Thus, although the data showed that some rabbits responded in a specific frequency range to the light stimulus (Table I), we could not demonstrate a change in the average power within the range. If the frequency range used for comparison were limited to 1.5—3.5 Hz (Fig. 7), we could conclude from an analysis of the average power that the rabbits detected the light (Table III). Nevertheless, no animal was individually responsive throughout this range (Table I). Thus (i) analysis of group response was not predictive of individual response, and (ii) although the pattern of individual responses was similar (in range), the particular frequencies at which the effect was manifested could not be predicted prior to measurement.

When light-induced reactions are produced in human subjects, they usually occur at 8—13 Hz as either an increase or decrease in power[4,30,31]. In contrast, in rabbits the changes always consisted in an increase in power, usually at 1—7 Hz.

The results using the light stimulus showed that the BEG from rabbits obtained during a period of (relative) sensory deprivation differed reliably in some animals from that obtained during presentation of a light stimulus; furthermore, sham exposure did not produce any false-positive decisions regarding detection. Therefore, within the framework of the procedures followed here, the BEG could validly be regarded as a state function of the organism which differed depending on the presence or absence of a specific stimulus. Unlike an evoked response, which occurs subsequently to the stimulus with a

TABLE III

Effect of light on the average power (within specific frequency ranges) on the rabbit EEG

The data was averaged over the indicated frequency range and over 2 measurements for each animal. A negative sign indicates that the power measured during presentation of the light was greater than that measured during the control epoch. Control refers to the average difference observed using sham exposure. The paired *t*-test was used.

Animal number	*Average power (μV^2)*			
	1—7 Hz		*1.5—3.5 Hz*	
	Light	*Control*	*Light*	*Control*
1	—1842	16	—2806	104
2	—1416	26	—2787	—278
3	64	77	120	257
4	—167	92	—559	—440
5	75	221	167	389
6	—4200	—30	—5266	—43
7	—783	222	—1309	—139
8	—317	—55	—232	157
9	103	86	294	210
	—942±1406	73±98	—1375±1889*	24±270

* $P < 0.05$.

