DNA RIBOPRINTING AND BIOCHEMICAL STUDIES ON THE EFFECT OF EXPOSURE TO PULSED ELECTROMAGNETIC FIELD ON RATS

Ebtisam Abd El-Mageed Marzook, Mohammed H. Awwad*, G.M. El-Aragi** and Nahed Mohamed EL-Abiad

Biological Application Department, Nuclear Research Center, Atomic Energy Authority, Cairo, Egypt. * Department of Zoology, Faculty of Science, Benha University Egypt. ** Plasma Department, Nuclear Research Center, Atomic Energy Authority, Cairo, Egypt

Key Words: Pulsed EMF, Lipid profile, Blood proteins, Testosterone, DNA genome, RFLPs.

ABSTRACT: In the present study pulsed electromagnetic field (PEMF) was used to evaluate the effect of exposure on some molecular and biochemical aspects in male albino rats (*Rattus rattus*). Three groups of rats were exposed to PEMF (10, IS, 20 pulses/day three times per week for three weeks) with frequency of 8-12 GHz. An unexposed group was considered the control group. At the end of experiment, serum levels of total protein, albumin, globulin, A/G ratio, testosterone, triglycerides and cholesterol were determined. The results revealed that exposure to electromagnetic field induced significant increases in serum total protein, globulin, triglycerides and testosterone hormone on the second exposed group only. Non significant changes were found in serum levels of albumin and cholesterol in the same group with decreased A/G ratio. All the tested parameters were not affected on the other two exposed groups. An indicative hypothesis for spotting the effect of electromagnetic spectrum on the liver of albino rats was proposed based on the examination of total DNA genome by using *Pac*I, *Sfi*I, *Not*I and *Swa*I restriction endonucleases. The results achieved have established the value of the physical map as an additional complement to the standard biodosimetric methods.

INTRODUCTION: Within the past twenty years, several studies indicated a linkage between the exposure to electromagnetic radiation and serious health problems. The widespread applications and use of radiofrequency and microwave devices (300 kHz to 300 GHz) in consumer households, for telecommunications and navigational aids, in

industry & in the military have increased the awareness of potential health hazards in a large cross section of the population (**Robert *et al*., 1997**).

Cellular phones and their base stations emit pulsed microwaves on the environment **(Santini *et al*., 2000 and Radon *et al*., 2006)**. Radar operators are exposed to electromagnetic radiations at frequencies ranging from 390 MHz to 10.96 GHz **(Bergier *et al*., 1990 and Liu *et al*., 2003)**. The largest source of human exposure to electromagnetic field in the home is from the fields generated by many common household appliances and tools, several of these mentioned devices produce local magnetic fields at a distance of 30cm from their surface **(Gauger, 1984).**

X-rays, ultraviolet (UV) light, visible light, infrared light (IR), microwaves (MW), radio - frequency radiation (RF), and magnetic fields from electric power systems are all parts of the electromagnetic (EM) spectrum. The components of the electromagnetic spectrum are characterized by their frequency or wave length. The frequency and wavelength are inversely related; as the frequency rises the wavelength gets shorter. The frequency is the rate at which the electromagnetic field goes through one complete oscillation (cycle) and is usually given in Hertz (Hz), where one Hz is one cycle per second **(John, 2004).**

Increasing number of reports has demonstrated a significant effect of pulsed EMF on aspects of animal and human behavior **(Growe *et al*., 2003 and Cook *et al*., 2004)**. It could promote movement of calcium ions, cell proliferation and the eventual production of pro- inflammatory cytokines in human lymphocytes in combination with high density of 4.75 Tesla **(Aldinucci *et al*., 2003)**. The growth of malignant tumor in mice was inhibited and the ability of immune cell to dissolve cancer cells was improved by pulsed magnetic field, and the DNA content decrease **(Zeng *et al*., 2002)**. While, pulsed magnetic field may lead to inconsistent suppression of nocturnal melatonin synthesis and serum melatonin levels in rats **(Reiter *et al*., 1998)**.

Non-ionizing radiofrequency radiation and microwave have been implicated in adversely affecting the hematopoietic system, the eye, the nervous system and reproductive system, in addition to having carcinogenic or tumor-promoting properties **(Malyapa *et al*, 1997).** Exposure to 2450 MHz electromagnetic radiation causes DNA single-strand breaks and double-strand breaks of rat brain tissue irradiated *in vivo* **(Lai and Singh, 1996).**

There are a lot of methods presently used for the determination of early biological effects of DNA-damaging mediators in environmental and

occupational surroundings. Presently, unstable chromosomal aberrations in peripheral blood lymphocytes, in particular dicentrics, form the majority of fully developed biological markers of ionizing radiation contact (**IAEA, 1986; Carrano and Natarajan, 1988; Bauchinger, 1995; Ramalho *et al*., 1998 and Leonard *et al*., 2005)**.

In the last few years, single cell gel electrophoresis (SCGE) or Comet assay has been extensively used for genotoxicity testing **(Singh *et al*., 1988; Tice *et al*., 1990; Fairbairn *et al*., 1995;Olive, 1999 and Yang *et al*., 2005)**. In the molecular studies, DNA damage estimated by the Comet assay and RFLPs technique have been used as a biomarker of radiation exposure **(Betti *et al*., 1994; Collins *et al*., 1997; Garaj-Vrhovac and Kopjar, 1998; Sram *et al*., 1998; Piperakis *et al*., 1999; Kopjar and Garaj-Vrhovac, 2001; Maluf *et al*., 2001; Garaj-Vrhovac *et al*., 2002 and Awwad and Ebtisam, 2005).**

There is common scientific and public interest in potential health hazards of exposure to electromagnetic fields (EMFs) associated with RF and MW radiation. That interest has resulted in many studies planned to measure both the occupational and residential health risk of EMFs **(Cleveland and Ulcek, 1999 and WHO, 2003)**.

Most *in vivo* studies suggest that RF and MW radiation are not mutagenic and are therefore unlikely to initiate cancer. The majority of studies reported lack of a clastogenic effect **(Verschaeve and Maes, 1998 and Trosic *et al*., 2004)**, but some studies reported an increase in the number of single-strand and double-strand DNA breaks in the brain cells of rats exposed to pulsed or continuous wave of 2.45 GHz radiation at specific absorption rates (SARs) of 0.6 and 1.2 W/kg **(Sarkar *et al*., 1994 and Lai and Singh, 1996)**. These studies have been criticized on the basis of deficiencies in the procedures used to process the DNA and the gel electrophoresis methods used to determine the presence of strand breaks **(Williams, 1996)**.

The current study was therefore conducted to study the effect of exposure to pulsed electromagnetic field on DNA and on some biochemical aspects in male rats, including serum total protein, albumin, globulin, albumin/globulin ratio, total lipids, triglycerides and the level of testosterone hormone.

