DUAL ACTIONS OF MICROWAVES ON SERUM CORTICOSTERONE IN RATS*

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ABSTRACT

Fifty-eight male, young adult Long-Evans rats acclimated to experimental procedures were exposed to 2.45 GHz CW microwaves for 4 hours at 0, 0.1, 1, 10, 25 and 40 mW/cm^2. They were sacrificed immediately after exposure at 1530 hours. The time of sacrifice is the mid-course of circadian increase of serum corticosterone (CS). While the CS levels in the shams (25.0 ± 2.1 $\mu g/dl$, S.E., n = 18) was not different from shams obtained previously (23.7 ± 4.3 $\mu g/dl$; S.E., n = 6), a significant lower CS was observed in groups exposed to 0.1 mW/cm^2 (16.4 ± 2.8 $\mu g/dl$; S.E., N = 8) and 1 mW/cm^2 (16.4 ± 3.1 $\mu g/dl$; S.E., n = 8). Significant adrenal stimulation was noted in rats exposed to 40 mW/cm^2 (41.8 ± 6.8 $\mu g/dl$; S.E., n = 8). Power densities of 10 and 25 mW/cm^2 did not induce any changes in the CS levels. A dual action of microwave on the CS is thus demonstrated. However, the lower CS levels in the 0.1 or 1 mW/cm^2 is still higher than the sham at 1230 hours. Thus, this can only be viewed as an inhibition of the expected circadian elevation of the CS levels. In the present study, colonic temperature was higher than shams in rats exposed to 10 mW/cm^2 or higher.

Evidence was obtained in 4 hour microwave exposed rats that there was a decreased CS dependence on the body temperature from the normal relation between CS and colonic temperature of sham-exposed rats between 1230 to 1930 hours. Therefore, the same degree of temperature increase in the microwave exposed rats may be less stressful than would be expected from the daily oscillation of biologic activity, if CS increase is considered to be indicative to a reaction to a stressor.

SUMMARY

"Lower intensities" (< 10 mW/cm^2) of microwave exposure have been reported to have a subtle biological effect on functions of the neuroendocrine system. The suggestion of direct action by low-level microwave exposure on the central nervous system and endocrine systems in addition to the well-established heating effects of microwaves have raised uncertainties in characterization of the total biological effects of microwaves.

The neuroendocrine system, a complex of hormone secreting glands and the central nervous system, functions as a chemical regulatory system in mammals to control and regulate metabolism and growth and protect the body from endogenous and exogenous alterations in homeostasis. In spite of the obvious importances of this subject, only sparse and not always sufficiently documented data are available. For the past several years, our research activities have been concentrated on neuroendocrine reactions of animals to microwave exposure.

Dumanskij and Sandala (1974) reported an increase in adrenal weight, a reduction in adrenal ascorbic acid content and an increased urinary 17-ketosteroids secretion in rats that were exposed chronically to "UHF" and "SHF" in intensities lower than 0.01 mW/cm^2. From these results, the exposure to electromagnetic fields was concluded to be unfavorable stimuli. On the other hand, Novitskii et al. (1978) found that after a 30 minute exposure of rats to 0.01 mW/cm^2, SHF, EM fields there was an increase in corticotropin releasing factor (CRF) and adrenocorticotropic hormone (ACTH) activity. Mikolajczyk (1974) has indicated that moderate or lower power density microwaves being insufficient to produce a thermal effect, probably cannot elicit a "stress" reaction. Similarly, Parker (1973) reported a negative adrenal activation in rats exposed to 2.45 GHz (CW) microwave for 16 hours at 10 mW/cm^2. Lotz and Michaelson (1978) observed that plasma corticosterone (CS) levels exhibited a variable power density/threshold pattern of response, with a different threshold for 120 minutes exposure (20 mW/cm^2; mean colonic temperature = 37.9°C) than for 30 or 60 minutes exposure (50 mW/cm^2; mean colonic temperature = 38.9°C) of rats to 2.45 GHz (CW). The acute adrenal stimulation is apparently under hypothalamus-hypophysis control (Lotz and Michaelson, 1977). Lu et al. (1977) reported inhibition of circadian elevation of serum CS levels in rats exposed to 20 mW/cm^2 for 8 hours to 2.45 GHz (CW) microwaves.

