BIOEM 2016

5-10 June 2016, Het Pand, Ghent, Belgium

The Joint Annual Meeting of
The Bioelectromagnetics Society and the
European BioElectromagnetics Association
co-organized with the European COST EMF-MED Action BM1309

Abstract Collection

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with functional and atomic groups of macromolecules, which leads to an increase in size of the ions or macromolecules, and the latter is the cause of density increase.The results of measurements of the density buffer and the DNA-solution are summarized in Table 1. As it can be seen from the table, there is almost the same dynamics of changing of the buffer and the DNA-solution densities. And the obtained data are in a good agreement with the results of DNA-melting. For irradiation G4-142 generator (Russian made) was used [2], the incident power density (IPD) at the location of object was about 50 $\mu W/cm^2$.

References

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Figures

Time of irradiation, min	Buffer	Buffer + DNA
0	0.999201 ± 0.000005	0.999232 ± 0.000004
30	0.999220 ± 0.000005	0.999242 ± 0.000005
60	0.999241 ± 0.000004	0.999269 ± 0.000004
90	0.999253 ± 0.000004	0.999291 ± 0.000005

Figure 1. Table 1. Magnitude of solution density (g/cm3) before and after exposure of MM-radiation at 64.5 GHz

PB-130 [14:00]

Thermostability of the Mitoxantrone-tumor DNA complexes irradiated by low power electromagnetic waves

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Keywords: In vitro, RF/Microwaves, Completed (unpublished)

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Thermostability of mitoxantrone (MTX) complexes with DNA of sarcoma 45 (tDNA) and healthy rat liver (hDNA) earlier irradiated by resonant and non-resonant frequencies of oscillations of water structures has been studied. It is shown that due to irradiation of DNA complexes by resonant frequencies, dehydration of nucleotides and Na^+ions, present in solution, occurs. As a result of this fact at relatively low concentrations of MTX, when 100 base pairs of DNA corresponds to one of MTX molecules the thermostability of complexes decreases, moreover, the change is more pronounced (~0.8^0C) at complex-formation with tDNA. The results of the work may be applied for treatment of new schemes of anti-tumor preparations in clinics.

We have investigated the thermal stability of the complexes antitumor drug Mitoxantron (MTX) with DNA of the liver normal rats (hDNA) and sarcoma 45 (tDNA), irradiated by low power and non ionizing millimeter electromagnetic waves (MEWs) resonant (64.5 and 50.3 GHz) and non-resonant (48.3 GHz) frequencies of water molecular structures [1].

Table 1 shows the melting parameters of hDNA and tDNA irradiated by resonance frequency of 64.5 GHz,

depending on the duration of exposure. As follows from Table 1, melting temperature T_m of hDNA and tDNA increases when the duration increases up to 90 minutes. Since the greatest change of the melting (1°C for hDNA and 1,5°C for tDNA) parameters occurs at 90 minutes irradiation, further investigations have been carried out after 90 minutes of exposure. Table 2 shows the parameters of the melting hDNA and tDNA irradiated at 90 minutes by resonant (50.3 GHz) and the non-resonant (48.3 GHz) frequencies of MEWs. As shown in Table 2, both resonance frequencies show similar patterns of change in the parameters of melting, however, changes in T_m and ΔT more pronounced by irradiation with a frequency of 64.5 GHz, which coincides with the resonant frequency of hexagonal ring molecular structure of water [1]. The observed stronger changes of the tDNA melting parameter may be due to structural differences compared tDNA to hDNA [2, 3], whereby the hydration of tDNA hyper methylated in certain areas may be very different from the rest of the hydration of DNA [4]. Based on literature data on the effect of non thermal coherent MEWs on the structure of water [1, 4-7] and comparing them with our experimental data, we can assume that as a result of radiation with resonant frequencies (for vibration of water structures) dehydration takes place of nucleotide pairs and Na^+ ions, being in close proximity with regard to the DNA molecule, so that they efficiently stabilize the double helix and even do that stronger in the case of millimeter waves of resonant frequencies (Table. 2). As can be seen from Table 2, the errors of changes of melting parameters of the irradiated by non-resonant frequency of 48.3 GHz samples are within the experimental error. Table 3 shows the decadences of the parameters of the melting of non-irradiated and irradiated DNA-MTX complexes on the concentrations of MTX. It is known that MTX is an intercalating drug [8,9], which increases the thermostability of DNA [8,10]. From Table 3 is follow that at relatively low concentrations of MTX, when one MTX molecule accounts for about 100 or more base pairs of DNA, T_m remains constant within the experimental error, and for irradiated DNA even decreases (0.8^0C for tDNA with MTX complexes). Further concentration increase of MTX, as might be expected, or leads to increase T_m for both non-irradiated and irradiated complexes. From Table 3 is also follow that at relatively low concentrations of MTX ($C_0/C_p < 0.01$), T_m complexes are more strongly modified by complexing with irradiated DNA, which more expressed for tDNA. Decrease of DNA stability can apparently be due to the external binding of MTX with DNA double helix [8,11]. The irradiation causes dehydration of nucleotides [4,6,7], thereby increasing the possibility of MTX molecules bind into the surface of the DNA, which leads to destabilization of the local DNA [12]. It is known that when a malignancy is increased the content of 5-methylcytosine of DNA in solid tumors as compared with DNA the number of healthy animals is increased [2,3,6]. In [13] it was shown that methylate cytosine support the MTX complexation with DNA.Therefore, we can assume that at binding MTX with tumor DNA as compared with DNA extracted from organs of healthy animals, a binding selectivity is observed that increases by preliminary irradiation of DNA molecules with MEW, especially with resonant frequencies for vibrations of molecular water structures. Due to the decrease in stability of tDNA, when they are involved in mitotic cycle, they become more "susceptible to degradation", and this is likely increases the activity of anticancer drugs at their combined in vivo use with radiation.