MATERIALS AND METHODS:

***Animal of study*:** Forty mature male albino rats aged approximately three months and weighing 100-120g. were used. Rats were bred at the animal

house of the Nuclear Research Center. They were caged in plastic cages and divided equally into four groups: one unexposed control group, and 3 exposed groups which were exposed to different number of pulses of electromagnetic radiations (first, second and third group were exposed to 10, 15 and 20 pulses/day, respectively). Rats were given food and water *ad-libitum*, kept under constant laboratory conditions and exposed three times/week for three weeks. The exposure system used to generate the electromagnetic radiations was the micro-focus plasma device (PF-0.l device), under static pressure of one mbar, frequency from 8 to 12 GHz, and operated at 15 Kv charging voltage, Electromagnetic spectrum includes radio waves, infrared, optical (visible light), ultraviolet and x-ray. Exposure was done in the Plasma and Nuclear Fusion Department.

The biochemical studies: The animals were sacrificed at the end of exposure time while they were fasting for about 12 hours, blood samples collected, centrifuged for 10 minutes at 5000 rpm, sera was separated and analyzed for laboratory assessment of serum total proteins, albumin, globulin, albumin/globulin ratio (A/G ratio), triglycerides, cholesterol and testosterone hormone. Calorimetric determination of serum total proteins based on the principle of the Biuret reaction **(Tietz, 1976)** and serum albumin was determined according to the method of **Doumas *et al*. (1971)**. Reagents needed were packaged in a commercial Kit supplied from Stanbio Laboratory Inc., San Antonio, Texas, USA. The concentration of globulin was calculated by subtracting the concentration of albumin from that of the total protein for serum and the A/G ratio was calculated as follows:

A/G Ratio =Albumin (g/100ml) / Globulin (g/100ml)

Total cholesterol and triglycerides were estimated by using enzymatic colorimetric method of **Allain *et al*. (1974) and Fossati and Prencipe (1982)** respectively. Testosterone hormone was measured by using radioimmuno-assay kits supplied by Diagnostica Co., Los Angeles based on the method of **Maruyama (1987)**.

The molecular biological studies: Total DNA was extracted from the liver of the rats as described by **Awwad (2003)**. One µl of the resuspended pellet was checked by gel electrophoresis for the presence of DNA (Fig. 1). In the experiments; the enzymes *Pac*I (Toyobo Biochemicals), *Sfi*I (Promega Corporation), *Not*I (Promega Corporation) and *Swa*I (Sigma Chemical Corporation) were evaluated for their ability

to differentiate between the normal and affected DNA samples of rat. One µl (10-12 units) was used for each digestion reaction, together with 1.2 µl of the respective enzyme buffer for a final volume of 12.2 µl. The digestion was performed for ~3.5 h at 25-50^{o}C, and the digestion products were evaluated on 2% TBE-agarose (FMC Bioproducts) gels and stained with ethidium bromide. Bands were detected upon ultraviolet transillumination and photographed **(Sangumetti *et al*., 1994)**.

Statistical Analysis: The data were analyzed by student's t-test and were considered significant at $p<0.05$.

RESULTS: Results of total protein, albumin, globulin and A/G ratio at different exposed groups to 10, 15 and 20 pulsed electromagnetic field 3 times per week for 3 weeks are presented in Table (1).

The obtained data revealed that exposing the rats to 15 pulses of electromagnetic field led to a significant increase ($p<0.05$) in mean values of total protein and globulin only in the second exposed group (7.1±.33, 3.5±0.37 g/dl, respectively) compared with the control group (6.32±0.59, 2.7±0.6 g/dl respectively), while A/G ratio was significantly lower ($p<0.05$) only in the second and third exposed groups (1.03±0.18, 1.01 ± 0.27) as compared to the control group (1.40 ± 0.32). On the other, hand insignificant differences were found on mean values of serum albumin in the three exposed groups compared with the control group. Also, insignificant differences were found on mean values of serum levels of total protein and globulin in the first and third exposed groups as compared to the control group.

Results of triglycerides, cholesterol and testosterone hormone at different exposed groups to 10, 15 and 20 pulses of electromagnetic field 3 times per week for 3 weeks are presented in Table (2).

The mean values of serum levels of triglycerides and testosterone were significantly increased ($p<0.05$) only in the second exposed group (103.3 ± 23.4 mg/dl, 4.56 ± 1.32 ng/ml respectively) as compared to the control group (64.3 ± 21.7, 2.56 ± 0.95 respectively), while insignificant differences were found on mean values of serum cholesterol in the three exposed groups compared with the control group. Also, insignificant differences were found on mean values of serum levels of triglycerides and testosterone hormone in the first and third exposed groups as compared to the control group.

Total cellular DNA was extracted &om the livers of the normal animal

group and the three treated rat groups (10, 15 and 20 pulses/day), and is represented in Figure 1; lanes 1-4 represented unaffected animal and the three treated rat groups (10, 15 and 20 pulses/day), DNA genome, respectively.

DNA genome has a high molecular weight, so the DNA genome of all ammals remained in the wheel of the agarose gel electrophoresis (lanes 1-4, Figure, 1). Whereas, the 10,000 bp DNA Ladder represented in the first iane was separated into bands with different lengths (250, 500, 750, 1000, 1500, 2000, 2500, 3000, 3500, 4000, 5000, 6000, 8000 and 10,000 bp, respectively; lane M).

*Pca*I, *Sfi*I, *Not*I and *Swa*I restriction endonucleases were used to detect the effect of the electromagnetic radiation on rat total DNA genome.

*Pca*I restriction enzyme digested the DNA genome of the normal rats into eight restriction fragments at the area of the 10,000 bp marker (~1100, ~1300, ~2100, ~2300, ~2600, ~3000, ~5500 and ~10000 bp; table 3 and figure 2: lane 1). The same restriction enzyme cut the first group of the treated rats (10 pulses/day) when its DNA genome was digested into 14 restriction bands (~750, ~900, ~1000, ~1100, ~1300, ~2100, ~2300, ~2600, ~3000, ~3500, ~3700, ~4500, ~5000 and ~10,000 bp; table 3 and figure 2: lane 2). The DNA genome of the second group of treated rats (15 pulses/day) was digested into 17 fragments (~600, ~750, ~900, ~1000, ~1100, ~1200, ~1500, ~2200, ~2500. ~2900, ~3500, ~3800, ~4000, ~5800, ~7500, ~8200 and ~10,000 bp; table 3 and figure 2: lane 3). PcaI enzyme digested the DNA genome of the third group of treated rats (20 pulses/day) into 12 bands (~300, ~400, ~600, ~1000, ~1200, ~2200, ~2500, ~2900, ~3000, ~3500, ~5500 and ~10,000 bp; table 3 and figure 2: lane 4).

