Thyroid morphology and activity does not respond to ELF electromagnetic field exposures

G.F. Lafrenière and M.A. Persinger[1]

Behavioral Neurobiology Laboratory, Laurentian University, Sudbury (Ontario, Canada P3E 2C6), 3 July 1978

Summary. The hypothesis that the thyroid is the sensitive organ for extremely low frequency electromagnetic field effects was tested. Rats that had been exposed either perinatally or as adults to several intensities of 0.5 Hz magnetic fields displayed no significant alterations in thyroid morphology or circulating hormone measures.

Extremely low frequency or ELF (0.1 Hz to 1 kHz) electromagnetic fields (EMFs) from both natural and man-made sources have been discussed recently[2-4] as biologically effective stimuli. Alterations in thyroid activity during or following exposure to ELF-EMFs have been inferred from both experimental and correlational studies[5,6]. Since rats exposed perinatally or as adults to 0.5-Hz EMFs have been reported to display behavioral-biological changes commensurate with thyroid involvement[7-9], we decided to test the thyroid theory for ELF-EMF effects directly by measuring blood T_3 (triiodothyronine) and T_4 (thyroxine) as well as thyroid follicle and perifollicular mast cell numbers in rats that had been exposed to such fields. The rationale for the morphological measures has been discussed.[10]

The experiment was designed to answer three specific questions. 1. Does perinatal exposure to 0.5-Hz EMFs at intensities known to influence adult behavior alter the thyroid measures? 2. Does adult exposure to 0.5-Hz EMFs at intensities approaching geomagnetic storms or unstable weather values influence these measures? 3. Does perinatal exposure to relatively intense 0.5-Hz fields enhance thyroid response to weak, near-natural intensity fields when the organism is exposed to these fields as an adult?

In 8 experiments, 40 (5 rats/experiment), 180–200-day-old male albino Wistar rats from 18 different litters were exposed for 5 days to one of 3, 0.5-Hz EMF intensities (produced by rotating horseshoe magnets[2]), to a sham field condition (0.5-Hz variation, if any, below 10^{-9} T) or to normal colony room conditions: temperature 22 ± 1 °C; L:D of 12:12; humidity $45 \pm 5\%$. The 3 field intensities (peak to peak) were: 10^{-6} T, 10^{-7} T or 10^{-8} T. Half the number of rats in each of the 5 conditions (10^{-6} T, 10^{-7} T, 10^{-8} T, sham field and colony controls) had been exposed perinatally (2.5 days before birth to 2.5 days after birth) to 10^{-3} T to 10^{-4} T, 0.5-Hz fields, while the other subjects had been exposed to the sham field condition. Other details and specific procedures of this experiment have been published[11].

Following the 5-day adult exposures, the rats were decapitated. Blood serum was collected and analyzed for T_3 and T_4 routinely. Thyroids were removed carefully and fixed in EFA (90 pts 80% ethanol; 5 pts 30% formaldehyde and 5 pts glacial acetic acid), processed, paraffin embedded and sectioned at 6 µm. Follicle numbers and perifollicular mast cell numbers were determined according to Persinger et al.[10] 2-way analyses of variance of the 4 measures according to the 5 adult conditions and the 2 perinatal conditions, as well as calculations of means and SD, were completed by computer.

The means and SD for T_3 and T_4 values and for thyroid follicle and mast cell numbers according to adult and perinatal conditions are shown in the table. Analysis of variance demonstrated a significant ($p < 0.05$) difference between adult conditions ($F = 3.78$, $df = 4, 30$); however, an a posteriori-test (Scheffe's) indicated the difference was primarily between the colony room controls and the other groups but not between intensity levels. All other differences, including interactions between adult and perinatal conditions, were not statistically significant ($F < 1$; $p > 0.05$).

The speculation of previous studies[8,9], suggesting that alterations in thyroid morphology/activity are primary correlates of ELF-EM exposures in rats, was not substantiated in this study, although these rats[11] demonstrated the previously reported[9] changes in testicle weights. These data, in conjunction with previous experiments involving female rats exposed as adults for 10 days to 10^{-3} T to 10^{-7} T, 0.5-Hz EMFs[10], appear to exclude the thyroid as 'the sensitive organ' for ELF-EMF exposures. In addition, the absence of statistically significant interactions between perinatal and adult conditions appears to eliminate the likelihood that early (perinatal) ELF-EMF exposure alters thyroidal response when the adult organism is exposed again to the ELF-EMF condition.

Means and SD (±) for serum triiodothyronine (T_3), serum thyroxine (T_4), perifollicular mast cell numbers and thyroid follicle numbers for male rats according to the 5 adult exposure conditions and to the 2 perinatal conditions

	T3 (percent binding site)	T4 (µg/dl)	Mast cells/mm²	Follicles/mm²
Adult condition				
0.5 Hz 10^{-6}T (n=8)	51.6±4.4	5.1±0.7	40±10	115± 4
0.5 Hz 10^{-7}T (n=8)	52.4±4.8	4.8±0.6	34± 6	117±10
0.5 Hz 10^{-8}T (n=8)	53.3±3.4	4.6±0.9	36±12	111±15
Sham field (n=8)	53.8±3.3	4.9±1.0	36± 7	117±12
Colony control (n=8)	51.3±4.7	6.1±0.6	34± 5	119±11
Perinatal condition				
0.5-Hz EMF (n=20)	52.6±4.6	5.1±0.9	35± 8	117±10
Sham field (n=20)	52.4±3.6	5.1±0.9	37± 9	115±11

