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Bioelectrochemistry and Bioenergetics 43 (1997) 27–33

BIOELECTROCHEMISTRY AND BIOENERGETICS

Role of field intensity in the biological effectiveness of millimeter waves at a resonance frequency

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Received 21 August 1996; revised 22 January 1997

Abstract

The study replicated the effect of low-intensity millimeter waves (MMW) on isolated nerve function and characterized its dependence on radiation intensity. MMW exposures lasted for 23 min at 0.02, 0.1, 0.5, or 2.6 mW cm^{-2} (41.34 GHz) and were accompanied by a high-rate electrical stimulation of the nerve (HRS, 20 twin pulses s^{-1}, 9 ms interpulse interval). MMW had no effect on the conditioning compound action potentials (CAPs), but significantly attenuated the HRS-caused decrease of the test CAPs. The magnitude of this effect was virtually the same (20–25%) at field intensities of 0.02, 0.1, and 2.6 mW cm^{-2}. Irradiation at 0.5 mW cm^{-2}, however, did not produce statistically significant changes. The results are consistent with our earlier observations of this MMW effect and provide further evidence for its nonthermal mechanism. © 1997 Elsevier Science S.A.

Keywords: Millimeter waves; Ratio of relative amplitudes; Nerve function

1. Introduction

Frequency-specific, nonthermal in nature biological effects of millimeter waves (MMW) have been demonstrated in many different studies [1–21] (see [22–24] for reviews). However, the dependence of these effects on field intensity was analyzed by only a few investigators, and their results were highly controversial. Bioeffects either linearly increased with increasing the field intensity [1], or reached a plateau [2–5], or did not substantially change [6–8]. The intensity thresholds reported in these studies differed by many orders of magnitude.

The lowest threshold of 10^{-19} W cm^{-2} was reported by Belyaev et al. [3,4]. These authors used an anomalous viscosity time dependence technique to reveal MMW effects on cell genome conformation, and discovered sharp frequency resonances. At a resonance frequency, the biological response gradually increased with the field intensity and reached a plateau between 10^{-17} and 10^{-8} W cm^{-2}, depending on cell concentration in the exposed suspension. The effectiveness of the radiation frequencies close to the resonance one was found to be dependent on the field intensity: the half-width of the frequency resonance increased from 3 MHz at 10^{-19} W cm^{-2} to 80–100 MHz at 10^{-4} W cm^{-2}. This effect was found to be dependent also on the polarization of the radiation [9] and on the haploid genome length [10].

Another MMW effect which has been consistently observed and studied for over 20 years is alterations of yeast cell growth rate [6,7,11–13]. Within a narrow frequency band of 41.8–42.0 GHz, some frequencies increased the growth rate by up to 15%, others decreased it by up to 29%, and still others were not effective. The latest studies by these authors demonstrated significant growth effects at field intensities down to 5×10^{-12} W cm^{-2} (8 kHz modulation). The magnitude of the bioeffect apparently did not change with increasing the field intensity from this lowest level to 1 mW cm^{-2}; however, the frequency resonance width increased from approximately 4 to 15 MHz. This dependence of the frequency resonance width upon the incident power density is in remarkable agreement with the findings mentioned above [4].

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PII S0302-4598(97)00022-6

Subtle, if any, dependence of the biological response on the field intensity was observed by Berzhanskaya et al. [8]. Reversible suppression of bioluminescence of a sea bacterium, *Photobacterium leiognathi*, was virtually the same at field intensities of 1.3×10^{-6} and 13×10^{-6} W cm^{-2} (36.2 GHz). This MMW effect could be repeated many times in the same cell culture, and intermittent 10-min periods with and without irradiation resulted in an 'oscillating' time pattern of the luminescence.

MMW-induced suppression of the locomotor activity of a Protozoan (*Paramecium caudatum*) had a threshold at about 0.02 mW cm^{-2} [5]. The effect reached maximum at 0.1 mW cm^{-2}, producing approximately a 20% decrease in the 'motility index'; higher radiation levels (up to 50 mW cm^{-2}) did not further increase the effect. Regardless of the field intensity, this effect had a resonance dependence on both the radiation frequency (peak at 42.25 GHz) and the frequency of modulation (peak at 0.0956 Hz).