At the same time, *Sf1*I restriction endonuclease clarified the electromagnetic radiation influencing liver of rats with the different pulses and the normal liver of the same rats. SfiI restriction enzyme fragmented the DNA genome of normal rat liver sample into 7 patterns (~1200, ~1300, ~700, ~2000, ~2500, ~3000 and ~10,000 bp; table 4 and figure 3: lane 1) and digested the DNA genome of liver of the first treated group (10 pulses/day) into 8 cuts (~800, ~1200, ~1300, ~2000, ~2500, ~3000, ~4500 and ~10,000 bp; table 4 and figure 3: lane 2). Also, the DNA genome of the rat exposed to 15 pulses/day was fragmented into eight patterns but in different sizes (~700, ~1300, ~1700, ~2000, ~2500, ~3000, ~3500 and ~4500 bp; table 4 and figure 3: lane 3) and the DNA genome of the third group of rats (20 pulses/day) into 9 different sizes of

fragments (~600, ~700, ~1300, ~1700, ~2000, ~2500, ~3000, ~3500 and ~4500 bp; table 4 and figure 3: lane 4).

The effect of electromagnetic radiation on the rats was clarified when the DNA genome of these rats was digested with *Not*l restriction endonuclease. The normal DNA genome of rat liver was digested with *Not*l restriction enzyme giving 10 restriction patterns (~200, ~750, ~850, ~1000, ~1450, ~2200, ~2700, ~3500, ~7800 and ~10,000 bp; table 5 and figure 4: lane 1). The same restriction endonuclease cut the DNA genome of the first treated group of rats (10 pulses/day) into 11 fragments (~200, ~700, ~800, ~1000, ~1450, ~2000, ~2300, ~2700, ~3500, ~5000 and ~7800 bp; table 5 and figure 4: lane 2). The DNA genome of the second treated group of rats (15 pulses/day) gave 11 different patterns (~200, ~700, ~800, ~1450, ~2300, ~3000, ~3700, ~4500, ~5500, ~6000 Gild ~8000 bp; table 5 and figure 4: lane 3) when digested with Natl restriction enzyme. *Not*l restriction endonuclease cut the DNA genome of the third treated group of rats (20 pulses/day) into 15 restriction fragments (~200, ~700, ~800, ~1000, ~1200, ~1700, ~2000, ~2300, ~3000, ~4200, ~5000, ~6000, ~7000, ~9000 and ~I 0,000 bp; table 5 and figure 4: lane 4).

Also, the hazardous effect of electromagnetic radiation on the liver of rats was shown when their DNA genome digested with *Swa*l restriction endonuclease. *Swa*l enzyme cut the DNA genome of nonual liver of rats into 10 restriction fragments (~200, ~900, ~1200, ~1400, ~1600, ~1700, ~2300, ~2700, ~3700 and ~8000 bp; table 6 and figure 5: lane 1). The same restriction endonuclease digested the DNA genome of the first treated group of rats (10 pulses/day) into 12 fragments (~200, ~900, ~1200, ~1400, ~1600, ~2200, ~2500, ~2700, ~3600, ~3800, ~5000 and ~6000 bp; table 6 and figure 5: lane 2). The DNA genome of the second group of treated rats (15 pulses/day) gave 13 restriction fragments (~200, ~500, ~1000, ~1100, ~1200, ~1400, ~1600, ~1700, ~2300, ~2700, ~4500, ~5500 and ~8000 bp; table 6 and figure 5: lane 3) when they were digested with *Swa*l restriction enzyme. Also, the same restriction endonuclease cut the DNA genome of the third group of treated rats (20 pulses/day) into 14 restriction patterns (~200, ~1000, ~1300, ~1400, ~1700, ~2000, ~2300, ~2500, ~3000, ~3500, ~4000, ~6000, ~8000 and ~9000 bp; table 7 and figure 5: lane 4).

DISCUSSION: Our surroundings are full of non-ionizing electromagnetic radiation of different frequencies emitted by television, computer and cellular phone sets. These emissions have been increasing

over the past few years **(Monfrecola *et al*., 2003).**

Most blood proteins are synthesized in the liver. Several factors affect the rate of protein synthesis such as hormones (e.g thyroxin and cortisol), liver function, nutritional and environmental factors. Protein functions include carriers of many physiological parameters besides signaling roles, thus it is not surprising that direct consequences can arise from mutation either in genes that encode proteins or in regions of DNA that control gene expression **(Margaret and Robert, 2000)**.

The results of the present work showed that total proteins and globulin levels in blood of rats were markedly increased in response to magnetic field in the second exposed group. The increase of globulin level after 15 pulses exposure observed in the present work might be explained by the enhanced production of immunoglobulin responsible for immune response as a defence mechanism of the body against radiations and the increased total proteins might be due to a stimulatory effect of EMF on thyroid. Humans occupationally exposed to EMF were reported to have increased hyperactivity of the thyroid gland function **(Dumansky *et al*., 1976 and Adey 1981)**. The current results were in agreement with those of **Kumosani *et al*. (1996)** who showed an elevation in plasma total proteins in mice exposed to different magnetic fluxes for 24 hrs. They demonstrated that this rise was due to either to loss of fluid from the body (dehydration) or to an increase in globulins. These results were in disagreement with those of **Dumansky *et al*. (1976)** and **Mohamed *et al*. (1996)** who found a significant decrease in plasma globulin in rats exposed to different intensities of magnetic fields.

A/G ratio was markedly decreased in the second and third exposed groups. Such results were in agreement with those of **Mohamed *et al*. (1996)**, who reported decreased A/G ratio in rats chronically exposed to EMF. The decreased level of A/G ratio observed in the present work might be explained by the increased values of globulin.

Triglycerides (Triacylglycerol) are a major energy reserve and the principal neutral derivatives of glycerol in animals. They are found primarily in the adipose tissue and serve as a storage sites for lipids **(Reginald and Charles 1995).**The significant increase in the plasma triglycerides in the current study in the second exposed group could be due to the inhibition of lipoprotein lipase activity which leads to post irradiation hypertriacylglycerols **(Sedlakova *et al*., 1988)**.

Testosterone is the principal male sex hormone steroid, which is synthesized from cholesterol. Testosterone is an androgen synthesized in

males primarily in the testes and in smaller amounts in the adrenal cortex. Androgens are necessary for sperm maturation; even non-reproductive tissues (liver, brain and skeletal muscle) are susceptible to the effect of androgens **(Reginald and Charles, 1995).** Thus, in the current study serum levels of testosterone hormone was affected by exposure to the 15 pulses electromagnetic field group, since sex hormones secretions depends mainly on gonads function. So, it could be suggested that exposure might affect the sex organs and hypothalamic-pituitary adrenal axis of male rats or might be due to the effect on the hypothalamic centers responsible for gonadotropin releasing hormone production which become less sensitive to feedback inhibition by gonadal steroid hormone since the interaction of testosterone with its receptor provides for feedback control of gonadotropin release **(Murray *et al.,* 1998)**. These results were in agreement with those of **Hiroyuki *et al.* (2000)** who reported that microwave exposure affects the endocrine and biochemical mediators including corticosterone.

