CHAPTER 8

Effects of Electromagnetic Energy on Biological Functions

Introduction

In the exploration of a new field of research, many experiments unavoidably are "fishing expeditions" in which a large number of variables are assayed. Often, valuable information is obtained in unexpected areas under such circumstances, and this leads to the problem of piecing together diverse results into a self-consistent viewpoint. In this chapter we review reports of effects in the areas of metabolism, reproduction, growth and healing, and mutagenicity.

Intermediary Metabolism

Metabolic indices of carbohydrate metabolism are sensitive to EMFs (1-6). Dumanskiy and Tomashevskaya (1) exposed rats to 2.4 GHz (2 hr./day), for up to 4 months. At 100 and 1000 μW/cm2 the animals exhibited a series of biochemical alterations in liver tissue that included a decline in cytochrome oxidase activity, an increase in glucose-6-phosphate dehydrogenase activity, and an activation of mixed-function oxidases in the microsomal fraction of the tissue. The largest changes were seen after 1 month's irradiation, following which there was a tendency for the various enzyme levels to return to baseline. Enzyme activities were unaffected by exposure to 10 μW/cm2. In another study, Dumansky et al. reported an increase in blood glucose in humans following exposure to 15 kv/m 50 Hz, I.5 hours/day for 6 days (2).

Chernysheva and Kholodov studied the effect of a 90-gauss, 50 Hz magnetic field on several aspects of carbohydrate, protein, and nucleic-acid metabolism in the rat (3). They found EMF-induced alterations in each area, including changes in liver glycogen, elimination of ammonia, glutamine content in the heart, and nucleic-acid levels in brain and liver (Table 8.1).

Table 8.1. Metabolic Parameters in Rats (in mg%) Exposed for 6 Months to 90 Gauss (3 Hr/Day)

TISSUE	PARAMETER				
	Glucose	Glycogen	Glutamine	RNA	DNA
Liver					
C	220 ± 27.5	1782.3 ± 214	8.5 ± 0.55	64.5 = 3	27.3 ± 0.7
E	179 ± 15.6	823.4 ± 147*	7.4 ± 0.33	78.3 ± 4*	31.0 ± 1.3*
Heart					
C	92.3 ± 4.4	593.8 ± 56.5	7.05 ± 0.30		
E	78.2 ± 3.6*	613.0 ± 32.3	8.65 ± 0.60		

NOTE: Date averaged over 8-10 animals. C, control; E, experimental.

$p < 0.05$

In a study of muscle metabolism (4), lactate dehydrogenase activity in skeletal and cardiac muscle of rats was measured by disk electrophoresis. There was an increase in the enzyme's activity in both kinds of muscle 1-2 days after exposure to 200 gauss, 50 Hz, for 24 hours; histological changes indicative of glycolytic processes were also found. These observations were consistent with an earlier report of impaired functional activity of muscle following EMF exposure (5). After 1 month, rabbits exposed to 30-40 kv/m, 05 Hz, were unable to lift a weight as large as that lifted by the nonexposed rabbits.

The sensitivity of metabolic parameters to EMFs is underscored by studies that involve EMFs which have intensities comparable to typical environmental fields; the Mathewson et al. study (6) is a blood example. Rats were exposed for 28 days to 2, 10, 20, 50 and 100 v/m in three replicate experiments, following which complete blood chemistries were performed; the serum glucose levels are listed in table 8.2A. Although some differences between the control and exposed groups were seen, no trend or dose-effect relationship was manifested and consequently, the authors regarded the data as having failed to show a biological effect of the EMF (6). But the 60-Hz electric field in the test cages was 0.18-9.15 v/m, depending on the particular test cage location (8). As a consequence, the 45-Hz, 2-v/m group is more properly viewed as a control group in relation to the 50-100 v/m exposed groups. When we did this, the Mathewson data revealed significant increases in serum glucose in each replicate (Table

Table 8.2. Average Glucose Levels in Three Replicate Experiments

Experiment		Serum Glucose Levels (mg/dl)					
		Control	2 v/m	10 v/m	20 v/m	50 v/m	100 v/m
(A)	1	281.1 ± 83.8	176.3 ± 74.4	259.4 ± 124.6	218.9 ± 100	286.0 ± 156.9	235.8 ± 49.3
	2	210.4 ± 55.4	237.3 ± 62.2	259.4 ± 95.9	241.6 ± 149.1	256.2 ± 118.6	269.4 ± 95.9
	3	187.2 ± 34.4	199.0 ± 30.8	199.1 ± 34.3	201.3 ± 40.9	199.1 ± 34.2	232.3 ± 44.0
		Serum Glucose Levels (mg/dl)					
		Control + 2 v/m		50 + 100 v/m		v/m	
(B)	1	228.7 ± 94.4		260.9 ± 117.2		p = 0.23	
	2	223.9 ± 59.5		284.2 ± 116.0		p < 0.02	
	3	193.7 ± 32.7		219.7 ± 42.4		p < 0.01	

NOTE: (A), data analysis reported by Mathewson et al. (6); (B), analysis of Mathewson data (9) by Marino and Becker (7), taking into account the 60-Hz background fields (8).

8.2B). (This approach to the Mathewson data also suggests the existence of effects on other parameters, including globulins, protolipids, and triglycerides.)

Cellular bioenergetics can be altered by EMFs (10-14): the changes seem to be adaptive in nature, and to depend on the exposure level and duration. A single 10-minute exposure at 25 $\mu W/cm^2$, 10 GHz, produced a decrease in the phosphorylation effectiveness factor (ADP/O) in liver mitochondria, and an increase in respiratory control (RC) in kidney mitochondria (10). After ten such exposures, the oxygen consumption and RC were both increased in kidney mitochondria. A single exposure at 100 $\mu W/cm^2$ caused a rise in oxygen consumption and an increase in ADP/O in liver mitochondria and a decrease in RC in kidney mitochondria (10). After ten such exposures, almost all the indices of oxidative phosphorylation in both mitochondria returned to normal, thereby suggesting that the enzyme systems had adapted to the EMF. A decrease in RC was also seen in guinea pig mitochondria exposed in vitro to 155 v/m, 60 Hz (11).

Rats were exposed to 10, 25, 50, 100, 500 and 1000 $\mu W/cm^2$, at 2.4 GHz, as follows: 40 minutes per day, 3 times per day, 5 days per week, for 4 months (intended to simulate the exposure received from household microwave ovens) (12). It was found that the EMF altered respiration and phosphorylation in liver mitochondria; there was an increase of non-phosphorylating oxidation of metabolites of the Krebs cycle, and a decrease in the oxygen consumption rates during phosphorylating respiration. A decrease in oxygen consumption rate was also found after 20 days' exposure to 1000 $\mu W/cm^2$, 46 GHz (13).

In a study of skeletal-muscle metabolism, rats were exposed to 300-900 gauss, 7 KHz for up to 6 months (1.5 hr./day) (14). Creatine phosphate and ATP levels decreased, and ADP levels increased following exposure. The changes were consistent with both an increased energy requirement, and an adverse effect on ATP formation. On the basis of *in vitro* studies of oxidative phosphorylation and oxygen consumption involving tissues from the exposed animals, the authors favored the latter possibility. Two consequences of the observed changes in cell bioenergetics involved carbohydrate and nitrogen metabolism. Decreased glycogen levels were found, indicating a compensatory glycogenolysis and, hence, an enhanced production of high-energy phosphate compounds. Secondly, EMF exposure produced an increase in tissue ammonia levels with no corresponding increase in glutamine synthesis. This may have been due to the ATP deficiency, although the influence of other factors involved in glutamine production—glutamic acid and manganese for example—could not be excluded.

