IRAQI Academic Scientific Journals
العراقية المجلات الأكاديمية العلمية
University of Kut Journal
ISSN (E): 2616 - 7808 II ISSN (P): 2414 - 7419
www.kutcollegejournal.alkutcollege.edu.iq
k.u.c.j.sci@alkutcollege.edu.iq
Special Issue for the Researches of the 6th Int. Sci. Conf. for Creativity for 16 -17 April 2025

Assessment of DNA damage in rats male exposure to electromagnetic radiation using comet assay

Kawther Neamah Najm [1] , Alyaa Neamah Najm Alsaedi [2] , Yasemin Khudiar Alghanimi [3]

Abstract

INTRODUCTION: The purpose of the current investigation was to find out the consequences of prolonged use of mobile phone radiation at the DNA level decomposition of rats.

METHODS: Sixty males were split up into three groups at random, each group contained (15) rats, each group exposed to the radiation from mobile phones for two months, three and six months, respectively, T1, the initial group, was exposed to a mobile phone. rays for three periods and two hours. (6-3-2) months For four hours over the course of three periods (6-3-2) months, the second group (T2) was exposed to mobile phone radiation.and the third group (T3) was exposed to mobile phone rays for a while Three times a day for eight hours (6-3-2) months, while the other groups were counted as a control group that was not exposed to mobile radiation. The Following the conclusion of the experiment's designated time frame, blood samples were taken from each of the three groups.

RESULTS: The level of DNA decomposition was measured, and the damage in DNA was evaluated using the Score Comet Assay technique. The DNA criteria adopted for determining the percentage of damage were DNA tail length, tail moment, and tail percentage.

CONCLUSION: The findings indicated that the final group T3 had the highest effect ratio for the aforementioned criteria, (63) (3), 44 (8), (1.74) respectively, and the mentioned values represent asignificant increase compared to the control group.

Keywords: DNA damage, rats male, electromagnetic radiation, comet assay

Affiliation of Authors

[1, 2] Kerbala University، Iraq, Kerbala, 56001

[3] College of Education for Pure Sciences, Kerbala University, Iraq, Kerbala, 56001

[1] kawther.n@s.uokerbala.edu.iq
[2] alyaa.neamah@s.uokerbala.edu.iq
[3]Yasemin.khelef@uokerbala.edu.iq

[1] Corresponding Author

Paper Info.
Published: Oct. 2025

تقييم تلف الحمض النووي لدى ذكور الفئران المعرضة للإشعاع الكهرومغناطيسي باستخدام اختبار المذنب

كوثر نعمه نجم [1] ، علياء نعمه نجم [2] ، ياسمين خضير خلف [3]

المستخلص

هدفت هذه الدراسة إلى معرفة آثار الاستخدام المطول لإشعاعات الهاتف المحمول على مستوى تحلل الحمض النووي لدى الفئران.

قُسّم ستون ذكرًا إلى ثلاث مجموعات عشوائيًا، ضمت كل مجموعة (15) فأرًا، وتعرضت كل مجموعة لإشعاعات الهواتف المحمولة لمدة شهرين، وثلاثة، وستة أشهر على التوالي. المجموعة الأولى (T1) تعرضت لأشعة الهاتف المحمول لمدة ثلاث فترات وساعتين (6-3-2) شهرًا، بينما تعرضت المجموعة الثانية (T2)لأشعة الهاتف المحمول لمدة أربع ساعات على مدار ثلاث فترات (6-3-2) شهرًا، والمجموعة الثالثة (T3)لأشعة الهاتف المحمول لمدة ثلاث مرات يوميًا لمدة ثماني ساعات (6-3-2) شهرًا، بينما اعتُبرت المجموعات الأخرى مجموعة ضابطة لم تتعرض لإشعاعات الهاتف المحمول. بعد انتهاء الفترة الزمنية المحددة للتجربة، أُخذت عينات دم من كل مجموعة من المجموعات الثلاث. النتائج: تم قياس مستوى تحلل الحمض النووي، وتقييم التلف فيه باستخدام تقنية فحص المذنبات السكّرية. وكانت معايير الحمض النووي المُعتمدة لتحديد نسبة التلف هي طول ذيل الحمض النووي، وعزم الذيل، ونسبة الذيل.

الخلاصة: أشارت النتائج إلى أن المجموعة النهائية T3 سجلت أعلى نسبة تأثير للمعايير المذكورة، (63) (3)، 44 (8)، (1.74) على التوالي، وتمثل القيم المذكورة زيادة معنوية مقارنةً بالمجموعة الضابطة.