Chernyakov et al. [1] studied MMW effects in various isolated preparations of excitable tissues (isolated heart, nerve, muscle, etc.). The authors have pointed out that both the frequency and intensity dependencies of MMW effects are highly individual and vary greatly from one preparation to another. The intensity thresholds to alter pacemaker rhythm in a strip of sinoatrial tissue differed between individual preparations by more than an order of magnitude. Regardless of the individual threshold, the reaction linearly increased with increasing the incident power density in the range from 0.02–0.03 to 0.5 mW cm^{-2}. For MMW effects on myocardium exposed in situ, the threshold for 'autumn' frogs (0.15–0.2 mW cm^{-2}) was an order of magnitude higher than for 'spring' specimens (0.02–0.025 mW cm^{-2}).

MMW irradiation at 5 mW cm^{-2} could block the transmembrane chloride current in giant algae cells (*Nitellopsis obtusa*), or, at different frequencies, irradiation enhanced this current 2–4 times [14]. These effects could be induced at a lower field intensity (0.4 mW cm^{-2}) as well, but were considerably delayed. In an isolated frog nerve preparation, exposure at 10 mW cm^{-2} for 40–90 min (77.7 GHz) markedly suppressed the compound action potential (CAP) conduction [15]. A single microwave treatment made the nerve far more sensitive to irradiation, i.e., the CAP decrease due to the second and subsequent exposures became increasingly steeper and took just 10–15 min. The rate of CAP suppression in the 'sensitized' nerves was proportional to the incident power density. Exposure of a 'fresh' (as yet unexposed) nerve at a lower power density of 5 mW cm^{-2} for up to 4 h caused neither CAP suppression nor nerve sensitization.

Our previous studies in isolated nerve, although performed in a different frequency range (38–53 GHz), did not confirm these CAP suppression or nerve sensitization effects [25]. On the other hand, MMW significantly increased the nerve ability to tolerate a high-rate electrical stimulation (HRS), namely, the irradiation attenuated the HRS-induced decrease of the test CAP [16]. We further compared the effectiveness of 15 different frequencies of the radiation, at either 0.25 or 2.0–2.6 mW cm^{-2} incident power levels [17,18]. The changes reached maximum (22 ± 7% above the sham control) at 41.34 GHz. This frequency was tested at 0.25 mW cm^{-2}; other frequencies (at either the same or a tenfold higher incident power density) produced a smaller effect or were not effective at all.

While these results testified to a nonthermal mechanism of the MMW effect, this conclusion needed further confirmation. The present study was intended to replicate this effect in an independent series of experiments, and to characterize its dependence upon the radiation intensity in more detail.

2. Materials and methods

2.1. Nerve preparation and data acquisition

Experiments were performed on an isolated nerve preparation (n. ischiadicus + n. peroneus) of the frog *Rana pipiens*. The use of animals was in compliance with the Animal Welfare Act and other federal statutes and regulations relating to animals and experiments involving animals. Active adult frogs (males) were kept in vivarium conditions (22–25°C, 30–70% relative humidity, 12 h light/12 h dark diurnal light cycle) for at least 1 week prior to experiments. The sciatic nerve was isolated in a conventional manner after mechanical destruction (pithing) of brain and spinal cord, and then was submerged into chilled Ringer's solution containing: NaCl 102.6; KCl 1.0; $NaHCO_3$ 0.7; $CaCl_2$ 0.9 (mmol l^{-1}); pH 7.4–7.6.

MMW exposures were performed in a special bath equipped with two pairs of artifact-free saline bridge electrodes and filled with mineral oil. A thin layer of the oil over the preparation (0.3–0.5 mm) effectively prevented the nerve from drying and was virtually transparent to MMW coming from a horn antenna above the bath. One pair of the saline electrodes was used for nerve stimulation, and the other one was for recording of the compound action potentials (CAP). To eliminate biphasic CAP signals, the nerve was crushed between the two electrodes connected to the amplifier. The electrodes were designed so as to provide exactly the same positioning of every nerve preparation in the bath; they also limited the MMW-reachable area to a 30 mm long middle portion of the nerve (between the two pairs of the electrodes).

CAPs were evoked by paired square pulses from a Grass Instruments Stimulator S8800. We used a supramaximal nerve stimulation, that was 2–5 V at 0.2 ms pulse width. Stimulating pulses were continually delivered at a rate of either 4 pair s^{-1} (low-rate stimulation) or 20 pair s^{-1} (high-rate stimulation, HRS). The interval between two pulses in a pair was 9 ms, so that the second CAP was evoked during the relative refractory period after the first

CAP. Automatic on-line averaging of 16 consecutive CAP records by a Tektronics 2430 digital oscilloscope improved the signal-to-noise ratio and deemphasized the incidental CAP variability. Peak amplitudes of the conditioning and test CAPs (i.e., the CAPs evoked by the first and the second stimuli in a pair, respectively) were measured with the help of digital cursors on the screen of this oscilloscope.