The second group was more affected by all tested parameters; two of the rats died during exposure. In the other two groups, some parameters were affected but not significantly. Also, there was a variation in mean value of the individual data within the same group. These observations could be explained by the fact that not every individual would be equally susceptible, even when exposed to exactly the same radiation for exactly the same length of time. Susceptibility depends not only on the radiation, but also on genetic predisposition and the physiological state of the individual when irradiated **(Jerald, 2002)**. However, not all the signals of the EMF are harmful since exposure to pulsed magnetic fields had a therapeutic effect in both animals and humans **(Shupak et. al., 2004)**.

Ionizing radiation is a ubiquitous environmental physical agent whose DNA-damaging effects are fairly well established. In physico-chemical interaction with cellular DNA it produces a variety of primary lesions, such as single-strand breaks (SSBs), alkali-labile sites, double-strand breaks (DSBs), DNA - DNA and DNA - protein crosslinks and damage to purine and pyrimidine bases **(Natarajan, 1993; Kruszewski *et al.,* 1998; Chaubey *et al.,* 2001 and Feinendegen and Neumann, 2006).**

Cytogenetic effects have been reported in various types of cells after exposure to electromagnetic radiation **(Garaj-Vrhovac *et al.,* 1990; 1991; Maes *et al.,* 1993; Narasimhan and Huh, 1991; Sagripanti and Swicord, 1986; Verschaeve *et al.,* 1994 and Nakamura *et al.,* 2006).** Several studies have reported cytogenetic changes in cells by

electromagnetic radiation, and these results could be an important indication of the health effects of electromagnetic radiation. **Singh *et al.* (1994)** reported significant decreases in poly-ADP-ribosylation, a process involved in chromatin functions, in the cells of rats after exposure to electromagnetic radiation. **Sarkar *et al.* (1994)** reported changes in DNA sequences in mouse cells after exposure to electromagnetic radiation. Also, **Lai and Singh (1995)** reported an increase in single strand DNA breaks in cells of rats after exposure to electromagnetic radiation.

Genetic damage to DNA genome of cells can result in carcinogenesis. However, since cells do not undergo mitosis, a more likely consequence of DNA damage is changes in functions and cell death, which could either lead to or accelerate the development of cell degenerative diseases. **Lai and Singh (1996)** have reported an increase in DNA double strand breaks in cells of rats after acute exposure to electromagnetic radiation. Double strand breaks, if not properly repaired, are known to lead to cell death. Indeed, **Lai and Singh (1996)** and **Reagan-Shaw *et al.* (2006)** have observed an increase in apoptosis (scheduled cell death) in cells exposed to electromagnetic radiation. That type of response would lead to an inverted-U response function in carcinogenesis and might explain reports of an increase **(Repacholi *et al.*, 1997)**, a decrease **(Adey *et al.*, 1996)**, and no significant effect **(Adey *et al.*, 1997)** on cancer rate of animals exposed to electromagnetic radiation.

The present study demonstrated that the DNA genome of rat cells was seriously affected by electromagnetic radiation when digested with *Pca*I, *Sfi*I, *Not*I and *Swa*I restriction endonucleases.

Each restriction enzyme proved that there are strong deviations between the normal DNA genome and the three treated groups (10, 15 and 20 pulses/day) of rat liver according to the physical map or the profile of digestion. Also, the high values of DNA migration recorded in some exposed animals are obviously the result of differential concurrent exposure. The restriction fragment length polymorphisms (RFLPs) of the total DNA genome revealed heterogeneity in the level of DNA mutation or breakage induced in hepatocytes by occupational exposure to ionizing radiation.

Therefore, the biochemical and the molecular studies revealed that the electromagnetic spectrum contains waves which are highly hazardous to living organisms. Moreover, radiation workers are expected to have a greater percentage risk of developing detrimental effects due to of their generally greater exposure.

REFERENCES

Adey, W.R. (1981): Tissue interaction with nonionizing electromagnetic field. Physiological Review 61 (2): 435-514.

Adey, W.R.; Byus, C.V.; Cain, C.D.; Haggren, W.; Higgins, R.J.; Jones, R.A.; Kean, C.J.; Kuster, N.; MacMurray, A.; Phillips, J.L.; Stagg, R.B. and Zimmerman, G. (1996): Brain tumor incidence in rats chronically exposed to digital cellular telephone fields in an initiation-promotion model. 18th Annual Meeting of the Bioeletromagnetics Society, Victoria, B.C., Canada, June 9-14.

Adey, W.R.; Byus, C.V.; Cain, C.D.; Haggren, W.; Higgins, R.J.; Jones, R.A.; Kean, C.J.; Kuster, N.; MacMurray, A.; Phillips, J.L.; Stagg, R.B. and Zimmerman, G. (1997): Brain tumor incidence in rats chronically exposed to frequency-modulated (FM) cellular phone fields. Second World Congress for Electricity in Biology and Medicine, Bologna, Italy, June 8-13.

Aldinucci, C.; Garcia, J.B.; Palmi, Md Sgaragli, G.; Benocci, A.; Meini, A.; Pessina, Fd Rossi, C.; Bonechi, C. and Pessina, G.P. (2003): The effect of exposure to high flux density static and pulsed magnetic fields on lymphocyte function. Bioelectromagnetics 24(6):373-379.

Allain, C.C.; Poon, L.S.; Chain, C.S.G.; Richmond, W. and Fu, C. (1974): Enzymatic determinations of total serum cholesterol. Clin. Chem., 20(4): 470-475.

Awwad, M.H. (2003): Molecular identification of *Biomphalaria alexandrina* and *Bulinus truncatus* using PCR-RFLP of Actin gene. J. Egypt. Acad. Soc. Environ. Develop., 3 (1): 39-52.

Awwad, M.H. and Ebtisam, A.M. (2005): The electromagnetic spectrum induces mutation in mitochondrial NADH ubiquinone oxidoreductase subunit gene. J. Egypt. Acad. Soc. Environ. Develop., 6 (1): 41-60.

Bauchinger, M. (1995): Quantification of low-level radiation exposure by conventional chromosome aberration analysis. Mutat. Res., 339: 177-189.

Bergier, L.; Lisiewicz, J.; Moszczynski, P.; Rucinska, M. and Sasiadek, U. (1990): Effect of electromagnetic radiations on T. lymphocyte subpopulations and immunoglobulin level in human blood serum after occupational exposure. Med. Pr., 41(4): 211-215.