Shandala and Nozracher (15) reported that kidney function and water-

salt metabolism in rabbits (diuresis, chloride elimination, acid-base balance) were altered following the exposure to 50 and 500 $\mu W/cm^2$, 2.4 GHz. In a comparable study (16), it was found that similar kinds of changes (urinary levels of potassium, sodium and nitrogen) were sex dependent; most of the metabolite levels were increased in females and decreased in males.

The altered nitrogen levels (16) suggested an EMF effect on protein synthesis. This was confirmed by Miro et al. (17) who found that 160 hours' exposure of mice to 2000 μW/cm2, 3 GHz, resulted in an increase in protein synthesis in the liver, thymus, and spleen as determined by cytohistological techniques.

The most important study to date on lipid metabolism was performed by Dietrich Beischer and his colleagues (18). Volunteers, confined for up to 7 days, were exposed to a 1-gauss magnetic field, 45 Hz, for 24 hours: they did not know which 24-hour period during their confinement would be chosen for the application of the EMF. It was found that the serum triglycerides in 9 of 10 exposed subjects reached a maximum value 1-2 days after EMF exposure (Fig. 8.1); similar trends were not seen in any of

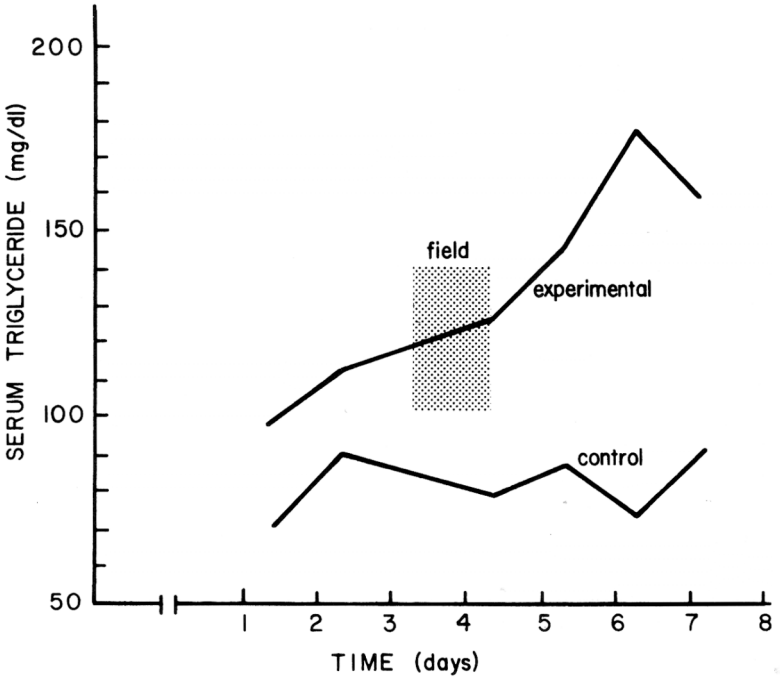

Fig. 8.1. Average serum triglyceride levels of exposed and control subjects.

the control subjects (18). Measurement of respiratory quotients for basal conditions established that the hyperlipemia could not have been caused by a change in the proportion of fats and carbohydrates being oxidized. Also, previous work had shown that confinement alone had no effect on serum triglycerides. This suggested that the observed effect may have been due to a change in the activity of one or several of the enzymes involved in lipid homeostatis, perhaps triglyceride lipase. The 1-2 day latency suggested that the action of the EMF involved an enzyme precursor, not the enzyme itself (the EMF influence would then be felt only after existing enzyme stores had been depleted).

There are several other studies involving low-frequency magnetic field effects on fat metabolism (19, 20). Rabbits that were maintained on a high-cholesterol diet were exposed to the field for 5 weeks and then examined for serum lipid levels and aortic plaque formation (19). A reduction of both cholesterinemia and plaque formation was found in the exposed animals. A reduction in blood cholesterol (50 mg/ml on the average) was also reported in ten human subjects following local application of a magnetic field (20).

Vitamin B_6 (pyridoxine) is involved in the nonoxidative degradation of amino acids, synthesis of unsaturated fats, and the hydrolysis of glycogen. Exposure of rats to 570 $\mu W/cm^2$, 2 GHz, for 15 days (3 hr./day) led to a decrease in vitamin B6 levels in blood, brain, liver, kidney, and heart; the levels of the vitamin in skeletal muscle increased (21) (Table 8.3).

Table 8.3. Effect of EMF on Vitamin B_6 Levels in Rat Tissues

TISSUE	VITAMIN B_6 (µg/g-tissue)		
	Control	Experimental	Statistical Significance
Blood*	0.072 ± 0.010	0.046 ± 0.009	< 0.001
Brain	3.7 ± 0.48	2.3 ± 0.19	< 0.05
Liver	3.9 ± 0.37	2.8 ± 0.20	< 0.05
Kidney	5.6 ± 0.47	3.8 ± 0.23	< 0.01
Heart	4.9 ± 0.45	2.0 ± 0.37	< 0.01
Muscle	3.45 ± 0.44	5.10 ± 0.40	< 0.02

*µg/ml

Trace levels of many metallic elements are found in body tissues; they are known to take part in enzyme activation, formation of proteins, redox reactions, and possibly in other biochemical processes. Both high- and low-frequency EMFs have been found capable of altering body trace-element distribution (22-24). Groups of 10 rats each were exposed to 2.4 GHz at 10, 100, and 1000 $\mu W/cm^2$, 8 hours/day for 3 months. At the end of the exposure period, the animals were sacrificed and the levels of