The exposure bath was cooled to 11–12°C by a constant flow of chilled water from a thermostabilized water bath. The temperature in the exposure bath was monitored by a Luxtron Instruments model 850 multichannel fluoroptic thermometer, with the probe situated in the mineral oil 2–3 mm from the middle of the nerve. As measured by this probe and confirmed by other methods [25], microwave heating of the nerve at 2.6 mW cm^{-2} (the maximum tested power density) did not exceed 0.3–0.4°C, while heating at the lower power densities was negligible.

2.2. Irradiation and dosimetry

The exposure bath was situated under a horn antenna connected to a microwave power source (model G4-141, Russia). The nerve was at a 52 mm distance from the edge of the horn and was aligned with the *E*-field. Exact radiation frequency and net input power to the horn were monitored via a bidirectional 10 dB waveguide coupler using an EIP model 548A frequency counter (USA) and M3-21 wattmeter (Russia). The incident power density in the air was measured with a broadband electric field probe (Narda Microwave Corp., model #8721). The probe readings in the 37–53 GHz range were verified by a comparison with the field values calculated by a free-space standard field method [26]. Local field intensity measurements and field mapping were completed using a calibrated miniature flat crystal detector (Narda part 4824) and a computer controlled robotic tri-axial scanner. As established by the mapping, maximum field nonuniformity over the exposed site of the nerve did not exceed 1 dB [18]. More details on the methods of field characterization and justification of the calibration procedures will be provided in a separate paper.

Irradiation was performed in a continuous-wave mode at a frequency of 41.34 GHz, which was previously found to be the most effective of 15 different frequencies tested [17,18]. The MMW generator was tuned to 41.340 ± 0.002 GHz at the beginning of each exposure; this frequency was monitored throughout the entire exposure period and adjusted in the case of drift. All devices, including the MMW generator, stayed turned on during sham exposure as well, but waveguide attenuators were tuned to maximum field attenuation (about 80 dB).

2.3. Experiment protocol and data analysis

The high-rate stimulation (HRS) test is a sensitive method to reveal functional changes in excitable tissues, even those which are not detectable by other methods. However, the ability to tolerate HRS varies a lot from one preparation to another, and this variability has to be taken into account. One way to deemphasize individual differences between nerve preparations is to compare the HRS tolerance in exposed and control sites of the same preparation [27,28]. Another way is to compare nerve performance in two consecutive HRS trials, which should be identical except for MMW or sham irradiation applied during the second trial only [17,18]. The second approach, although more laborious, was more appropriate for our exposure conditions.

Thus, each successful nerve preparation was subjected to two consecutive HRS trials, 22 min each (see Table 1 and Fig. 1 for illustration of the procedures). The peak amplitude of the conditioning and test CAPs was measured

Table 1
Timeline of experimental procedures employed to establish the effect of millimeter waves (MMW) on isolated nerve tolerance to high-rate stimulation (HRS)

Procedures	Procedure duration /min	Stimulation rate/paired pulse s^{-1}	Exposure	CAP recording timepoints /min
Stabilization	15–20	4	No	
1st HRS	22	20	No	0, 15, 20, 25 [a]
Recovery	53	4	No	
2nd HRS	22	20	MMW or sham [b]	0, 15, 20, 25 [a]

[a] The time count began 4 min before the HRS onset.
[b] MMW or sham irradiation began 2 min prior to the onset of the 2nd HRS and continued till the end of the experiment.