Betti, C.; Davini, T.; Gianessi, L.; Loprieno, N. and Barale, R. (1994):

Microgel electrophoresis assay (comet test) and SCE analysis in human lymphocytes from 100 normal subjects. Mutat. Res., 307: 323.

Carrano, A.V. & Natarajan, A.T. (1988): Considerations for population monitoring using cytogenetic techniques. Mutat. Res., 204: 379

Chaubey, R.C.; Bhilwade, H.N.; Rajagopalan, R. and Bannur, S.V. (2001): Gamma ray induced DNA damage in human and mouse leukocytes measured by SCGE-Pro: a software developed for automated image analysis in data processing for Comet assay. Mutat. Res., 490: 187-197.

Cleveland, R.F. and Ulcek, J.L. (1999): Questions and answers about biological effects and potential hazards of radio frequency electromagnetic fields. OET Bull., 56: 1-36.

Collins, A.; Dusinska, M.; Franklin, M.; Somorovska, M.; Petrovska, H.; Duthie, S.; Fillion, L.; Panayiotidis, M.; Raslova, K. and Vaughan, N. (1997): Comet assay in human biomonitoring studies: reliability, validation & applications. Environ. Mol. Mutagen., 30: 139.

Cook, C.M.; Thomas, A.W. and Prato F.S. (2004): Resting EEG is affected by exposure to a pulsed ELF magnetic field. Bioelectromagnetics 25(3):196-203.

Doumas, B.T.; Watson, W.A. and Homer, C.B. (1971): Albumin standard and the measurements of the serum albumin with bromocresol green. Clin. Chim. Acta 31: 87-96.

Dumansky, T.U.; Popovich, V.M. and Prokhvatilo, E.V. (1976): Hygienic evaluation of the electromagnetic field created by high voltage electric power transmission lines. Gig. Saint., 8: 19-23.

Fairhairn, D.W.; Olive, P.L. and O'Neill, K,L. (1995): The comet assay: a comprehensive review. Mutat. Res., 33: 37-59.

Feinendegen, L.E. and Neumann, R.D. (2006): The issue of risk in complex adaptive systems: the case of low-dose radiation induced cancer. Hum. Exp. Toxicol., 25(l):11-17.

Fossati, P. and Prencipe, L. (1982): Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clin. Chem., 28: 2077-2080.

Garaj-Vrhovac, V. and Kopjar, N. (1998): The comet assay - a new technique for detection of DNA damage in genetic toxicology studies and human biomonitoring. Period. Biol., 100: 361-366.

Garaj-Vrhovac, V.; Horvat, D. and Koren, Z. (1990): The effect of microwave radiation on cell genome. Mutat. Res., 243:87-93.

Garaj-Vrhovac, V.; Horvat, D. and Korea, Z. (1991): The relationship

between colony-forming ability, chromosome aberrations and incidence of micronuclei in V79 Chinese hamster cells exposed to microwave radiation. Mutat. Res., 263: 143-149.

Garaj-Vrhovac, V.; Kopjar, N.; Raziem, Dd Vekic', B.; Miljani, S. and Ranogajec- Komor, M. (2002): Application of the alkaline comet assay in biodosimetry: assessment of in vivo DNA damage in human peripheral leukocytes after gamma radiation incident. Radiat. Prot. Dosim, 98: 407-416.

Gauger, J.R. (1984): Household appliance magnetic field survey. IIT Res. Inst. Rep. No E 06549-3. U.S. Noval Eledien. Syst. Command. Washington. DC.

Growe, M.G.; Sun, Z.P.; Battocletti, J.H.: Macias, M.Y.;Pintar,F.A. and Maiman, D.J. (2003): Exposure to pulsed magnetic fields enhances motor recovery in cats after spinal cord injury. Spine 28(24):2660-2666.

Hiroyuki, N.; Hirofumi, N.; Keilei, O. and Echiyo, M. (2000): Uteroplacental circulatory disturbance mediated prostaglandin F2u in rats exposed to microwave. Reprod. Toxicol., 14(3):235-240.

IAEA (1986): Biological Dosimetry -- Chromosomal Aberration Analysis for Dose Assessment, International Atomic Energy Technical Report Series no. 260. IAEA, Vienna: 1-69.

Jerald, J.H. (2002): How exposure to GSM and TETRA Base-Station Radiation can adversely affect humans. In: International Institute of Biophysics Neuss-Holzheim, Germany.

John, E.M. (2004): Power lines and cancer FAQs. Medical college of Wisconsin. Milwaukee, Wise, U.S.A.

Kopjar, N. and Garaj-Vrhovac, V. (2001): Application of the alkaline comet assay in human biomonitoring for genotoxicity: a study on Croatian medical personnel handling antineoplastic drugs. Mutagenesis 16: 71-78.

Kruszewski, M.; Wojewodzka, M.; Iwanenko, T.; Collins, A.R. and Szumiel, I. (1998): Application of the comet assay for monitoring DNA damage in workers exposed to chronic low-dose irradiation. II. Base damage. Mutat. Res., 416: 37-57.

Kumosani, T.A.; Bokhari, M.M. and EL-Mashak, E.M. (1996): Effect of environmental elecuomagnetic fields on glucose and total protein in the blood plasma of mice. Comparat. physioL, 333-348.

Lai, H. and Singh, N.P. (1995): Acute low-intensity microwave

exposure increases DNA single-strand breaks in rat brain cells. Bioelectromagnetics, 16: 207-210.

Lai, H. and Singh, N.P. (1996): DNA Single- and double-strand DNA breaks in rat brain cells after acute exposure to low-level radiofrequency electromagnetic radiation. Int. J. Radiat. BioL, 69: 513.

Leonard, A.; Rueff, J.; Gerber, G.B. and Leonard, E.D. (2005): Usefulness and limits of biological dosimetry based on cytogenetic methods. Radiat. Prot. Dosimetry 115(1-4):448-454.

Liu, X.; Yan, S.W.; Ding, X.P.; Zhang, N.; Lu, H.O. and Tang, J. (2003): Evaluation of radiation damage to the sperm DNA of radar operators. Zhonghua Nan Ke Xue. 9(7): 494-496.

Maes, A.; Verschaeve, L.; Arroyo, A.; De Wagter, C. and Vercruyssen, L. (1993): In vitro cytogenetic effects of 2450 MHz waves on human peripheral blood lymphocytes. Bioelectromagnetics 14: 495-501.

Maluf, S.W.; Passos, D.F.; Bacelar, A.; Speit, G. and Erdtmann, B. (2001): Assessment of DNA damage in lymphocytes of workers exposed to X-radiation using the micronucleus test and the comet assay. Environ. Mol. Mutagen., 38: 311-315.

Malyapa, R.S.; Eric, W.A.; William, L.S; Eduardo, G.M.; William, F.P. and Joseph, L.R. (1997): Measurement of DNA damage after exposure to 2450 MHz electromagnetic radiation. Rad. Res., 148: 608.