In the present experiment, procedure acclimated rats (Lu et al., 1977) were exposed to 2.45 GHz (CW) for 4 hours to power densities of 0, 0.1, 1, 10, 25 and 40 mW/cm^2. Environmental factors were maintained at 24 $\pm$ 1° C and 60 $\pm$ 3% relative humidity. Body weight of these animals was 299.0 $\pm$ 2.7

grams (S.E., n = 58). Details of the procedure and facility have been previously reported (Lu et al., 1977). Animals were acclimated from 0830 to 1130, exposed from 1130 to 1530 hours and sacrificed immediately after exposure.

Serum CS levels of the sham-exposed rats in the present experiment were comparable to those obtained previously [25.0 $\pm$ 2.1 μg/dl (18) vs. 23.7 $\pm$ 4.3 μg/dl (6); S.E.] in this laboratory (Lu et al., 1977). Rats exposed to 0.1 [16.4 $\pm$ 2.8 (8)] and 1 mW/cm^2 [16.4 $\pm$ 3.1 (8)] had significantly lower CS levels than the sham-exposed rats. No changes in CS levels were noted in rats exposed to 10 [22.6 $\pm$ 3.3 (8)] or 25 mW/cm^2 [19.2 $\pm$ 4.7 (8)]. Significant adrenal stimulation was noted in rats exposed to 40 mW/cm^2 [CS = 41.8 $\pm$ 6.8 μg/dl (8)].

Colonic temperature was significantly higher in rats exposed to 10, 25 and 40 mW/cm^2 than the sham-exposed rats. No change in the colonic temperature was noted in rats exposed to 0.1 or 1 mW/cm^2.

As indicated, the sacrifice time of rats in this experiment was 1530 hours. At this time, the CS levels were significantly higher than those at 1230 hours, 2.1 $\pm$ 0.7 μg/dl (S.E., n = 15), which is consistent with early afternoon circadian increase of CS. The CS levels in rats exposed to 0.1 and 1 mW/cm^2 for 4 hours were higher than CS levels of shams at 1230 hours. The decrease in the CS levels is thus an inhibition of circadian elevation of CS instead of a real inhibition of adrenocortical function.

To further characterize the changes, we combined all the data obtained in our laboratory. In the 56 sham exposed rats sacrificed between 1230 and 1930 hours, a significant correlation between CS and colonic temperature was noted (Pearson's product-moment correlation coefficient, $r = 0.38$, $p < 0.01$). The correlations were also significant for rats exposed for one hour and sacrificed at 1230 hours to 1 to 70 mW/cm^2 ($r = 0.84$; $n = 32$; $p < 0.001$) and for 4 hours and sacrificed at 1530 hours to 0.1 to 40 mW/cm^2 ($r = 0.52$; $n = 56$; $p < 0.001$). The temperature coefficients of CS levels were 20.2 μg/dl/$^\circ$C for the sham exposed group, 15.3 μg/dl/$^\circ$C for 1-hour exposed and 8.8 μg/dl/$^\circ$C for 4-hour exposed animals. A significant difference was noted in the temperature coefficients between sham and 4-hour exposed groups.

The mechanism of this inhibition is not clear at present. It can be a circadian shift as reported in the changes of colonic temperature (Lu et al., 1977) or an initial process leading to microwave acclimation. Examination of these observed effects on CS can be summarized as an inhibition of circadian elevation and a stimulatory action by microwave exposure. Two different action spectra for these two effects may be assumed. Therefore, three different end results can be noted.

If CS level is considered to be indicative of stressful stimuli, the lower level microwaves can, in fact, modify the extent of reactions of the animal to the daily rhythmic physiologic stimuli.

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