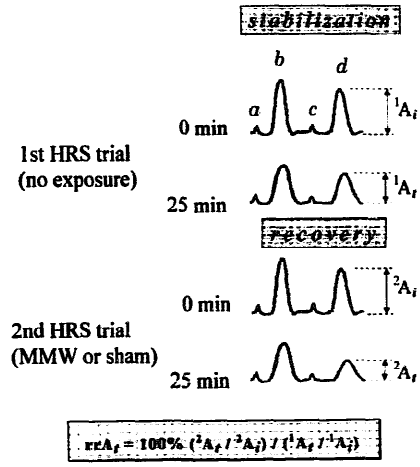

Fig. 1. Illustration of the experimental procedures. Each experiment consists of two sequential high-rate stimulation (HRS) trials, separated by a 53-min interval necessary for nerve recovery. The 2nd HRS trial is accompanied by either MMW or sham exposure. (a, c) Paired electric stimuli (9-ms interval); (b, d) conditioning and test compound action potentials (CAPs), respectively. Measurement of the peak amplitude of the test CAPs (shown to the right) and of the conditioning CAPs (not shown) was performed before the HRS (0 min), and during the HRS (15, 20, and 25 min). The equation at the bottom of the graph was used to calculate the ratio of the relative amplitudes (rrA). See text and Table 1 for more explanation.

before each trial (0 min) and three times during it (at 15, 20, and 25 min; the HRS lasted from 4 to 26 min). The first trial began after sufficient stabilization of the nerve activity, that was at least 15–20 min after its transfer into the exposure bath. The interval between the trials was set at 53 min. The first trial was always performed without any irradiation. MMW or sham exposure began 2 min before the onset of the second HRS trial and continued till the end of the experiment. The stimulation rate before and between the HRS trials was 4 pair s^{-1}.

This low-rate stimulation (4 pair s^{-1}) could be tolerated by the nerve for hours, without substantial changes in CAP conduction. In contrast, the HRS causes gradual and reversible decrease of the CAPs. The nerve ability to tolerate HRS was quantitatively assessed by using a relative amplitude (rA) index:

$$rA_t = A_t/A_i$$

where A_i is the initial CAP amplitude (before HRS), and A_t is the amplitude at a timepoint t during the HRS. The relative amplitude values were calculated separately for the conditioning and test CAPs, and for every timepoint of the two HRS trials. The ratio of the relative amplitudes (rrA) in the matching timepoints t of the second and the first HRS trials showed whether irradiation had affected the nerve ability to tolerate HRS:

$$rrA_t = 100\%\left({}^2rA_t/{}^1rA_t\right) = 100\%\left({}^2A_t/{}^2A_i\right)/\left({}^1A_t/{}^1A_i\right)$$

where the superscripts 1 and 2 correspond to the first HRS trial (without any exposure) and to the second trial (with either MMW or sham exposure). Obviously, if the nerve tolerance to HRS has not been changed during the second trial, the rrA will be equal to 100%. However, not only MMW irradiation could cause a deviation of the rrA value from 100%: this could also result from gradual loss of the preparation viability during its maintenance in vitro and from incomplete nerve recovery after the first HRS trial. To take into account the possible impact of the last two factors, the obtained rrA values were compared not just to the 100% level, but to the corresponding rrA values in experiments with sham exposure.

Each nerve preparation received a single MMW or sham exposure lasting for 23 min. MMW exposures were performed at four different field intensities, namely 0.02, 0.1, 0.5, and 2.6 mW cm^{-2}. Experiments with different field intensities and with sham exposure alternated in a random manner, but with several exceptions: (1) Nerves from two legs of the same frog were always exposed in different regimens, (2) To increase the statistical reliability of the results, sham exposures were performed more often than MMW exposures in each regimen, and (3) We actually had two independent groups exposed at 0.1 mW cm^{-2}; these groups received different treatment after being exposed in the same conditions. For the present study, we considered only those MMW effects which occurred during the exposure; this allowed us to pool these two groups together and regard them as a single group.

Statistical analysis of the data employed a χ^2 test and t-test with Dunnet's correction [29] when applicable. The MMW-induced increase of the test CAP rrA had already been established in our previous experiments [16–18], so one-sided statistical tests were now used for verification of this effect.

3. Results

A total of 33 experiments with MMW exposures at 4 different field intensities and 9 experiments with sham irradiation were carried out. The average rrA values for the conditioning and test CAPs in all the groups are presented in Fig. 2.

Consistent with our earlier results, the conditioning CAP was not affected by MMW irradiation. The rrA values in all the groups, including sham, were close to 100%. In other words, the conditioning CAP decrease during the second HRS trial (with irradiation) was not different from its decrease during the first HRS trial (without any irradiation). No effect was detected even with microwave heating of the preparation, which reached 0.3–0.4°C at the incident power density of 2.6 mW cm^{-2}.