الكلمات المفتاحية: تلف الحمض النووي، ذكور الجرذان، الإشعاع الكهرومغناطيسي، فحص المذنبات

انتساب الباحثين

[1، 2] جامعة كربلاء، العراق، كربلاء، 56001

[3] كلية التربية للعلوم الصرفة، جامعة كربلاء، العراق، كربلاء، 56001

[1] kawther.n@s.uokerbala.edu.iq
[2] alyaa.neamah@s.uokerbala.edu.iq
[3]Yasemin.khelef@uokerbala.edu.iq

[1] المؤلف المراسل

معلومات البحث

تأريخ النشر: تشرين الاول 2025

Introduction

Some studies report effects on DNA at exposure levels close to indicative limits, but there is little agreement between studies, and the significance of the observed effects remains unclear [1]. Moreover, several studies have shown that the biological effects of magnetic fields Low frequency can penetrate deep tissue. [2].

[3] showed that mobile phone radiation may have an effect on many cellular functions such as cell proliferation and differentiation [3] and many other researchers followed them, such as [4] who showed the effect of electromagnetic radiation on programmed cell death. In another study, it showed an effect on cell death. DNA synthesis [5] there are also reports on the genetic effects of electromagnetic radiation [6; 7]. In addition to the rise in DNA breaks [8; 9; 10], electromagnetic radiation also shows that chromosomal damage is caused by [11; 12]

There are conflicting accounts in the literature about how RF-EMW affects, mitochondrial, dwarfing pathway, heat shock proteins, free metabolism, cell differentiation, and DNA damage [13]. There have been various studies examining the effects of electromagnetic radiation on DNA damage in the last decade and dealing with DNA breakage and apoptosis [14] noted a rise in unmarried and multiple DNA fragments in rat cells in the brain. They also found that exposure to electromagnetic radiation caused crosslinks in enhanced apoptosis and DNA-protein and DNA-DNA interactions in biological samples from mice [15]. [16] revealed that the growing brain cells of rats have more DNA of a single chain gene. identified 35 days following 2.45 and 16.5 GHz exposure. A low increase in the amount of tight DNA was also recorded in the double strand of mouse embryos following acute exposure to 1.7 GHz [16] Nevertheless, the findings of more recent research are about the impacts of electromagnetic radiation on the DNA[17] where Tice [2002] reported that exposure of human Leukocytes and lymphocytes exposed to RF-EMW for 24 hours at a SAR rate of 5–10 W/kg because of chromosomal damage [18] revealed that exposure to 900 MHz, but not 1800 MHz, altered the expression of several genes in human endothelial cells. while a study showed that exposure to 1950MHz of electromagnetic radiation for 24 hours induces in vitro genotoxicity in cells. Human fibroblasts but not in lymphocytes [8] In a study using rat granulosa cells and human fibroblasts exposed to cell phone signals [1800 MHz during 4, 16, and 24 hours], [9] reported the presence of DNA that is single and double-stranded molecules within these cells [8]. Recent studies by [17] on human fibroblasts and Cell-T cells, respectively did not exhibit any noteworthy toxicological effects of EMR and therefore, [17 and 18] DNA damage may depend on cell type in addition to (Exposure duration, RF-EMW frequency, specific absorption, etc.). A recent study by [19] showed increased genetic damage from exposure to radiation in many cases. However, the dosimetry mechanism was deficient in studies [19].

Implicit DNA damage in cells may be significant. It is usually cumulative. DNA is able to repair itself through the homology mechanism. The delicate balance between DNA damage and repair is maintained by cells. Most cells are able to repair single-strand DNA. However, it is known that double strand DNA breakage, if not repaired properly, will result in apoptosis, or cell death [20]. The impact of electromagnetic radiation on Apoptosis is also debatable. [21]. as previously discussed, and research indicates that

electromagnetic radiation may affect the plasma membrane accessory receptors [22; 23] However, the Along with the kind and length of radiation exposure, the kind of cell may also affect the induction of apoptosis.

Items of Research

EXPERIMENTAL ANIMALS

In this experiment, (60) white male rats obtained from the laboratory of the College of Pharmacy - University of Karbala were used, with an average age ranging between (12-10) weeks and weights ranging between (190-240) g. The animals were raised outside the college and placed in breeding cages under Thermal conditions at a rate of 25 m, 12 hours of illumination per day, good ventilation, fed on a special ration, water, and the animals were left to acclimatize for a week.

EXPERIMENTAL DESIGN

The experiment's laboratory animals were equally divided into 12 groups at random, with 20 rats in each group. The totals were as follows:

THE FIRST EXPERIMENT

The first group, which numbered (5), was left without exposure to radiation doses and considered as a control group. The second group of 5 were subjected to two hours of phone radiation. and for a period of two months the third group, which numbered 5, were subjected to two hours of phone radiation. and for a period of three months the fourth group, which numbered 5, were subjected to two hours of phone radiation. and for a period of six months.

Second experiment:

The first group, which numbered 5, was left without radiation doses and considered as a control group.