Table 8.4. Trace Elements in Rat Tissues following Exposure at 2.4 GHz

TISSUE	TRACE ELEMENT LEVELS (μg%, fresh weight)			
	Control	10 ($\mu W/cm^2$)	100 ($\mu W/cm^2$)	1000 ($\mu W/cm^2$)
Copper				
Liver	446.8 ± 12.3	416.5 ± 10.4*	389.8 ± 17.6*	331.1 ± 15.3*
Kidney	479.2 ± 25.4	405.9 ± 10.6*	309.2 ± 18.3*	398.2 ± 18.7*
Bone	237.7 ± 12.8	226.8 ± 15.3	266.5 ± 20.8	277.7 ± 25.3
Teeth	344.0 ± 25.3	360.0 ± 19.8	312.2 ± 26.7	306.3 ± 23.2
Bone marrow	380.0 ± 22.7	396.0 ± 29.3	540.3 ± 40.4*	673.2 ± 61.3*
Spleen	185.0 ± 11.4	203.5 ± 14.2	486.5 ± 30.9*	971.2 ± 60.4*
Brain	198.0 ± 13.5	198.0 ± 15.3	233.6 ± 11.1	298.2 ± 28.4*
Lung	258.0 ± 26.2	389.6 ± 35.3*	525.0 ± 50.4*	913 ± 82.4*
Myocardium	113.4 ± 8.2	142.8 ± 7.4*	189.2 ± 17.9*	266.6 ± 27.3*
Skeletal muscle	27.6 ± 1.5	31.0 ± 2.9	49.2 ± 5.1*	15.2 ± 1.8*
Skin	66.0 ± 4.8	24.5 ± 3.3*	40.7 ± 4.3*	81.3 ± 9.3*
Blood	56.2 ± 4.4	66.0 ± 5.7	85.0 ± 7.4*	74.0 ± 4.5*
Manganese				
Liver	139.2 ± 7.1	167.3 ± 10.6*	221.3 ± 14.7*	230.0 ± 16.9*
Kidney	45.2 ± 2.6	48.3 ± 3.1	66.7 ± 4.1*	70.3 ± 3.1*
Bone	71.3 ± 3.6	63.2 ± 4.7	60.5 ± 3.9	55.2 ± 2.6*
Teeth	83.9 ± 5.1	93.3 ± 4.9	97.1 ± 6.1	69.2 ± 4.3*
Spleen	9.8 ± 0.4	16.6 ± 0.8	17.8 ± 0.8*	29.4 ± 1.7*
Brain	22.0 ± 0.8	24.1 ± 1.5	23.7 ± 1.2	24.8 ± 0.9*
Myocardium	13.8 ± 0.7	15.6 ± 1.1	18.5 ± 1.2*	20.3 ± 1.2*
Skeletal muscle	6.6 ± 0.3	5.8 ± 0.2	6.8 ± 0.4	7.8 ± 0.3*
Lung	15.2 ± 1.2	18.6 ± 1.4	12.7 ± 1.1	10.3 ± 0.9*
Skin	6.5 ± 0.3	6.6 ± 0.4	8.0 ± 0.6	9.1 ± 0.5*
Blood	2.1 ± 0.1	2.1 ± 0.1	2.2 ± 0.1	2.8 ± 0.1*
Molybdenum				
Liver	58.9 ± 3.2	57.8 ± 2.9	47.9 ± 3.1*	40.8 ± 2.5*
Kidney	9.5 ± 0.5	12.9 ± 1.1*	15.2 ± 1.4*	24.0 ± 2.8*
Femur	532.1 ± 11.6	532.6 ± 12.4	514.1 ± 11.9	485.6 ± 12.4*
Teeth	718.8 ± 19.8	714.8 ± 15.2	710.8 ± 16.7	907.1 ± 32.3*
Spleen	9.7 ± 0.4	8.3 ± 0.2	6.9 ± 0.2*	6.1 ± 0.4*
Brain	9.9 ± 0.3	8.9 ± 0.24*	7.9 ± 0.9*	5.9 ± 0.6*
Lung	5.8 ± 0.1	4.8 ± 0.18*	4.3 ± 0.1*	3.8 ± 0.25*
Myocardium	3.6 ± 0.1	3.6 ± 0.4	3.6 ± 0.3	3.6 ±0.2
Skeletal muscle	7.8 ± 0.32	5.3 ± 0.4*	3.3 ± 0.27*	2.2 ± 0.15*
Skin	4.4 ± 0.1	3.2 ± 0.2*	2.6 ± 0.18*	9.7 ± 0.32*
Blood	2.4 ± 0.12	2.4 ± 0.14	1.7 ± 0.2*	1.4 ± 0.14*
Nickel				
Liver	35.6 ± 2.53	25.1 ± 1.9*	23.2 ± 1.7*	19.9 ± 0.8*
Kidney	33.9 ± 2.1	24.0 ± 2.2*	19.8 ± 2.1*	15.1 ± 1.7*
Femur	435.3 ± 24.4	338.4 ± 17.0	336.2 ± 23.1	473.0 ± 38.4*
Teeth	285.8 ± 19.7	352.1 ± 24.1*	448.1 ± 33.4*	638.9 ± 41.6*
Spleen	33.3 ± 4.2	31.4 ± 2.7	29.2 ± 1.8	18.1 ± 1.2*
Brain	27.9 ± 2.5	22.2 ± 2.7	37.9 ± 3.1*	49.7 ± 5.2*
Lung	28.0 ± 3.1	28.8 ± 2.9	25.2 ± 2.9	16.2 ± 1.4*
Myocardium	15.0 ± 0.9	35.7 ± 3.9*	84.2 ± 6.7*	106.0 ± 13.8*
Skeletal muscle	7.9 ± 0.9	5.5 ± 0.4	3.3 ± 0.6*	1.2 ± 0.1*
Skin	22.6 ± 0.7	38.3 ± 1.4*	43.5 ± 3.4*	23.5 ± 1.2
Blood	10.7 ± 0.9	11.2 ± 0.6	14.4 ± 0.8*	21.4 ± 1.0*

$p < 0.05$

copper, manganese, nickel and molybdenum in the major organs were determined by optical spectroscopy. Changes in the level and distribution of all four elements were found (Table 8.4). The copper level decreased in both liver and kidney, possibly as a result of increased synthesis of ceruloplasmin-this would be consistent with the observed increase of copper in blood. There was, generally, an increase in copper in those organs that use the element in hemopoiesis and redox processes, possibly indicating a basic compensatory response to EMF radiation. The copper content of hard tissue was virtually unchanged by the field.

In comparison to copper, manganese metabolism was less influenced by the EMF; it increased in most organs, and decreased in hard tissue.

Teeth and bone were the principal reservoirs for molybdenum, and they exhibited no change in molybdenum concentration except following exposure to the highest strength EMF. In contrast, the molybdenum levels in the soft tissues, which accounted for less than 10% of the total body molybdenum, were altered at even 10 $\mu W/cm^2$.

The content of nickel in the various organs was influenced by each EMF intensity. It rose in some tissues, and fell in others; the heart, which exhibited a sixfold increase, was the most strongly affected tissue.

Trace element analysis has also been performed on rats exposed to low-frequency EMFs. Following exposure to 1, 2, 4, 7, and 15 kv/m, 50 Hz, for 4 months (2 hr./day), significant changes were found in the distributions of copper, molybdenum, and iron among the tissues, even at 1 kv/m, the lowest field strength employed (23) (Table 8.5). In subsequent studies by the same authors, similar changes were found after exposure to 7-15 kv/m for only 30 minutes/day (24).

Reproduction, Growth and Healing

Studies of the cells and organs of the reproductive system have revealed a general debilitating effect of EMF exposure (25-30). Altered spermatogenesis was reported in rats following exposure to 5000 v/m, 50 Hz, for up to 4.5 months (25). After 1.5 months' exposure, the number of atypical sperm cells was significantly greater in the exposed animals (30.7% vs. 15.9%, $p<0.0I$); the percentage of pathological forms increased with the duration of exposure and reached 36.8% after 4.5 months. The exposed rats also produced fewer sperm cells and exhibited a higher ratio of living to dead cells; both effects became significant after 3.5 months. Comparison of the parameters of respiration and phosphorylation of testicular mitochondria following 4.5 months' exposure revealed decreased phosphorylatic respiration, speed of phosphorylation of ADP, and respiratory control.