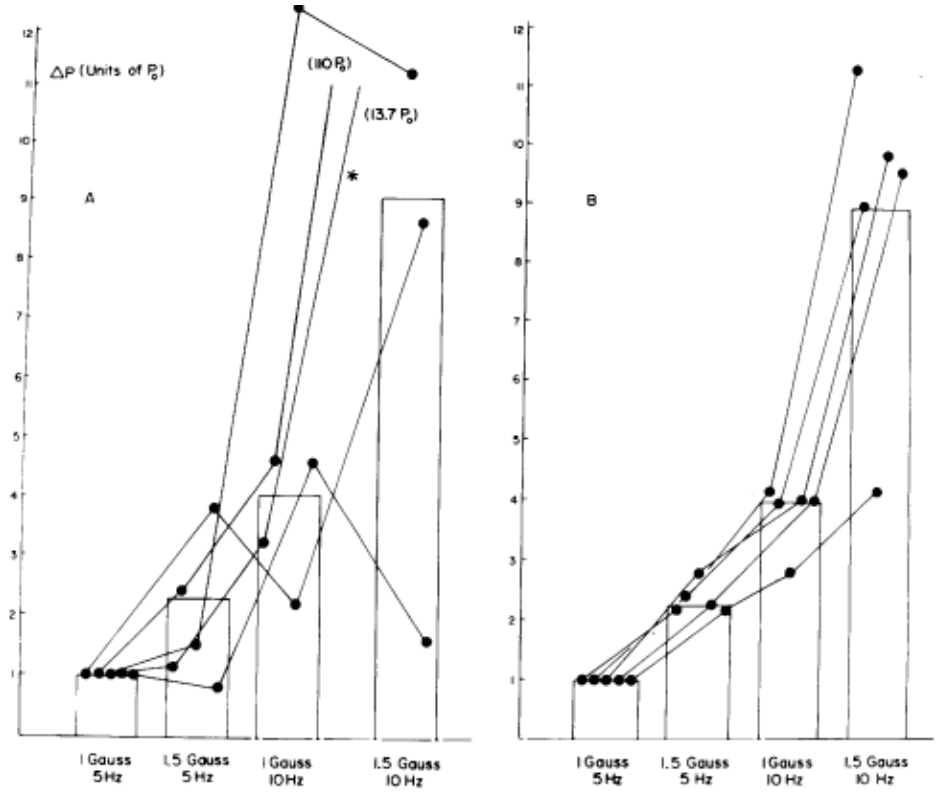


Fig. 6. A: observed values of change in EEG power in 5 rabbits for various conditions of stimulation (combination of field strength and frequency). At any one condition, ΔP is the average change in power ($\Delta P = P_B - P_C$ where P_B, P_C is the average power (over 50 trials) at the frequency of stimulation in the magnetic-field and control epochs, respectively). Assuming that EEG is unaffected by the field, and that the measured increase in energy associated with the presence of the magnetic field is due to magnetic induction, $\Delta P = a_0(fB)^2$, where a_0 is a constant in proportionality, f is the frequency, and B is the R.M.S. magnetic field strength. If $P_0 = a_0(fB)^2$ when f = 5 Hz and B = 1 Gauss, then ΔP = 2.25 P_0, 4 P_0, and 9 P_0 for 5 Hz and 1.5 Gauss, 10 Hz and 1 Gauss, 10 Hz and 1.5 Gauss, respectively (depicted by the bars). In a group of *5* rabbits *(#5—9)*, the predicted changes were not observed; in only one case (indicated by *) was the Pearson correlation coefficient significantly different from 0. B: each rabbit was killed, and the measurement was repeated. In this case, the power measurements were as predicted by electromagnetic theory based on the assumption that the observed increased power was due solely to an inductive artifact.

delay time determined by the neural propagation path for the pertinent sensory pathway, the state function defined by our procedures was a physiological condition that existed during the presentation of a stimulus, but not after it was removed. Since the state function could be objectively defined, measured, and related statistically to the presence or absence of a light stimulus, it could be used to ascertain the existence of a response to other external stimuli whose effect is also mediated by the nervous system. For this reason, we followed a similar procedure to determine whether magnetic fields affected the BEG.

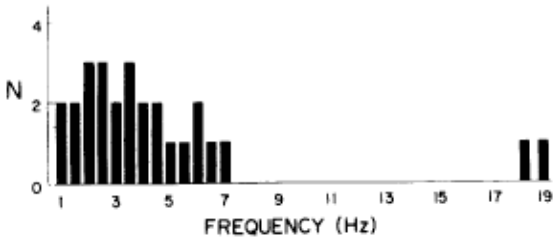


Fig. 7. Distribution of frequencies in which an effect due to light was seen (data from Table I). *N*, number of rabbits that exhibited a statistically significant change in power.

Support was not found for the hypothesis that B(1,25*)* — chosen without regard for a hypothetical physical or physiological resonance mechanism of interaction — could affect the rabbit EEG. Only one animal (out of 9) satisfied the a priori condition for acceptance of a field-induced effect, and the condition was satisfied at only one frequency. No animal exhibited an EEG response under conditions optimized for K^+ resonance, and therefore the IR model of EMF interaction was also not supported by our results. Perhaps Na^+ or Ca^{2+} would have been better choices as potential mediators of a resonance interaction.

A suggestion that optimal EMF interaction would occur at frequencies corresponding to those in the central nervous system was made in 1965[17]. Supporting evidence was provided by observations of entrainment of monkey hippocampal activity[18], and frequency-specific responses

in cat brain[6]. The effects reported here using B(1,5) may have involved electrode artifacts and the issue will not be fully resolved until methods are developed for measuring EEG that are unaffected by the EMF. Such methods are not presently available, but we tested the assertion that the change in power which we measured was solely an induction artifact. If so, when B(1,5) was changed, we should have observed the pattern of change in spectral power shown in Fig. 6. The predicted changes did not occur when the rabbits were alive, but did occur when the measurements were made after the animals were killed. To the extent that the results (Fig. 6) rule out the most likely artifact, the data (Table I) support the hypothesis that B(1,5) produced a physiological change that was reflected in the EEG. The results in Table II independently support the view that the apparent effect of B(1 ,5) was not solely an inductive artifact. In this case we measured the average power before and after exposure to B(1 ,5), and an induction artifact was not possible because EEGs were not measured during presentation of B(1,*5)*. The null hypothesis for this study was that the average change in average energy before and after exposure to the magnetic field was the same as that measured when the animals were subjected to sham exposure; the hypothesis was rejected (Table II) thereby indicating that application of B(1,5) affected ongoing physiological processes at 5 Hz. No other frequency in the periodogram was similarly affected, and consequently the interaction was specific for the frequency of stimulation.

Typically, animal BEG studies employ implanted electrodes (to increase measurement sensitivity) and test statistical hypotheses regarding group means. Our method, which was based on that described by Pfurtscheller and Aranibar[4,31], permitted consideration of hypotheses regarding individual subjects. We sought such a capability because it was our intention to use the method for evaluating the reaction of individual human subjects. For this reason we used surface electrodes and accepted the associated (relative) insensitivity. Placement of the measuring electrode over the occipital region was not optimal for testing the stressor hypothesis (which predicts altered electrical activity in the diencephalon and its efferent targets). It was, however, a good location to validate the overall procedure for deciding whether an external factor caused a change in the EEG. We applied the method to human subjects (where multi-electrode arrays were possible), and observed that sensitivity to 0.25—1.0 Gauss, 10—60 Hz is a general characteristic of human subjects[10,2].

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