Margaret, L.R. and Robert, K.M. (2000): Plasma proteins, immunoglobulins and blood coagulation. In: Harper's Biochemistry. Robert, K.M.; Dary L.K.G.; Peter, A.M. and Victor, W.R. ed. A LANGE medical book. McGraw-Hill - USA 59: 737-741.

Maruyama, Y.; Aoki, N.; Suzuki, Y.; Ohno, Y. and Imamura, M. (1987): Sex-steroid-binding plasma protein (SBP), testosterone, estradiol & DHEA in prepuberty & puberty. Acta Endocrinol., 114: 60.

Mohamed, A.A.; Mervat, A.A.; Hoda, E.F and Abdel R.S. (1996): A study on some metabolic and endocrinal effects of exposure to electromagnetic radiation. Bull. Egypt Soc. Physiol. Sci., 16 (I), 290.

Monfrecola, G.; Moffa, G. and Procaccini, E.M. (2003): Non-ionizing electromagnetic radiation emitted by a cellular phone, modify cutaneous blood flow. Dermatology 207(1): 10-14.

Murray, R.K.; Granner, D,K.; Mayes, P.A. and Rodwell, V.W. (1998): Biochemistry of extracellular and intracellular communications: Hormones of the gonads. In: Harper's Biochemistry. Chapter 5. (21 edition), 530-546.

Nakamura, N.; Cullings, H.M.; Kodama, Y.; Wada, T.; Miyazawa, C.; Lee, K. and Awa, A.A. (2006): A Method to Differentiate between the Levels of ESR Signals Induced by Sunlight and by Ionizing Radiation in Teeth from Atomic Bomb Survivors. Radiat. Res., 165(3): 359-64.

Narasimhan, V. and Huh, W.K. (1991): Altered restriction patterns of microwave irradiated lambdaphage DNA. Biochem. Inter., 25: 363.

Natarajan, A.T. (1993): Mechanisms for induction of mutations and chromosome alterations. Environ. Health Perspect., 101 (3): 225-229.

Olive, P.L. (1999): DNA damage and repair in individual cells: applications of the comet assay in radiobiology. Int. J. Radiat. Biol., 75: 395-405.

Piperakis, S.M.; Visvardis, E.E. and Tassiou, A.M. (1999): Comet assay for nuclear DNA damage. Methods Enzymol., 300: 184-194.

Radon, K.; Spegel, H.; Meyer, N.; Klein, J.; Brix, J.; Wiedenhofer, A.; Eder, H.; Praml, G.; Schulze, A.; Ehrenstein, V.; von Kries, R. and Nowak, D. (2006): Personal dosimetry of exposure to mobile telephone base stations. An epidemiologic feasibility study comparing the Maschek dosimeter prototype and the Antennessa SP-090 system. Bioelectromagnetics 27(l):77-81.

Ramalho, A. T.; Costa, M.L.P. and Oliveira, M.S. (1998): Conventional radiation biological dosimetry using frequencies of unstable chromosome aberrations. Mutat. Res., 404: 97-100.

Reagan-Shaw, S.; Breur, J. and Ahmad, N. (2006): Enhancement of UVB radiation-mediated apoptosis by sanguinarine in HaCaT human immortalized keratinocytes. Mol. Cancer Ther., 5(2): 418-29.

Reginald, H.G. and Charles, M.G. (1995): In:Biochemistry. "Synthesis and metabolism of steroid hormones". Library of Congress. USA. Chapter 24. pp: 799.

Reiter, R.J.; Tan, D.X.; Poeggeler, B. and Kavet, R. (1998): Inconsistent suppression of nocurnal pineal melatonin synthesis and serum melatonin levels in rats exposed to pulsed DC magnetic fields. Bioelectromagnetics 19(5): 318-329.

Repacholi, M.H.; Basten, A.; Gebski, V.; Noonan, D.; Finnie, J. and Harris, A.W. (1997): Lymphomas in Em-Piml transgenic mice exposed to pulsed 900-MHz EM fields. Radiat. Res., 147: 631-640.

Robert, S.M.; Eric, W.A.; William, L.S; Eduardo, G.M.; William, F.P. and Joseph, L.R. (1997): Measurement of DNA damage after exposure to 2450 MHz EM radiation. Radiat. Res., 148: 608.

Sagripanti, J.L. and Swicord, M.L. (1986): DNA structural changes caused by microwave radiation. Int. J. Rad. Biol., 50: 47-50.

Sanguinetti, C.; Neto, K. and Simpson, A. (1994): Rapid silver staining and recovery of PCR products separated on polyacrylamide gels. Biotechniques 17: 915- 918.

Santini, Rd Seigne, M.; Bonhomme-Faivre, L. (2000): Danger of cellular telephones & their relay stations. Pathol. BioL, (Paris). 48: 525.

Sarkar, S.; Ali, S. and Bahari, J. (1994): Effects of low power microwave on the mouse genome: a direct DNA analysis. Mutat. Res., 320: 141-147.

Sedlakova, A.; Paulikova, E. and Timko, J. (19SS): Lipids in bone marrow and thymus of continuously irradiated rats. Radiobiol. Radiother., 29(2): 171.

Shupak, N.M.; Prato, F.S. and Thomas, A.W. (2004): Human exposure to a specific pulsed magnetic field: effects on thermal sensory and pain thresholds. Neuroscience (lett.). 363(2): 157-162.

Singh, N.; Rudra, N.; Bansal, P.; Mathur, R.; Behari, J. and Nayar, U. (1994): Poly ADP ribosylation as a possible mechanism of microwave-biointeraction. Indian J. Physiol. Pharmacol., 38: 181-184.

Singh, N.P.; McCoy, M.T.; Tice, R.R. and Schneider, L.L. (1988): A simple technique for quantitation of low levels of DNA damage in individual cells. Exp. Cell Res., 175: 184-191.

Sram, R.J.; Podrazilova, K.; Dejmek, J.; Mrackova, G. & Pile; k,T. (1998): Single cell gel electrophoresis assay: sensitivity of. peripheral white blood cells in human population studies. Mutagenesis 13: 99.

Tice, R.R.; Andrews, P.W.; Hirai, O. and Singh, N.P. (1990): The single cell gel assay: an electrophoretic technique for the detection of DNA damage in individual cells. In Witmer, C.M4 Snyder, R.R.; Hollow, D.J.; Kalf, G.F.; Kocsis, J.J. and Sipes, J.G. (eds), Biological *Reactive Intermediates. IV Molecular and Cellular Effects and their Impact on Human Health.* Plenum Press, New York, 157- 164.

Tietz, N.W. (1976): Biuret method for determination of total protein in serum In: Fundamental of Clinical Chemistry, W.B. Saunders Co., Philadelphia. London. Toronto, P. 503 and P 876.