Table 8.5. TRACE ELEMENTS IN RAT TISSUES FOLLOWING EXPOSURE AT 50 HZ

TISSUE	TRACE ELEMENT LEVELS (µg%, fresh weight)					
	Control	1 kv/m	2 kv/m	4 kv/m	7 kv/m	15 kv/m
Copper						
Liver	759.3 ± 39.9	603.0 ± 42.2*	603.1 ± 24.1*	389.4 ± 19.5*	331.4 ± 19.9*	288.7 ± 14.4*
Kidney	224.1 ± 13.4	224.1 ± 11.2	346.9 ± 20.8*	380.4 ± 15.2*	537.4 ± 37.6*	724.9 ± 36.2*
Spleen	65.6 ± 2.6	94.9 ± 5.7*	94.9 ± 4.7*	106.4 ± 7.4*	108.9 ± 7.6*	176.7 ± 10.6*
Brain	58.4 ± 3.5	61.2 ± 4.3	64.1 ± 4.5	193.5 ± 9.7	260.9 + 10.4*	306.7 ± 21.4*
Myocardium	106.0 ± 7.4	111.0 ± 4.4	111.1 ± 5.5	168.1 ± 11.8*	221.6 ± 11.1*	278.9 ± 11.1*
Skeletal muscle	19.5 ± 1.2	21.4 ± 1.7	30.9 ± 2.2*	25.8 ± 2.1*	25.5 ± 0.6*	24.6 ± 1.5*
Skin	11.5 ± 0.6	9.7 ± 0.7	8.7 ± 0.5*	13.5 ± 0.7*	16.9 ± 1.0*	19.5 ± 0.8*
Bone	335.4 ± 26.8	376.4 ± 22.6	496.2 ± 19.8*	496.2 ± 37.7*	624.6 ± 43.7*	823.5 ± 41.2*
Teeth	237.8 ± 16.6	285.9 ± 14.3	285.9 ± 17.1	611.6 ± 36.7*	806.1 ± 32.2*	833.7 ± 53.0*
Molybdenum						
Liver	42.7 ± 2.6	40.8 ± 2.8	40.8 ± 1.6	29.5 ± 1.5*	26.3 ± 1.3*	23.5 ± 1.2*
Kidney	2.1	27.4 ± 0.8	27.4 ± 0.8	26.9 ± 1.6	32.4 ± 1.9	36.3 ± 1.4
Spleen	8.6 ± 0.4	7.7 ± 0.5	5.7 ± 0.3*	5.7 ± 0.4*	5.6 ± 0.3*	4.8 ± 0.2*
Brain	14.0 ± 0.8	14.2 ± 0.7	9.7 ± 0.8*	7.9 ± 0.5*	7.9 ± 0.5*	5.9 ± 0.3*
Myocardium	3/6 ± 0.1	3.6 ± 0.2	3.6 ± 0.2	3.6 ± 0.2	2.7 ± 0.1	2.7 ± 0.1
Skeletal muscle	3.3 ± 0.1	3.3 ± 0.3	3.3 ± 0.1	1.6 ± 0.1*	2.5 ± 0.1*	9.6 ± 0.6*
Skin	2.9 ± 0.2	3.3 ± 0.3	4.4 ± 0.2*	6.6 ± 0.3*	2.1 ± 0.1*	1.1 ± 0.05*
Bone	1110.6 ± 55.5	1180.0 ± 59.0	1398.2 ± 83.9*	804.5 ± 64.3*	804.5 ± 56.3*	519.4 ± 31.1*
Teeth	1086.8 ± 76.1	1038.3 ± 62.3	1038.3 ± 72.7	968.7 ± 77.5	824.4 ± 41.2*	640.1 ± 25.6*
Iron						
Liver	39.8 ± 2.4	29.5 ± 1.8*	12.6 ± 0.6*	11.5 ± 0.4*	11.5 ± 0.6*	10.9 ± 0.6*
Kidney	9.1 ± 0.4	9.1 ± 0.4	11.2 ± 0.4*	13.5 ± 0.6*	14.5 ± 0.8*	14.7 ± 1.0*
Spleen	4.3 ± 0.3	5.6 ± 0.3*	5.6 ± 0.4*	5.6 ± 0.3*	4.8 ± 0.3	3.4 ± 0.2*
Brain	12.5 ± 0.8	9.7 ± 0.6*	8.4 ± 0.4*	8.4 ± 0.5*	8.5 ± 0.7*	8.2 ± 0.6*
Myocardium	2.8 ± 0.2	2.8 ± 0.2	2.8 ± 0.1	2.9 ± 0.1	2.9 ± 0.2	3.2 ± 0.2
Skeletal muscle	0.5 ± 0.02	0.5 ± 0.03	0.5 ± 0.01	0.5 ± 0.03	0.5 ± 0.03	0.4 ± 0.01
Skin	2.6 ± 0.1	2.1 ± 0.08*	2.1 ± 0.1*	2.1 ± 0.09*	1.8 ± 0.09*	1.6 ± 0.06*
Bone	37.6 ± 1.5	31.3 ± 1.6*	21.2 ± 1.3*	21.2 ± 1.5*	16.8 ± 0.7*	9.4 ± 0.5*
Teeth	28.6 ± 2.0	22.7 ± 1.8	22.7 ± 1.3*	22.7 ± 1.1*	22.7 ± 0.9*	12.8 ± 0.5*

$p < 0.05$

In a study of carbohydrate metabolism in testicular tissue, Udinstev and Khlyin exposed rats continuously (for 24 hr.) or intermittently (6.5 hr./ day, for 5 days) to 200 gauss, 50 Hz (26). In the case of the 24-hour exposure, he observed a brief initial activation of enzyme activity followed by a depression of activity and then a return to normal levels. Intermittent exposure to the field, however, was characterized by a prolonged depresssion of the activity of several enzymes, including hexokinase, glucose-6-phosphate dehydrogenase, and cytochromoxidase. These changes pointed to a depression in tissue respiration which would be consistent with the authors' previous work that showed a decrease in testosterone production following exposure to the EMF.

Chronic exposure of mice to a 7-KHz pulsed magnetic field produced morphological changes in the testes of rats: the seminal epithelium, ducts and sperm cells were each altered at 30 gauss, but not at 5 gauss (27).

Female rats exhibited estrous-cycle dysfunction and some pathological changes in the uterus and ovaries following exposure to 5 kv/m 50 Hz (28). In males, the EMF caused a decrease sperm count and an increase in the number of dead and atypical spermatozoa. When the exposed animals were mated with unexposed rats, decreased birth rates and increased postnatal mortality were found in the offspring (28). Constant exposure to a 130-140 gauss magnetic field, both DC and so Hz, also produced changes in the estrous-cycle of female rats (29). Disturbances in ovarian morphology and fertility, and alterations in postembryonic development were seen following exposure of female mice to 10-50 $\mu W/cm^2$, 2.4 GHz (30).

Because the developing organism is particularly sensitive to external influences, several investigators have exposed immature animals to EMFs and studied their impact on growth rate. Rats exposed to an intermittent EMF at 3 GHz, 153 μW/cm2, exhibited a smaller weight gain than the control animals (31). The difference became statistically significant after 4 months' exposure, and it persisted during the subsequent 3 months' exposure.

Noval et al. (32), studied the effect on growth rate of rats of exposure to 0.5-100 v/m, 45 Hz, as compared to the growth rate of control rats maintained under Farady-cage conditions. He found a consistent depresssion of the body weights of the exposed animals, even for fields as low as 0.5 v/m (Table 8.6). Low-frequency fields—electric and magnetic—also produced growth depression in 25-day-old chicks (33).

By the mid-190's, no studies had been done to assess the possible impact on successive generations of animals of the continuous presence of a low-frequency EMF; we therefore undertook such a study (34). Initially, mature male and female mice were split into horizontal, vertical, and

Table 8.6. CHANGES IN AVERAGE BODY WEIGHTS OF RATS EXPOSED TO 45-HZ VERTICAL ELECTRIC FIELDS

EXPERIMENT	FIELD (v/m)	NO. OF RATS	EXPOSURE TIME (days)	WEIGHT GAIN (gm)
1	25-100	143	36	142 ± 14*
	control	47	36	209 ± 20
2	10-50	47	40	150 ± 19*
	control	16	40	215 ± 11
3	2-10	94	32	131 ± 12*
	control	32	32	166 ± 12
4	0.5-2	32	30	131 ± 11*
	control	32	30	170 ± 11