The second group, which numbered 5, were subjected to two hours of phone radiation. and for a period of two months.

The third group, which numbered 5, were exposed to phone rays for four hours and for a period of three months.

The fourth group, which numbered 5, were exposed to phone rays for four hours and for a period of six months.

The third experiment:

The first group, which numbered 5, was left without radiation doses and considered as a control group

The second group of 5 were subjected to phone radiation for eight hours over the course of two months. The third group, which numbered 5, was eight hours of exposure to phone radiation and for a period of three months.

The fourth group, which numbered 5, was revealed to radiation from telephones for eight hours and for a while of six months.

Irradiation Animals

The animals were irradiated using electromagnetic radiation (EMR) emitted from a Korean-made Samsung S3 mobile phone. Rats were irradiated daily at a time of two hours for two, three and six months, respectively, for the first experiment, treated T1 at a time of four hours, for two months, three and six months, respectively, for the second experiment, treated with T2. Eight for two, three and six months, respectively, for the third experiment, treatment T3, noting that the rats were placed in cages, and each cage contained (5) rats.

Obtaining blood samples

Chloroform, an anesthetic, was administered to the animals by putting a cotton container on the

anesthetic material in a sizable box where the rat was placed to be put to sleep by breathing, and then blood (5 ml) was extracted straight from the heart by puncturing it in order to extract the most blood possible, 3 ml were determined for physiological tests and the rest for genetic tests. In order to prevent blood clotting during genetic testing, blood samples were placed in tubes containing EDTA, an anticoagulant. In contrast, blood samples used for biochemical testing were placed in test tubes devoid of any anticoagulant. The blood serum was separated by centrifuging it for 15 minutes at 4000 rpm, and the sera were then stored in a refrigerator at a temperature of (4°C) to complete the biochemical measurements.

3- Comet Assay

1- Prepare the infusion solution and cool it at 4°C for 20 minutes before use.
2- Dissolve the gel in a beaker of boiling water for 5 minutes, then place it in a water bath at 37°C 20 minutes before work.
3- The cells were mixed at a concentration of 〖1×10〗^5 with the soluble gel at 37°C at a ratio of 1:10 (vol/vol) and pulled directly by pipette into the comet slide and if necessary, the side space of the plastic nozzle of the pipette was used to spread the gel and cells over an area. The sample for the slide to make sure that all the sample area is covered and if it is not evenly distributed, warm the slide at 37 degrees Celsius before completing the application.
4- If working with several samples, the gel should be divided into vials or tubes at 37°C, cells added, gently stirred, and 50 µl spread over the sample area. Samples are placed on a flat and orderly surface at 4°C in the refrigerator for 10 minutes. A clear drop of 0.55 mm in diameter will appear at the edge of the specified area of the sample. Increasing the crystallization time to 30 minutes increases the adhesion of samples in the case of high humidity.
5- The samples were immersed in the lysis solution at 4°C for 30-60 minutes. In order to increase the sensitivity of the test, the incubation period can be continued at the same temperature for 12 hours.
6- The excess solution must be removed from the sample and immersed in the anti-wrinkle base solution, provided that it is prepared immediately before use.
7- The immersion in the previous solution continues either 20 minutes at room temperature or 1 hour in the dark at 4 °C.
8- To perform the comet test, a ml of the base relay solution is added at 4°C, then the sample is transferred to the electrical relay and covered with the special cap with the device set to 21 volts for 30 minutes.
9- The excess electrolyte solution is removed gently and the sample is immersed in dH2O for five minutes, the process is repeated twice, and then immersed in 70% alcohol solution for five minutes.
10- The model is dried at 37°C for 10-15 minutes and the drying works to make the cells in one level, which facilitates the work of monitoring them. Then the samples are stored at room temperature with pre-drying to make the measurements in the stage.
11- Put 100 ml of SYBR Green dye in a dry acoustic circle for 30 minutes at room temperature in the dark, then lift the form gently to remove the excess dye and rinse in

water for a short time, then allow the form to dry completely at 37 ° C.

12- The model is placed in a fluorescence microscope, as the flash filter is sufficient to perform the test. Fifty different measurements can be made in this test, measured from the ratio L/W, the fluorescence index, and the range 1.2-2.0 indicates that the level of damage is low LD (DNA damage).

RESULTS

The effect of mobile phone radiation on DNA damage

The results of the current study showed in Table (1) a noteworthy rise ($P < 0.05$) in DNA damage in lymphocytes, in contrast to the group of healthy controls of T1, T2, T3 groups for the time periods (6-3-2) months for (8-) 4-2 hours in a row in groups exposed to the radiation from mobile phones, and the highest percentage of impact was in the last group T3 with a period of six months and eight to four and two hours, respectively.