Trosic, I.; Busljeta, I. and Modlic, B. (2004): Investigation of the genotoxic effect of microwave irradiation in rat bone marrow cells: *in vivo* exposure. Mutagenesis 19(5): 361-364.

Verschaeve, L. and, Maes, A. (1998): Genetic, carcinogenic and teratogenic effects of radiofrequency fields. Mutation Res., 410: 141.

Verschaeve, L.; Slaets, D.; Van Gorp, U.; Maes, A. & Vankerkom, J. (1994): In vitro and in vi vo genetic effects of microwaves from mobile telephone frequencies in human and rat peripheral blood lymphocytes. Proceedings of Cost 244 Meetings on Mobile Communication and Extremely Low Frequency Field: Instrumentation and Measurements in Bioelectromagnetics Research. D. Simunic (ed.), 74-83.

Williams, G.M. (1996): Comment on "Acute low-intesity microwave exposure increases DNA single-stand breaks in rat brain cells" by Henry Lai and Naredra Singh (Letter). Bioelectromagnetics, 17: 165.

World Health Organization (2003): International EMF Project. Available online at: WHO.int/peh-emf/; (accessed 14 April 2003).

Yang, H.; Kong, L. and Zhao, J.S. (2005): DNA damage induced by methyl tertiary-sbutyl ether in viva and in vitro. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 23(5): 362-365.

Zeng, F.;Zheng, G.;Zhang, X.; Li, Z.; Li, C.;Wang, X. and Zhang, H. (2002): Experimental studies on ultra-low frequency pulsed gradient magnetic field inducing apoptosis of cancer cell and inhibiting growth of cancer cell. Science. China Series.C.Life Science 45.(1): 33.

Table (1): Serum level of total protein, albmin, globlin and A/G ratio in control and different exposed groups to pulsed EMF.

Parameter	Control	10 pulsed	15 pulsed	20 pulsed
Total protein (g/dl)				
Mean±S.D	6.32±0.59	5.6 ± 0.58	7.1 ± 0.33	6.4 ± 1.3
Range	(5.4 – 6.8)	(5.0 – 6.5)	(6.8 – 7.7)	(4.7 – 8.2)
P		0.06	0.02*	0.88
Albumin (g/dl)				
Mean±S.D	3.62 ± 0.32	3.275± 0.41	3.58±0.28	3.15±0.62
Range	(3.2 − 0.41)	(2.8 – 3.9)	(3.3 – 4.1)	(2.5 – 4.1)
P		0.13	0.84	
Globlin (g/dl)				
Mean±D	2.7 ± 0.6	2.3 ± 0.34	3.5 ± 0.37	3.26 ± 0.88
Range	(2.1 – 4.1)	(1.75 – 2.7)	(3 – 4.1)	(2.1 – 4.1)
P			0.02*	0.22
A/G ratio				
Mean±S.D	1.40 ± 0.32	1.42± 0.28	1.03 ± 0.18	1.01 ± 0.27
Range	(0.89 – 1.76)	(1.07 – 1.86)	(0.87 – 1.37)	(0.62 – 1.43)
P		0.89	0.03*	0.04*

* Significant $p<0.05$

Table (2): Serum level of triglycerides, cholesterol and testosterone in control and different exposed groups to pulsed EMF.

Parameter	Control	10 pulsed	15 pulsed	20 pulsed
Triglycerides(mg/dl) Mean±S.D Range P	64.3 ±21.7 (33 – 00)	86 ± 21.0 (62 – 112) 0.1	103.3±23.4 (77 – 146) 0.01*	81.3 ± 14.4 (68 – 98) 0.14
Cholesterol (g/dl) Mean±S.D Range P	99 ± 26.1 (62 − 126)	79.8 ± 13.8 (60 – 100) 0.14	79 ± 15.2 (76 – 117) 0.86	84.5 ± 21.2 (63 – 117) 0.31
Testosterone (g/dl) Mean±S.D Range P	2.56 ± 0.95 (1.85 – 4.05)	2.86 ± 0.25 (2.21 – 3.60) 0.56	4.56 ± 1.32 (3.16–3.60) 0.02*	4.91 ± 2.2 (2.89 – 7.70) 0.06

* Significant $p<0.05$

Table (3): Represents the length of DNA (between ~1000 bp), which was digested with the endonucleases *Pac*I in the studied samples.

Band #	Normal	10 p/d	15 p/d	20 p/d
1	~1100	~750	~600	~300
2	~1300	~900	~750	~400
3	~2100	~1000	~900	~600
4	~2300	~1100	~1000	~1000
5	~2600	~1300	~1100	~1200
6	~3000	~2100	~1200	~2200
7	~2500	~2300	~1500	~2500
8	~10000	~2600	~2200	~2900
9	--------	~3000	~2500	~3000
10	--------	~3500	~2900	~3500
11	--------	~3700	~3500	~5500
12	--------	~4500	~3800	~10000
13	--------	~5000	~4000	--------
14	--------	~10000	~5800	--------
15	--------	--------	~7500	--------
16	--------	--------	~8200	--------
17	--------	--------	~10000	--------

Table (4): Represents the length of DNA (between ~1000 bp), which was digested with the endonucleases *Sfi*I in the studied samples.

Band #	Normal	10 p/d	15 p/d	20 p/d
1	~1200	~800	~700	~600
2	~1300	~1200	~1300	~700
3	~1700	~1300	~1700	~1300
4	~2000	~2000	~2000	~1700
5	~2500	~2500	~2500	~2000
6	~3000	~3000	~3000	~2500
7	~10000	~4500	~3500	~3000
8	--------	~10000	~4500	~3500
9	--------	--------	--------	~4500

Table (5): Represents the length of DNA (between ~1000 bp), which was digested with the endonucleases *Not*I in the studied samples.

Band #	Normal	10 p/d	15 p/d	20 p/d
1	~200	~200	~200	~200
2	~750	~700	~700	~700
3	~850	~800	~800	~800
4	~1000	~1000	~1450	~1000
5	~1450	~1450	~2300	~1200
6	~2200	~2000	~3000	~1700
7	~2700	~2300	~3700	~2000
8	~3500	~2700	~4500	~2300
9	~7800	~3500	~5500	~3000
10	~10000	~5000	~6000	~4200
11	--------	~7800	~8000	~5000
12	--------	--------	--------	~6000
13	--------	--------	--------	~7000
14	--------	--------	--------	~9000
15	--------	--------	--------	~10000

Table (5): Represents the length of DNA (between ~1000 bp), which was digested with the endonucleases *Saw*I in the studied samples.