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Figures

Duration of irradiation min.	hDNA		tDNA	
	T_m, ^0C	ΔT, ^0C	T_m, ^0C	ΔT, ^0C
0	83,0±0,1	5,7±0,1	82,0±0,2	6,6±0,2
30	83,1±0,2	5,8±0,1	82,1±0,2	6,6±0,2
40	83,6±0,1	5,7±0,1	82,3±0,1	6,5±0,2
60	83,9±0,2	5,6±0,2	82,9±0,1	6,3±0,1
90	84,1±0,2	5,6±0,2	83,5±0,2	6,2±0,2
120	84,0±0,2	5,6±0,2	83,5±0,2	6,2±0,2

Figure 1. **Table 1.** The temperature and the melting range of DNA isolated from healthy rats and liver tumor sarcoma 45 irradiated by 64.5 GHz MEW.

Frequency irradiation (GHz)	hDNA		tDNA	
	T_m, ^0C	ΔT, ^0C	T_m, ^0C	ΔT, ^0C
0	83,0±0,1	5,7±0,2	82,0±0,2	6,6±0,2
64,5	84,1±0,2	5,6±0,2	83,5±0,2	6,2±0,2
50,3	83,8±0,1	5,6±0,2	83,2±0,2	6,3±0,2
48,3	83,3±0,2	5,7±0,1	82,3±0,2	6,5±0,1

Figure 2. **Table 2.** The temperature and the melting range of DNA isolated from the liver of normal rats and tumor sarcoma 45, irradiated by MEM at 90min.

The relativ concentration MTX (C_0/C_p)	Non-irradiated				Irradiated (90 min.)			
	hDNA		tDNA		hDNA		tDNA	
	T_m, ^0C	ΔT, ^0C	T_m, ^0C	ΔT, ^0C	T_m, ^0C	ΔT, ^0C	T_m, ^0C	ΔT, ^0C
0	83,0±0,1	5,7±0,2	82,0±0,1	6,6±0,2	84,1±0,2	5,6±0,2	83,5±0,2	6,2±0,2
0,002	82,9±0,2	6,2±0,2	81,9±0,1	7,0±0,2	83,9±0,2	6,3±0,2	82,9±0,2	6,9±0,2
0,005	82,9±0,2	7,3±0,2	81,7±0,2	7,5±0,2	83,7±0,1	7,2±0,2	82,7±0,2	7,8±0,2
0,008	83,1±0,1	7,8±0,2	82,0±0,1	8,2±0,2	83,9±0,1	8,3±0,2	83,2±0,2	8,1±0,2
0,02	84,2±0,2	8,3±0,2	83,5±0,2	8,9±0,2	84,8±0,2	8,8±0,2	83,6±0,2	8,9±0,2
0,04	85,1±0,1	8,4±0,2	84,3±0,2	8,7±0,2	86,6±0,1	8,8±0,2	84,3±0,2	9,0±0,2

Figure 3. **Table 3** Values of the melting parameters unexposed and exposed (with a frequency of 64.5 GHz) hDNA and tDNA in combination with mitoxantrone.

PB-132 [14:00]

Effect of 1950 MHz radiofrequency fields exposure on the biology of human neural stem cells

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Keywords: In vitro, RF/Microwaves, Work in Progress

Presented by: Nam Kim

In the present study, we examined the effect of 1950 MHz radiofrequency fields (RF) radiation on the biology of human neural stem cells. Our results demonstrate that RF exposure significantly reduced the proliferation, decreased the expression of stem cell factor, nestin and increased cell migration factor, N-cadherin in these cells.

Introduction

Rapid increase of mobile phone usage world-wide causes great concern about the potential health risk of radiofrequency (RF) fields exposure in public (1). Since mobile phones usually placed on the ear for use, many studies focused on the impact of RF-EMF on the brain. Among them, some studies suggest that RF exposure may have negative impact on the brain. It has been reported that long-term exposure to RF-EMF from cordless phones is able to cause brain tumor initiation and progression (2). RF-EMF may change gene expression and morphology, and even cell death by trigger genomic and mitochondrial DNA damage in brain cells (3). On the contrary, other reports insist that RF exposure may have beneficial effects on cognitive functions, especially in neurodegenerative diseases including Alzheimer's disease (AD) (4). Given the debates on the impact of RF on the brain, it would be important to define the effect of RF on the brain cells in vitro. In this study, we evaluated the influence of RF against the biology of human neural stem cells.

Methods

In this study, we used immortalized human neural stem cells. Cells were cultured with DMEM/F12 containing B27 supplement with growth factors [20 ng/ml of epidermal growth factor (EGF) and basic fibroblast growth factor (bFGF)] and antibiotics (penicillin and streptomycin). Cell proliferation rate was measured by MTT