NOTE: The control rats were housed in a field-free environment.
*$p < 0.001$

control groups. Mice in the horizontal group were allowed to mate, gestate, deliver, and rear their offspring in a horizontal 60-Hz electric field of 10 kv/m. At maturity, randomly selected individuals from the first generation were similarly allowed to mate and rear their offspring while being continuously exposed. Randomly selected individuals from the second generation were then mated to produce the third and final generation. A parallel procedure was followed for the vertical group wherein three generations were produced in a 60 Hz vertical electric field of 15 kv/m, and for the control group wherein three generations were produced in the ambient laboratory electric field. In the first and second generations, males and females reared in both the horizontal and vertical electric field were significantly smaller than the controls when weighed at 35 days after birth In the third generation, the only group whose body weights were significantly affected were the males exposed to the vertical field. In both the second and third generation, a large mortality rate in the vertical-field mice was seen during the 8-35 day postpartum period. We repeated the multigeneration study at 3.5 kv/m using an improved exposure system (55) (Fig. 8.2 and 8.3). In the first generation, no consistent effect on body weight attributable to the EMF was seen throughout a 63-day observation period. In both the vertical and horizontal groups, however, infant mortality was increased; in the vertical-control group 48 animals (about 17%) died between birth and weaning. In the vertical-exposed group, if the electric field wasn't a causative factor, a 17% mortality rate should also have been seen. However, that group exhibited a 31% mortality—82 animals died and not the expected 44. Thus, 38 animals, about 16% of those born, failed to live to weaning because of the electric field. A similar result was obtained in the horizontal-exposed group—about 11% of the animals born failed to live to weaning because of the electric field.

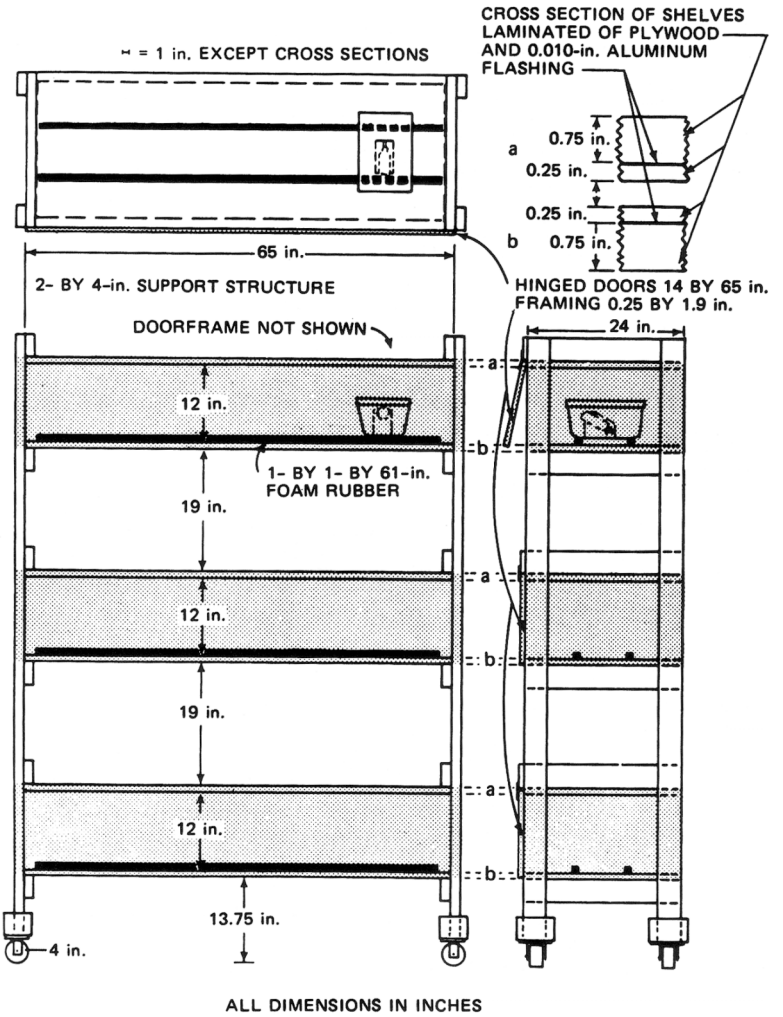

Fig. 8.2. Assembly for vertical-field study. The metal plates were grounded in the control assembly.

In the second generation, no consistent effect on body weight attributable to the field was seen throughout a 108-day observation period. The vertical-exposed group, however, again exhibited a higher mortality; about 6% of the animals alive at weaning failed to live to the final day of observation due to the presence of the electric field. In the third generation,

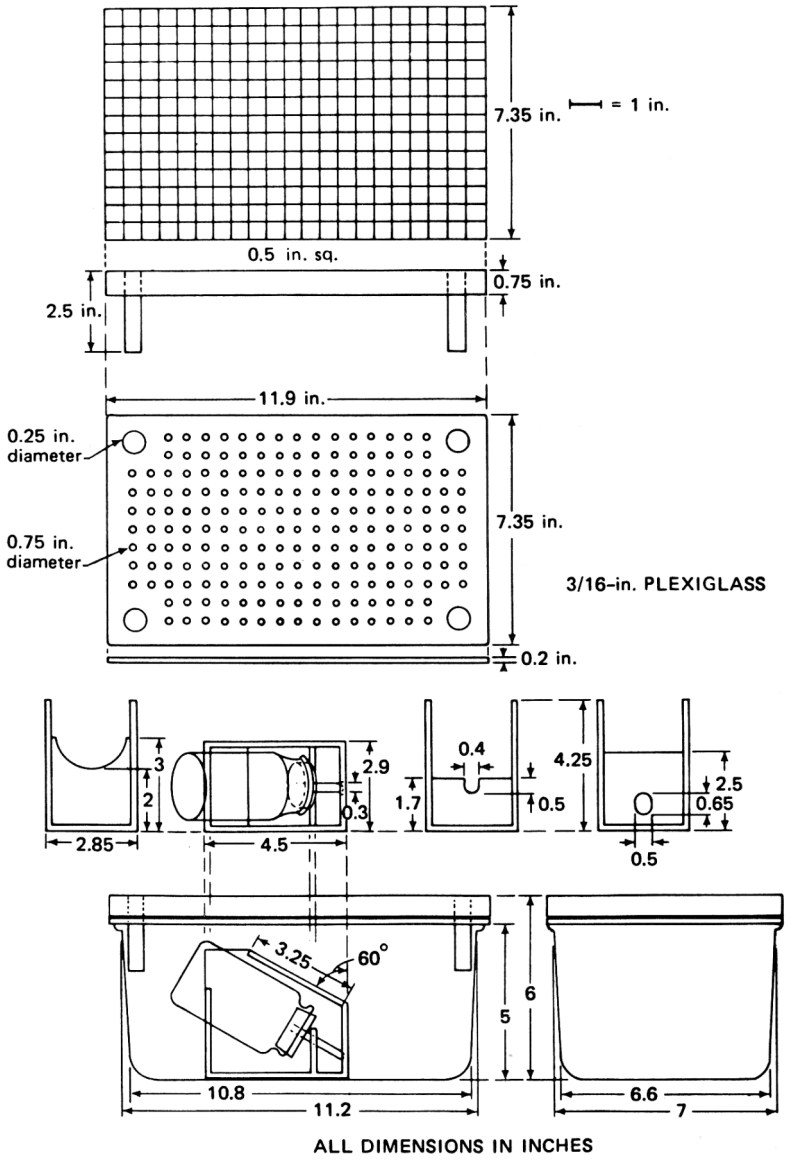

Fig. 8.3. Cage and water-bottle holder.

the exposed animals had higher body weights, particularly in the horizontal-exposed group. At 49 days after birth, the males and females in each exposed group were significantly heavier than their respective controls. At 119 days after birth only the females in the horizontal-exposed group were significantly heavier, but this was part of a consistent trend for that group. Again we saw an increased mortality in the vertical-exposed group—10% of the weaned animals failed to survive to the end because of the electric field. Heavier body weights in animals (monkeys) exposed to low-frequency EMFs were also reported by Grissett (35).