The test CAP, which was evoked during the relative refractory period (9 ms after the conditioning CAP), was more sensitive to the HRS and manifested different changes

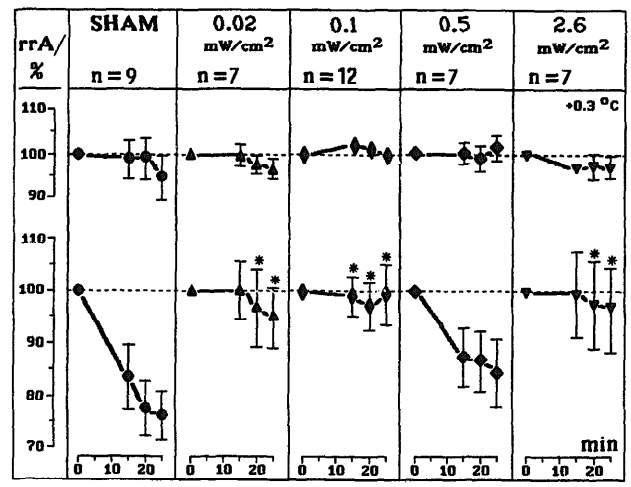

Fig. 2. Effect of millimeter waves (41.34 GHz) at different field intensities on isolated nerve ability to sustain a high-rate stimulation (HRS). Horizontal axes: time into the second HRS trial. In all groups, irradiation began at 2 min, and the HRS began at 4 min; both continued till the end of the experiment. Vertical axes: relative amplitude ratio values (rrA, mean ± SE) for the conditioning and test compound action potentials (top and bottom graphs, respectively). The field intensity (in mW cm^{-2}) and the number of independent experiments in each group (n) are indicated above the graphs. Asterisks show statistically significant differences from the sham exposure ($p < 0.05$, t-test with Dunnet's correction). See text for more details on the rrA calculation.

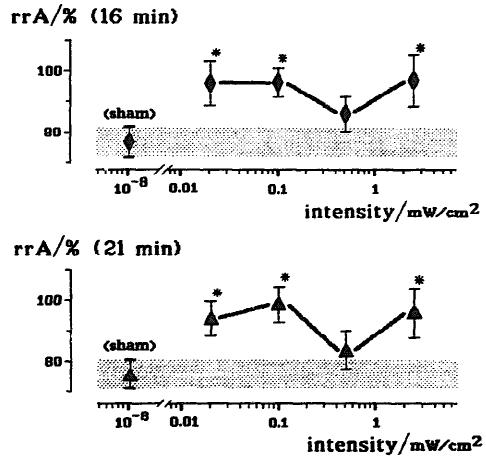

Fig. 3. Dependence of the biological effect of millimeter waves at 41.34 GHz on the field intensity. The top and lower graphs correspond to the 16 and 21 min timepoints into the second high-rate stimulation trial. Horizontal axis: incident power density; the level of 10^{-8} mW cm^{-2} corresponds to sham irradiation. Vertical axis: relative amplitude ratio values (rr*A*, mean ± SE) for the test compound action potential. The number of experiments in each group is the same as in Fig. 2. Dotted areas show the rr*A* value in sham-exposed nerve preparations. See Fig. 2 and text for more explanation.

with and without MMW irradiation. In sham exposed preparations, the test CAP decreased faster during the second HRS trial than it did during the first one, and the rr*A* index decreased to 70–80%. MMW irradiation at 0.02, 0.1, and 2.6 mW cm^{-2} prevented this faster decrease of the test CAP, and the rr*A* index remained near 100%. The maximum effect at all these radiation intensities equaled 20–25% ($p < 0.01$ for 0.1 mW cm^{-2}, $p < 0.05$ for the others). Microwave heating of the preparation at 2.6 mW cm^{-2} did not increase the effect, but could explain greater variability of the data. Unexpectedly, an intermediate intensity of 0.5 mW cm^{-2} induced less changes than higher and lower field intensities.

The dependence of the MMW effect on the radiation intensity is presented in Fig. 3. This dependence was virtually the same for 16 min and 21 min timepoints of the HRS. The rr*A* values at 0.5 mW cm^{-2} were not significantly different from either sham exposure or the other MMW exposure data; so, the reality of the 'dip' at this field level remains questionable. Otherwise, the dependence appeared rather flat, with a threshold intensity at or below 0.02 mW cm^{-2}.