Band #	Normal	10 p/d	15 p/d	20 p/d
1	~200	~200	~200	~200
2	~900	~900	~500	~1000
3	~1200	~1200	~1000	~1300
4	~1400	~1400	~1100	~1400
5	~1600	~1600	~1200	~1700
6	~1700	~2200	~1400	~2000
7	~2300	~2500	~1600	~2300
8	~2700	~2700	~1700	~2500
9	~3700	~3600	~2300	~3000
10	~8000	~3800	~2700	~3500
11	--------	~5000	~4500	~4000
12	--------	~6000	~5500	~6000
13	--------	--------	~8000	~8000
14	--------	--------	--------	~9000

Fig. 1: DNA genome hepatocytes of albino rats. Lane M is the DNA ladder (250 – 10,000 bp). Lane 1 represents the DNA genome of normal group and lanes 2 - 4 represent the three groups of exposed albino rats (10, 15 and 20 pulsed/day, respectively).

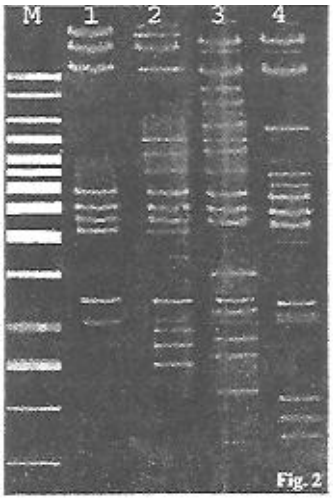

Fig. 2: Representive RFLPs patterns from the unexposed rats and three exposed groups with *Pca*I restriction endonuclease.

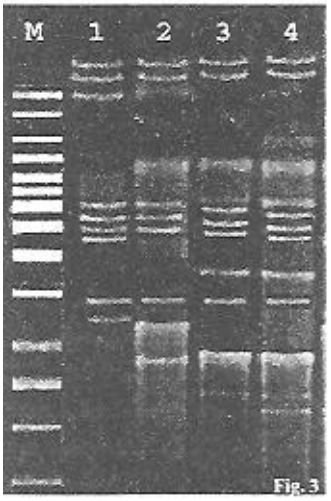

Fig. 3: Representive RFLPs patterns from the unexposed rats and three exposed groups with *Sfi*I restriction endonuclease.

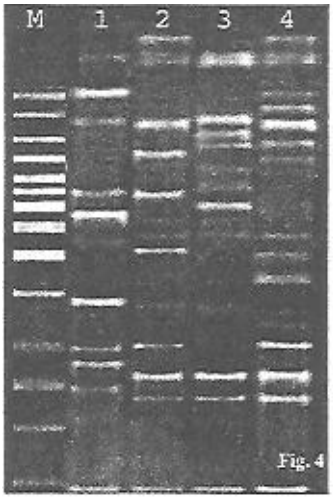

Fig. 4: Representive RFLPs patterns from the unexposed rats and three exposed groups with *Not*I restriction endonuclease.

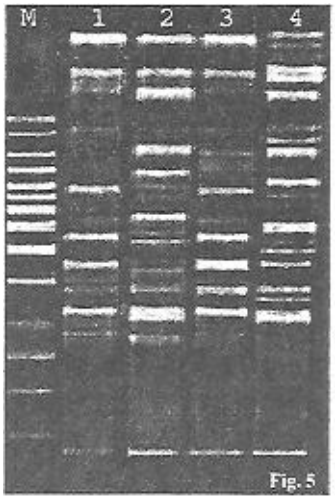

Fig. 5: Representive RFLPs patterns from the unexposed rats and three exposed groups with *Swa*I restriction endonuclease.

الملخص العربى

دراسات تشخيصية جزيئية وبيوكيميائية على تأثير التعرض لأطياف الموجات الكهرومغناطيسية النبضية على الفئران

إبتسام عبدالمجيد مرزوق- محمد حسين عواد*- جمال جابرمصطفىوناهد محمد الأبيض**

قسم التطبيقات البيولوجية- مركز البحوث النووية- هيئة الطاقة الذرية- *قسم علم الحيوان- كلية العلوم- جامعة بنها- **قسم البلاوما- مركز البحوث النووية- هيئة الطاقة الذرية

أكدت العديد من الدراسات خلال العشرون عاما الماضية على وجود علاقة بين التعرض للمجال الكهرومغناطيسى والعديد من المشاكل الصحية الخطيرة والناتجة عن أنظمة التوليد والتوزيع الكهربائى وكل الأجهزة التى تنتج تيارا كهربائيا وذلك فى المنازل والمصانع وأماكن العمل والتى تحدث تأثيرا على الخلايا والأعضاء مسببة بعض التغييرات البيولوجية، ولذلك كان الهدف من الدراسة الحالية هو الآتى:

١- معرفة مدى تأثير المجال الكهرومغناطيسى النبضى على بعض المقاييس الكيميائية الحيوية.

٢-دراسة مدى تأثير المجال الكهرومغناطيسى النبضى على الحمض النووى الديوكسى ريبوزى.

استخدمت الجرعة من المجال الكهرومغناطيسى المتقطع بتردد ٨-١٢هرتز وذلك باستخدام جهاز البلازما فوكس لتقييم تأثير التعرض على بعض القياسات فى ذكور الفئران، وعرضت ثلاث مجاميع من الفئران إلى ١٥،١٠ و ٢٠ نبضة فى اليوم ثلاث مرات اسبوعيا ولمدة ثلاث اسابيع بالإضافة إلى المجموعة الضابطة التى لم تتعرض للمجال الكهرومغناطيسى، وأسفرت النتائج عن التالى:

أن التعرض للمجال الكهرومغناطيسى يسبب زيادة ذات قيمة إحصائية فى مستوى البروتين الكلى والجلوبيولين والجلسريدات الثلاثية وهرمون الذكورة وذلك فى المجموعة الثانية المعرضة فقط بينما لم يتأثر الألبيومينوالكوليسرول فى نفس المجموعة وحدث نقص ذو دلالة إحصائية فى نسبة الألبيومين إلى الجلوبيولين فى المجموعة الثانية المعرضة مقارنة بالمجموعة الضابطة الغير معرضة ولم يحدث أى تغير فى المجموعتين الأولى والثالثة المعرضة مقارنة بالمجموعة الضابطة الغير معرضة وذلك بالنسبة لكل من البروتين الكلى والألبيومين والجلوبيولين والكوليستيرول والجلسريدات الثلاثية وهرمون الذكورة.

وقد كان هناك فرضية دلالية عل تأثير التعرض لأطياف الموجات الكهرومغناطيسية النبضية على كبد الفئران وذلك اعتمادا على تحليل الحمض النووى الديوكسى ريبوزى باستخدام انزيمات القصر (*Pac*I, *Sfi*I, *Not*I, *Swa*I).

وقد دلت النتائج عن عملية تشخيصية جيدة وعمل خريطة للتأثير الخطير للموجات الكهرومغناطيسية النبضية على الحمض النووى بالإضافة للتأثير الكيميائى الحيوى.