EMFs can alter the growth and development of some tumors. Batkin and Tabrah found that the development of a transplanted neural tumor could be affected by a 12-gauss, 60 Hz EMF (36); they reported a slowing of early tumor growth in the exposed mice. We found that 5 kv/m, 60 Hz, had no material effect on the development of Erhlich ascites tumor in mice; the average length of time between tumor implant and death was not altered by the fields.

The process of wound-healing has been found to be susceptible to EMFs; not surprisingly, the nature of the effect depends on both the exposure conditions and the particular EMFs employed (37-40). One of the first such reports was that of Bassett et al. (37) involving dogs. Electrical circuits, attached to leg bones that had been surgically fractured, produced a pulsed 65-Hz magnetic field at the fracture site. After 28 days, the organization and strength of the repair process as judged by the mechanical strength of the healing callus had increased significantly. We observed an opposite effect on fracture healing in rats exposed to a

Table 8.7. HISTOLOGICAL GRADINGS OF RATS EXPOSED TO 5 KV/M

CRITERION	REPLICATE 1		REPLICATE 2	
	Control (N = 17)	Experimental (N = 18)	Control (N = 20)	Experimental (N = 20)
Union	5.3 ± 0.9	4.2 ± 1.0*	5.4 ± 0.8	4.4 ± 0.8*
Alignment	1.9 ± 0.3	1.4 ± 0.7	1.6 ± 0.7	1.4 ± 0.8
Callus size	2.9 ± 0.8	1.9 ± 0.8*	3.0 ± 1.0	2.0 ± 0.9*
Anchoring callus	8.2 ± 1.8	5.2 ± 1.2*	8.2 ± 1.5	4.6 ± 1.4*
Bridging callus	6.8 ± 1.9	4.0 ± 0.9*	6.7 ± 1.5	3.9 ± 1.2*
Uniting callus	7.0 ± 2.1	3.8 ± 1.2*	5.6 ± 1.2	3.7 ± 0.9*
Sealing callus	7.3 ± 2.0	4.6 ± 1.5*	6.8 ± 1.1	4.2 ± 1.7*
Healing index	39.3 ± 7.7	25.2 ± 3.5**	37.2 ± 6.1	24.0 ± 3.2**

NOTE: Numerical scales were as follows: Union (1-7); Alignment (0-2); Callus Size (1-4); Callus (0-5).

*$p < 0.01$
**$p < 0.001$

full-body vertical electric field of 5 kv/m, 50 Hz (38). Midshaft fractures were done on the rats following which half the group was exposed to the field and half was maintained as a control. The rats were housed individually in plastic enclosures maintained in wooden exposure assemblies (see Figures 8.2 and 8.3). The extent of healing was evaluated at 14 days postfracture on the basis of blind scoring of serial microscopic sections. We used a numerical grading system that characterized both the healing process as a whole, and its anatomical components. In two replicate studies, we found a highly significant retardation in fracture healing (Table 8.7); the fractures in the exposed rats exhibited the development normally seen in a 10-day fracture. We found no effect on fracture-healing following exposure at 1 kv/m. The adverse effect of a 60 Hz electric field on fracture healing in the rat was confirmed by Phillips in three replicate studies (39).

There is also a report of a beneficial effect of microwave EMFs on healing (40). Under sterile conditions, a linear 5-cm wound down to the dermis was made on the backs of guinea pigs. The wounds were then closed and sutured and the animals were exposed to 4000 $\mu W/cm^2$ and sacrificed up to I1 days after surgery. Microscopically, the wounds from the exposed animals exhibited a more advanced stage of healing, and this was confirmed by mechanical testing data; from 30% to 72% more force was required to re-open the wounds of the animals exposed to the EMF (Table 8.8).

Table 8.8. EFFECT OF EMF EXPOSURE ON THE FORCE REQUIRED TO DISRUPT A SKIN WOUND

DURATION OF EXPOSURE (days)	FORCE (gm)	
	Control	Experimental
3	220 ± 2.3	340 ± 2.7*
5	360 ± 2.5	520 ± 2.5*
7	460 ± 2.1	790 ± 2.3*
9	680 ± 2.4	1050 ± 2.3*
11	1100 ± 2.7	1420 ± 2.6*

NOTE: Each control and experimental group consisted of 6 and 10 animals respectively.
*$p < 0.001$

Mutagenesis

Rats were exposed for 7 hours/day to 50 and 500 $\mu W/cm^2$, 2.4 GHz (total exposure of 1 and 10 days at the higher and lower intensities respectively) (41). At 50 $\mu W/cm^2$, the number of chromosomal abnormalities increased by 55% compared to the controls when assayed 18 hours after the end of the exposure period; 2 weeks after exposure the increase was

150% . Eighteen hours following exposure at 500 $\mu W/cm^2$, the number of chromosomal abnormalities was more than 5 times that of the controls, and it remained elevated (340%) even after z weeks. In a comparable study (42) (3 GHz, 3500 $\mu W/cm^2$, 3 hr./day for 3 mo.) mitotic disorders were seen in guinea-pig and rabbit lymphocytes.

We studied the mutagenetic effect of 60 Hz electric fields on the cells of a free-floating peritoneal-cavity tumor implanted in mature female mice (43, 44). The tumor was propagated in control mice by injecting the host intraperitoneally with tumor-containing fluid that had been freshly removed from an unexposed animal. After 14 days, a few drops of tumor were removed and the tumor cells were processed for chromosomal analysis. Tumor propagation in the 2-week exposed groups was identical except that the mice were exposed to DC electric fields of 8-16 kv/m. The tumor cells were ordinarily lethal to the host about 3 weeks after injection. To propagate the tumor for longer periods it was therefore necessary to transplant it to a new host every 7-14 days. Consequently, tumor cells exposed for 4-15 weeks required serial inoculations into 2-9 continu ously exposed mice. On the day the chromosomal analysis was to be performed, the host was injected with colcemid to arrest cell division in metaphase and allow direct visualization of the chromosomes. Cells exposed to horizontal EMFs for 2 weeks exhibited almost a threefold increase in the percentage of abnormal chromosomes when compared to control cells (Table 8.9); cells exposed to vertical EMFs for the same period, however, had a percentage of abnormal chromosomes comparable to that of the control cells. Extended exposure to both EMFs appeared to produce opposite results. The percentage of cells with abnormal chromosomes tended to decrease systematically in the horizontal EMF but increase systematically in the vertical EMF. The number of mice analyzed

Table 8.9. EFFECT OF DC ELECTRIC FIELDS IN THE RANGE 8-16 KV/M ON THE INCIDENCE OF CHROMOSOMAL ABERRATIONS IN EHRLICH ASCITES TUMOR CELLS EXPOSED IN VIVO

FIELD	WEEKS OF EXPOSURE	NO. OF MICE	NO. OF CELLS COUNTED	% CELLS WITH ABNORMAL CHROMOSOMES	AV. NO. OF ABNORMAL CHROMOSOMES PER ABNORMAL CELL
Horizontal	2	8	400	22.5 ± 6.6*	2.1 ± 0.6*
Horizontal	4-15	11	490	13.0 ± 9.1	1.3 ± 0.6
Vertical	2	8	370	5.8 ± 5.8	1.5 ± 1.5
Vertical	6-15	12	600	9.2 ± 7.6	1.0 ± 0.4
Control		10	500	8.8 ± 7.1	1.1 ± 0.5

NOTE: The abnormalities included chromatid exchanges, isochromatid breaks, dicentrics, rings, and long acrocentrics.