1 Thanks to the technical staff of the Clinical Chemistry Laboratory at Laurentian Hospital.
2 M.A. Persinger (ed.), ELF and VLF Electromagnetic Field Effects. Plenum Press, New York 1974.
3 J.G. Llaurado, A. Sances and J.H. Battocletti (ed.), Biologic and Clinical Effects of Low-Frequency Magnetic and Electric Fields. C.C. Thomas, Springfield 1974.
4 J.H. Battocletti, Electromagnetism Man and the Environment. Westview Press, Boulder 1976.
5 M.A. Persinger, H.W. Ludwig and K.-P. Ossenkopp, Percept. Mot. Skills *36*, 1131 (1973).
6 M.A. Persinger (ed.), in: ELF and VLF Electromagnetic Field Effects, p. 275. Plenum Press, New York 1974.
7 M.A. Persinger, Devl Psychobiol. *2*, 168 (1969).
8 K.-P. Ossenkopp, Psychol. Rep. *30*, 371 (1972).
9 K.-P. Ossenkopp, W.T. Koltek and M.A. Persinger. Devl Psychobiol. *5*, 275 (1972).
10 M.A. Persinger, G.F. Lafrenière and N.J. Carrey, Int. J. Biomet. *22*, 67 (1978).
11 M.A. Persinger, N.J. Carrey, G.F. Lafrenière and A. Mazzuchin, Int. J. Biomet. *22*, in press (1978).

Effects of postnatal progesterone treatment on ovarian function in adult rats

A. Bukovský, J. Presl and Z. Krabec

Institute for the Care of Mother and Child, nábř. K. Marxe 157, CS-147 10 Praha 4-Podoli (Czechoslovakia), 31 October 1977

Summary. Long-lasting postnatal progesterone administration in female rats induced an early or delayed ovulatory failure with persistent vaginal estrus. Short-term treatment was ineffective. The beginning and incidence of ovulatory failure appeared to depend on the beginning and duration of progesterone treatment. The necessary duration of progesterone administration exceeds the critical postnatal steroid sensitive period of sexual differentiation of the hypothalamus. Moreover, long-lasting progesterone treatment results in ovulatory failure even if started after termination of this period.

In laboratory rodents, the presence or absence of testicular testosterone during the critical steroid sensitive period of sexual differentiation of the hypothalamus (1 week of life in the rat) organizes the male or female pattern of hypothalamic control of gonadotropin secretion[1]. Unlike exogenous testosterone or estradiol both of which masculinize the hypothalamus in newborn female rats, properly timed progesterone treatment protects the developing female brain from the «damaging» effects of early postnatal testosterone or estradiol administration[2–4]. A similar protective role is suggested for placental progesterone in primate fetuses of both female and male sex, thus explaining the potentially operative positive feedback effect of estrogens in male primates[5].

In fact, high doses of progesterone injected during the early postnatal critical period of hypothalamic sexual differentiation do not impair the cyclic ovulatory release of gonadotropins in adult female rats[6–8]. It has been reported, however, that postnatal long-lasting small-dose progesterone treatment impairs the late ovulatory function in rats[9–12].

The present experiment was designed to assess the effective period of postnatal progesterone treatment required to induce an impairment of the ovulatory function in adult female rats.

Material and methods. Immature female Wistar rats of our laboratory colony were injected s.c. with different progesterone doses during different periods of time. The amount of oil solvent was 0.05 ml in ages up to 19 days and 0.1 ml from the age of 20 days. The control animals were injected with oil only. Vaginal smears were taken daily for 4 weeks after finishing the 2nd and the 5th month of life. Only then were the animals decapitated and the ovaries evaluated histologically, using standard technique. All experimental groups are summarized in the table.

Results and discussion. An early ovulatory failure, which is characterized by persistent vaginal estrus observed already after the 2nd month of life, was induced with daily progesterone administration up to the 40th day of life (experiment 1). Shortening this period to 26 days with the dosage of 200 µg progesterone daily was similarly effective (experiment 2). Lowering the daily dose of progesterone to 100 µg on days 1–19 and using the daily dosage of 200 µg on days 20–26 did not change the incidence of the early ovulatory failure (experiment 3), whereas further lowering of the daily progesterone dose to 100 µg on days 1–26

Experiment No.	Number of rats	Day of treatment Dose of progesterone daily			Total dose of progesterone (µg)	Persistently estrous type of smear*	
		1200 µg	100 µg	200 µg		3rd month	6th month
1	6	–	1–19	20–40	6100	6/6	6/6
2	12	–		1–26	5200	12/12	12/12
3	9	–	1–19	20–26	3300	9/9	9/9
4	23	–	1–26		2600	10/23	20/23
5	9	–	10–19	20–26	2400	0/9	9/9
6**	8	–	1–19	–	1900	0/8	8/8
7	5	–	1–10	–	1000	0/5	2/5
8	16	–	10–19	–	1000	0/16	3/16
9	18	5	–	20–26	3600	0/18	0/18
10	15	–	–	20–26	1400	0/15	0/15
11	11	5	–	–	1200	0/11	0/11
12***	14	–	–	–	–	0/14	0/14

* Number of animals with persistent estrus/total number. ** Experiment published previously[12]. *** Control group treated with oil solvent from days 1 to 26.