Human sensitivity to weak magnetic fields

SIR,—Exposure to electromagnetic fields (EMF) may increase the risk for cancer.[1-3] This implies the existence of a mechanism for detecting the EMF and transducing it into a biological signal. We have been testing the hypothesis that the EMF is a stressor which causes signals in the central nervous system that subserve detection and response.[4] We have found evidence for these signals in rabbits[5] and describe here our first study in man.

After they had given their informed consent fourteen healthy volunteers were exposed to magnetic fields produced by Helmholtz coils.[6] The field strengths were similar to those produced near household electrical appliances. The saggital plane was perpendicular to the coil axis; the head and upper chest were within a field region that was uniform to within 5% of its nominal value. The average background 60 Hz magnetic field was less than 0.1 mG. All measuring equipment was located remotely from the room that contained the coils and the volunteer.

We measured P(f)—the power in the electroencephalogram (EEG) in μV^2 at frequency f in Hz averaged over 2 s as determined by Fourier transformation of the EEG voltage signal— with the field on and off. The magnetic field was presented for 2 s, with a mean period between stimuli of 8 s (range 5-11 s, varied randomly). The volunteer did not know when the magnetic field was on, and there were no visual or auditory cues. The effect of the EMF was assessed by comparing the EEG recorded during the application of the field with that recorded during the 2 s immediately preceding the application. About 60 trials were done, and the first 50 artifact-

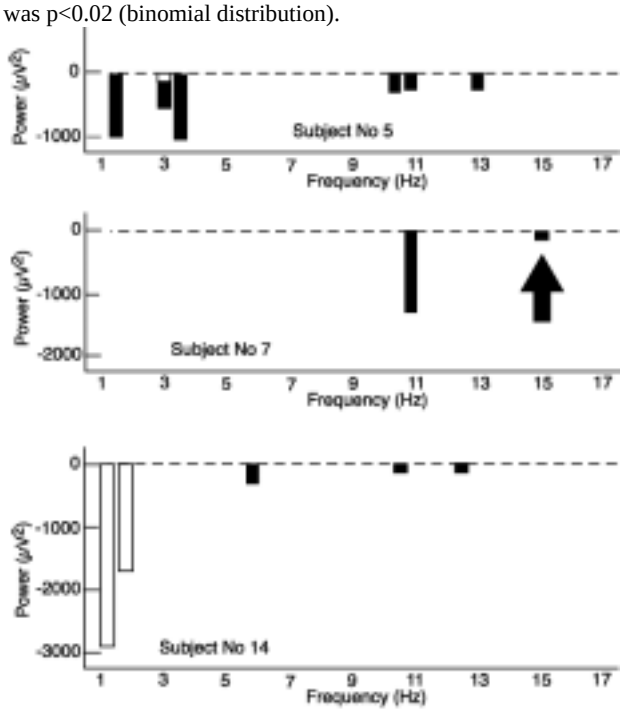
EEG FREQUENCIES AFFECTED BY EXPOSURE TO MAGNETIC FIELDS

Volunteer	Magnetic Field (G)	EEG frequency (Hz) affected
1 (31,F)	0.25	None
2 (37,M)	0.25	O—2.5,17
3 (30,M)	0.25	P—11,12
4 (23,M)	0.5	O—5.5,7
5 (22,F)	0.5	C—1.5,3,3.5,10,10.5,13
6 (47,M)	0.5	None
7 (23,M)	0.5	C—10.5,15
8 (18,M)	0.5	None
9 (25,M)	0.5	None
10 (28,M)	0.25	None
11 (30,M)	0.5	P—11,12
12 (36,M)	0.5	None
13 (35,F)	0.5	None
14 (30,M)	0.25	C—6,10,12; O—1,1.5

C, P, O are central, parietal, and occipital electrodes, respectively.

free ones were used in the subsequent analysis. Sham exposure was used as a control.

The EEG was recorded from the central, parietal, and occipital electrodes (10-20 system) filtered to pass 0.3-35 Hz, and the signal was then divided and simultaneously recorded on an electroencephalograph and sampled at 200 Hz. The coefficients at 1-18.5 Hz in increments of 0.5 Hz were obtained from the Fourier transform and analysed by Wilcoxon signed rank test.[7] The criterion for concluding that a volunteer had detected the magnetic field was that the field produced at least two bilateral successes (difference between corresponding exposed and control epochs significant at $p<0.05$) in at least one pair of electrodes, provided those changes were in the same direction. The probability that an effect might be due to chance was $p<0.02$ (binomial distribution).



Change in EEG power (P) from 3 volunteers at frequencies significantly affected by the magnetic field.

$P=P_S-P_O$, where P_S and P_O are the mean power values recorded during the field and control epochs, respectively. Data from C (n) or O (o) electrodes.

Half the volunteers responded to the EMF by significant changes in EEG (table); no significant effects were observed during sham stimulation. P(f) for the frequencies significantly affected in three volunteers is shown in the figure. In all cases, less power was observed during the stimulus epochs compared with the control epochs.

Since the field-on and field-off epochs lasted only 2 s the locus of field transduction was probably in the nervous system— either a neuron or a perineural cell. Classic somatosensory pathways involve spike potentials and conscious perception of the stimulus, but information may be added at non-spiking regions of a neuron via voltage-gated channels whose cumulative effect is encoded by a subsequent spike.[8] If the EMF produced an afferent signal consisting of such a modification of spontaneous neural activity, that could explain our observation of altered brain electrical activity in the absence of conscious perception.

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