4. Discussion

This study was the third in a row which demonstrated the same effect of MMW, despite considerable differences in experimental procedures. In the first study [16], each nerve preparation was exposed or sham exposed several times, at different radiation frequencies and power levels. We were looking for any MMW exposure-related changes in the CAP conduction velocity, shape and amplitude. The results established several effects of MMW; however, all the effects but one apparently resulted from microwave heating. The only effect which could possibly be produced by a nonthermal mechanism was an attenuation of the HRS-induced decrease of the test CAP. The second study [17,18], when each preparation underwent just one exposure (to exclude possible aftereffects), confirmed this MMW effect and provided strong evidence for its nonthermal mechanism. In both these studies, the rr*A* index in sham exposed nerves stayed about 100%, while MMW at certain frequencies significantly increased it. This effect reached maximum by the end of the HRS, which corresponded to the peak of nerve exhaustion. The frequency of 41.34 GHz was the most effective, providing for the rr*A* increase to 122 ± 7%.

In the present study, we have chosen the frequency of 41.34 GHz to establish the role of field intensity. In addition, we increased the HRS duration from 17 to 22 min: We expected that the MMW effect could possibly become more pronounced if the nerve were driven to a greater exhaustion. However, a side effect of the increased HRS duration was an incomplete restoration of nerve properties after the first HRS trial. As a result, the rr*A* index in sham exposed preparations fell to 70–80%, while MMW irradiation now increased it to about 100%. Contrary to our expectations, the longer HRS did not further increase the MMW effect, and the difference between MMW-exposed and sham exposed preparations was remarkably the same (20–25%) in this and the previous two series of experiments.

Thus, within the studied limits, the MMW effect appeared almost independent from the field intensity. These limits were greater or similar to those used by the other authors in experimentation with excitable tissues [1,14,15], though far less then in the cell culture studies [2–4,6,7]. Due to technical conditions of electrophysiological experiments with isolated preparations, it would have been excessively laborious to use the nerve model to look for the exact threshold or to check whether the frequency resonance width decreases with decreasing the incident power, as found in the cell studies [4,6,7]. At this time we can only conclude that MMW irradiation assists the nerve to sustain the HRS at field levels as low as 0.02 mW cm^{-2} and above, and that this effect is produced by a nonthermal mechanism (the effect did not increase with increasing the field intensity).

The rr*A* value under exposure at 0.5 mW cm^{-2} did not significantly differ from the control; however, it did not significantly differ from the other exposed groups either. No reaction at this power level could indicate the presence of a 'power window', but could be a chance fluctuation as well.

It is important to note that for sham irradiation we tried to imitate the exposure conditions as closely as possible. That is why all devices, including the microwave genera-

tor, were turned on, but the delivery of MMW to the preparation was blocked by waveguide attenuators. This blockage could not be complete, and the field intensity still reaching the preparation was assessed to be about 10^{-8} mW cm^{-2}. There were no findings of MMW effects in isolated organs or organisms at such power levels, but the results obtained with cell cultures supposed that even this very low field intensity could be effective. Therefore, one cannot exclude that the rrA index of 70–80% in sham exposed preparations could be even less if the generator was actually turned off. Though the present data do not allow us to establish it incontrovertibly whether MMW at 10^{-8} mW cm^{-2} was effective or not, this inadequacy does not obscure the existence nor the nonthermal nature of the effect.

Our results are in agreement with observations that nonthermal MMW effects do not necessarily increase with increasing the field intensity, at least within a certain range [2–8,18]. From a physiological point of view, the observed MMW effect can be interpreted either as an improvement of the nerve refractory properties, or as an improvement of the nerve ability to tolerate the HRS. Improvement of the refractory properties could indicate, for example, that MMW facilitate reactivation of membrane sodium channels after spike conduction, but this does not explain why no MMW effect could be found without the HRS [25]. Improvement of the nerve ability to tolerate the HRS could possibly result from a faster resorption of potassium ions from the extracellular space back into nerve fibers; but, in turn, this does not explain why only the test CAPs (and not the conditioning CAPs) were affected by MMW. Understanding of physiological mechanisms underlying this MMW effect will require more sophisticated experimentation, including single-fiber recording and voltage clamp methods.

Acknowledgements

The work was performed while A.G. Pakhomov held a National Research Council-AMRMC Research Associateship, and was supported by the US Army Medical Research and Materiel Command under contract DAMD17-94-C-4069 awarded to McKesson BioServices. The views, opinions and findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy or decision.

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