$p < 0.005$

prohibited a precise determination of the dependence on exposure time, and in both cases, when the results were averaged over the entire extended exposure period (4-15 weeks for the horizontal EMFs, and 6-15 weeks for the vertical EMFs), no statistically significant results were seen (Table 8.9).

EMFs have also been reported to produce chromosomal aberrations in nonsomatic cells (45). Adult male mice were exposed 1 hour/day for 2 weeks to 9.4 GHz, 100-10,000 $\mu W/cm^2$. After exposure, the animals were sacrificed and the sperm-cell chromosomes were analyzed. At each intensity, there was an increase in both translocations and univalent chromosome pairs.

Mutagenetic effects of EMFs have been reported in *in vitro* studies, and in studies involving insects and plants (46-51). Rat kangaroo cells ex posed *in vitro* to 2.4 GHz for 10-30 minutes exhibited chromosomal aberrations similar to those induced by X-rays (46). The results also showed that the EMF disrupted RNA synthesis and reduced protein production and cell proliferation. EMFs in the 15-40 MHz range and at K band (23 GHz) caused chromosomal abnormalities in Chinese hamster lung cells in culture (47). When cells of the same type were exposed as a monolayer for 15 minutes to a DC magnetic field of 15,000 gauss, it was found that of the 400 metaphase cells examined in the 24-hour period after exposure, approximately 3% exhibited a chromosomal aberration; this rate was 6 times higher than that seen in the controls (48). Exposure of monkey epithelial cells to 7000 μW/cm2, 2.9 GHz also caused chromosomal abnormalities (49).

Radiowave pulses (20-30 MHz) applied to male *Drosophila* for 5-6 minutes resulted in the production of numerous mutations in the off spring, including singed bristle, white eye, spotted eye, yellow body, and blister wing (50). The genetic effect exerted on the male germ cells was similar to that seen from the application of ionizing radiation (50). An increased incidence of inheritable abnormalities following exposure to EMFs has also been reported in plants (51).

Uncontrolled Variables

When the long bones are immobilized, (e.g., by casting) a frequent physiological response is a loss of bone material—a condition known as osteoporosis. As we have seen in chapter 2, bone is a piezoelectric material and, consequently, it exhibits the converse piezoelectric effect (mechanical deformation under the influence of an applied electric field). McElhaney et al. (52) hypothesized that an electric field could simulate the naturally present mechanical stresses in bone via the converse piezoelectric effect,

and thereby eliminate the osteoporosis associated with disuse. The theory was tested by immobilizing the hind limbs of rats and then observing the effect of an electric field; it was found that the osteoporosis caused by immobilization was reduced by exposure to 7 kv/m, 3-30 Hz. However, in addition, 44% of the EMF-exposed animals developed bone tumors; none were seen in the sham-irradiated rats. Martin and Gutman (53), (Martin worked with McElhaney et al. on the original study) performed a replicate study and confirmed the observation that the EMF ameliorated the immobilization-induced osteoporosis. No tumors, however, or other malformations were observed either by gross or microscopic examination.

The two studies were done under essentially identical conditions, but tumors were seen only in the first study. Statistically, it is unlikely that they were unrelated to the field and developed only in the exposed group by chance. This suggests that an uncontrollable variable (UV) capable of inducing tumors in conjunction with an EMF was present in the McElhaney study.

We too observed an EMF-related biological effect that was not seen in a replicate study; in our case, however, it was possible to preselect the animals in the second study and thereby gain information about the UV associated with the biological effect. In the initial study, we found secondary glaucoma in 10 of 60 rats that had been exposed for 30 days to 0.6-19.7 kv/m vertical electric fields; the glaucoma was not seen in 43 rats exposed to horizontal fields (0.3-9.7 kv/m) or in 72 controls (43). None of the rats had been subjected to an ophthalmic examination prior to field exposure because the appearance of eye diseases had not been anticipated. It was, therefore, not possible to determine whether the glaucoma resulted from a worsening of an already existing defect, or was caused solely by the EMF. These alternatives were examined in two vertical-field studies (2.8 kv/m, 19.7 kv/m) in which all animals were subjected to a pretest eye examination with the bimicroscope and the indirect ophthalmoscope. Rats that exhibited any identifiable disorder (iris hemorrhage, anterior synechia, dacyroadenitis, keratitis) were destroyed, and only defect-free animals were placed on study. Following 30-day exposures, no cases of secondary glaucoma were seen in either the exposed or sham-exposed rats 50 in each group). It seems to us, therefore, that our initial observations of secondary glaucoma most likely stemmed from an exacerbation of preexisting eye defects by the EMF. The EMF, in any event, could not have been the sole cause of the glaucoma.

The clearest example of the operation of a UV may be the multigeneration study done at the Battelle Laboratories (54). Following the publication of our first multigeneration study (34), Battelle was commissioned to replicate the work. The investigators first developed an exposure system

Table 8.10. AVERAGE BODY WEIGHTS IN THE BATTELLE MULTIGENERATION STUDY

REPLICATE	GENERATION		DAY 1	DAY 14	DAY 28	DAY 35	DAY 70
	Males						
A	F_1	E	1.8 ± 0.2* (30)	7.0 ± 0.8* (30)	16.4 ± 2.4* (27)	23.8 ± 2.4 (27)	34.6 ± 2.1* ·27)
		C	2.0 ± 0.2 (28)	7.5 ± 0.7 (28)	19.7 ± 1.9 (27)	24.8 ± 2.4 (27)	36.9 ± 2.1 (26)
	F_2	E	1.8 ± 0.2* (23)	7.5 ± 0.6 (22)	20.4 ± 2.2* (22)	26.2 ± 1.6 (11)	35.9 ± 1.6 (22)
		C	2.0 ± 0.2 (28)	7.3 ± 0.8 (28)	18.6 ± 3.0 (28)	25.8 ± 2.4 (24)	36.0 ± 2.1 (28)
	F_3	E	1.9 ± 0.2 (33)	7.2 ± 0.7 (33)	19.0 ± 2.2 (33)	25.0 ± 2.1* (31)	34.2 ± 1.8* (32)
		C	1.9 ± 0.2 (34)	7.4 ± 0.8 (32)	18.4 ± 2.6 (34)	26.2 ± 2.5 (32)	36.9 ± 2.7 (32)
B	F_1	E	1.9 ± 0.2 (17)	7.4 ± 0.9 (17)	20.3 ± 1.8 (17)	27.3 ± 1.4 (17)	37.0 ± 2.1 (17)
		C	1.9 ± 0.2 (28)	7.6 ± 0.7 (28)	20.4 ± 2.4 (28)	27.5 ± 1.7 (28)	36.7 ± 2.1 (28)
	F_2	E	2.0 ± 0.2 (28)	7.2 ± 1.2 (28)	19.3 ± 3.7 (28)	26.2 ± 2.8 (28)	37.0 ± 2.3* (28)
		C	2.1 ± 0.1 (23)	7.1 ± 0.6 (18)	19.5 ± 1.9 (21)	26.4 ± 2.0 (21)	35.4 ± 2.4 (20)
	F_3	E	2.0 ± 0.1 (35)	8.0 ± 0.7* (35)	19.8 ± 3.2 (34)	26.8 ± 2.6 (34)	38.9 ± 2.3* (33)
		C	2.0 ± 0.2 (30)	7.5 ± 0.6 (30)	19.6 ± 1.9 (30)	26.5 ± 1.6 (30)	36.4 ± 2.3 (30)
	Females						
A	F_1	E	1.8 ± 0.2 (34)	7.1 ± 0.6 (34)	16.4 ± 1.9 (34)	20.3 ± 1.6* (34)	28.9 ± 1.4* (34)
		C	1.9 ± 0.2 (28)	7.4 ± 0.6 (28)	17.1 ± 1.8 (27)	21.3 ± 1.5 (27)	29.9 ± 1.6 (26)
	F_2	E	1.8 ± 0.2* (22)	7.6 ± 0.4 (22)	19.0 ± 1.7* (22)	23.6 ± 1.6 (11)	29.2 ± 1.9* (22)
		C	1.9 ± 0.1 (25)	7.2 ± 0.9 (28)	17.2 ± 2.8 (27)	22.9 ± 2.0 (23)	30.7 ± 1.9 (27)
	F_3	E	1.8 ± 0.1 (24)	7.0 ± 0.8 (24)	16.7 ± 2.4 (24)	22.1 ± 1.8* (24)	28.6 ± 1.1* (23)
		C	1.8 ± 0.1 (30)	7.2 ± 0.8 (30)	17.5 ± 1.9 (29)	23.0 ± 1.4 (29)	29.9 ± 1.9 (28)
B	F_1	E	1.8 ± 0.2 (23)	7.3 ± 1.0 (23)	16.5 ± 2.1* (23)	22.8 ± 1.5* (23)	29.5 ± 2.5 (23)
		C	1.9 ± 0.2 (27)	7.6 ± 0.6 (27)	18.8 ± 1.8 (25)	23.7 ± 1.0 (25)	29.0 ± 1.6 (24)
	F_2	E	1.9 ± 0.2* (36)	7.1 ± 1.1 (36)	18.1 ± 2.3 (36)	23.2 ± 1.9 (36)	29.4 ± 1.3 (36)
		C	2.0 ± 0.1 (30)	7.5 ± 0.9 (19)	17.9 ± 1.7 (29)	23.2 ± 1.9 (29)	29.3 ± 1.9 (28)
	F_3	E	2.0 ± 0.1* (29)	7.8 ± 0.6* (29)	18.0 ± 2.8 (27)	22.3 ± 1.9* (27)	29.9 ± 1.8* (27)
		C	1.8 ± 0.2 (34)	7.3 ± 0.5 (34)	18.0 ± 1.5 (34)	23.3 ± 1.3 (34)	28.5 ± 1.8 (34)

The number of mice weighed is indicated in parentheses. E, experimental; C, control.
*$p < 0.05$

that was unexcelled with regard to field homogeneity and reproducibility of electrical environment. Every aspect of the animals' physical environment—light, temperature, humidity, presence of pathogens in the air, air flow, for example—was rigorously monitored and controlled by automatic equipment. The investigators then constructed two complete exposure facilities: each consisted of a completely characterized exposure unit, an identical unit for sham-irradiation, and a completely controlled environment suitable for housing both units.

The multigeneration study was begun in the first exposure facility, and 3 weeks later a replicate study was begun in the second facility; both replicates were done double blind. The body-weight data for the males and females of each of the three generations in each replicate is shown in table 8.10. Despite the fact that the maximum level of human intervention and control was exercised, and despite the unprecedented resources devoted to the study, it was obviously not possible to eliminate the role of a UV: at the end the study, the males and females in the first replicate were statistically significantly smaller than the controls, but in the second replicate they were significantly larger.

When an experiment is replicated and different results are observed, there is no general rule by which it can be decided whether the first or the second replicate (or both or neither) are the true descriptions of nature. In each case an analysis must be made of the details of the studies and their relation to other studies. Only in this manner can it be decided whether an UV likely was present (in which case both experiments would correctly describe nature, but under different circumstances), or whether a Type I or Type II error was made in one of the replicates.

Summary

The reports described in this chapter involve the effects of EMF on metabolism, growth, and reproduction. When they are considered in conjunction with the previous three chapters, it becomes clear that there is no biological function which can be said to be impervious to nonthermal EMFs—they are a fundamental and pervasive factor in the biology of every living organism. The nature, extent, and physiological significance of the effects to be expected in different organisms, and their dependence on the spectral characteristics of the field remain, for the most part, to be determined by future studies. We have no doubt that some of the reports described here are erroneous in the sense that some investigators have reported effects that ultimately will be found to be artifacts or statistical anomalies. But this is true with regard to every area of biological

experimentation-the mathematical precision of the physical sciences is simply unattainable. It means only that the details regarding the biological effects of specific EMFS have not been established with certainty, and it does not detract form the fundamental point that the nonthermal EMF is a physiologically active agent. The scope of the observed effects, and some of the factors which influence them, are shown in Figure 8.4.

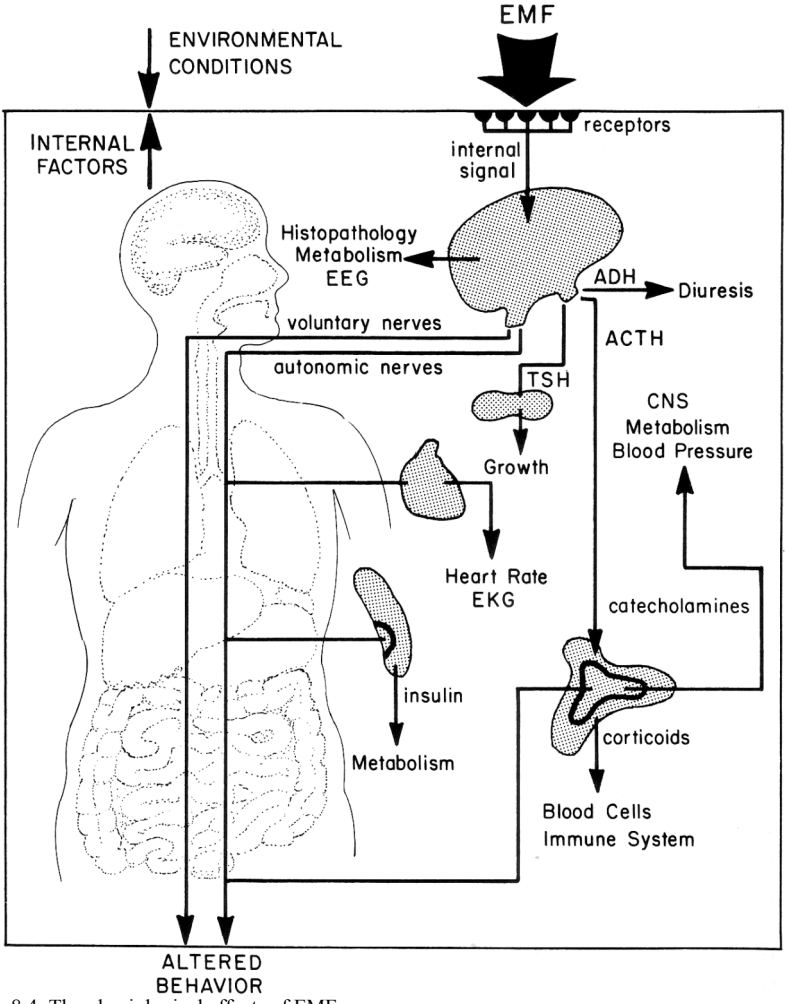

Fig. 8.4. The physiological effects of EMFs.

Although the point has frequently been disputed in the stormy controversy that has developed regarding some of the practical implications of our conclusion (56-60), it is nonetheless true that a biological phenome non need not be understood at the molecular level as a prior condition to the acceptance of its existence by science. On the other hand, every biological phenomenon obviously has some molecular basis, and the reports of the biological effects of EMFs will not be fully satisfactory until their molecular basis is either established or shown to be unknowable. Some progress has been made in understanding the origin of EMF-induced biological effects, and this work is described in the next chapter.

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