Table 1:Effect of mobile phone radiation on DNA damage in male albino rats

Parameters / Groups	(%) Tail DNA (mean±SD)			LSD	P-value
Control	2 months	3 months	6 months		
	A,a 1.44±0.031	A,a 1.44±0.031	A,a 1.44±0.031		
T1 2 hrs	B,a 15.58±2.17	B,a 15.94±2.82	B,b 18.44±1.96	2.16	**0.0058 Sig.**
T2 4 hrs	B,a 17.37±3.05	C,a 18.37±1.95	C,b 24.33±2.50	3.47	**0.0073 Sig.**
T3 8 hrs	C,a 29.41±4.29	D,ab 33.41±4.16	D,b 36.49±5.02	4.59	**0.0047 Sig.**
LSD	3.68	2.15	3.63		
P-value	0.0106 Sig.	0.0090 Sig.	0.0064 Sig.		

Average ± standard error, n = 5

Significant differences at the probability level ($P < 0.05$) are indicated by different capital letters in the vertical direction.

T1 = represents the group of animals exposed to radiation from phones for two months.

T2 = represents the animals exposed to radiation from cell phones for three months.

T3 = represents animals exposed to radiation from cell phones for six months.

The impact of mobile phones radiation on the length of the comet in DNA

The results of the current study showed in Table (2) a noteworthy rise ($P < 0.05$) in the causal

DNA's length in lymphocytes, compared with the healthy control group for T1, T2, T3 groups in the time periods (6-3-2) months for (8-4-2 hours in a row in groups exposed to the radiation from cell phones, and the highest percentage of impact was in the last group 3 T with a period of six months and eight to four and two hours, respectively.

Table 2: Effect of mobile phone radiation on the length of the comet of DNA in male albino rat

Parameters / **Groups**	**Tail length(px) (mean±SD)** 2 months	3 months	6 months	**LSD**	**P-value**
Control	A,a 0.844 ±0.026	A,a 0.844 ±0.026	A,a 0.844 ±0.026		
T1 2 hrs	B,a 32.75±4.62	B,a 34.51±5.07	B,b 45.26±4.73	4.37	**0.0083 Sig.**
T2 4 hrs	C,a 51.36±6.41	C,a 54.52±6.68	C,b 62.54±7.55	5.04	**0.0095 Sig.**
T3 8 hrs	D,a 68.44±7.05	D,b 74.36±9.05	D,c 89.44±10.72	4.71	**0.0104 Sig.**
LSD	6.15	5.92	8.44		
P-value	0.0073 Sig.	0.0038 Sig.	0.0061 Sig.		

Average ± standard error, n = 5

Different capital letters in the vertical at the probability level (P < 0.05), direction indicates the existence of significant differences.

T1 = represents the group of animals subjected to the radiation from mobile phones for two months.

T2 = represents Rats in the group exposed to radiation from mobile phones for three months.

T3 = represents Rats in the group exposed to radiation from mobile phones for six months.

The effect of mobile phone radiation on the average appearance of DNA guilt

The results of the current study in Table (3) showed a significant (P < 0.05) increase in the average appearance of tail DNA in lymphocytes compared with the healthy control group for T 1, T 2, T 3 groups in the time period (3-6-). 2) Months for (8-4-2) hours in a row in groups exposed to the radiation from phones, and the highest percentage of impact was in the last group T3 with a period of six months and eight to four and two hours, respectively.

Table 3: Effect of mobile phone radiation on the average appearance of tail DNA for male white rats

Parameters / **Groups**	**Tail mean moment (mean±SD)** **2 months**	**3 months**	**6 months**	**LSD**	**P-value**
	A,a	A,a	A,a		

Control	0.048±0.006	0.048±0.006	0.048±0.006		
T1 4 hrs	B,a 2.074±0.032	B,a 2.14±0.81	B,a 2.29±0.068	**2.16**	**0.311 Non Sig.**
T2 4 hrs	C,a 4.61±1.94	C,b 6.38±1.73	C,b 7.033±2.104	**3.47**	**0.0061 Sig.**
T3 8 hrs	D,a 8.218±2.18	D,a 9.437±2.16	D,b 12.25±3.27	**4.59**	**0.0019 Sig.**
LSD	**1.53**	**1.72**	**1.74**		
P-value	**0.0094 Sig.**	**0.0085 Sig.**	**0.0052 Sig.**		

Average ± standard error, n = 5

Different capital letters in the vertical at the probability level (P < 0.05), direction indicates the existence of significant differences.

T1 = represents the group of animals exposed to radiation from phones for two months.

T2 = represents Rats in the group exposed to radiation from mobile phones for a period of three months.

T3 = represents Rats in the group exposed to radiation from mobile phones for six months. Table (4) showed.

Table 4: normal non-breaking DNA

Parameters / **Groups**	**No damage % (mean±SD)** 2 months	3 months	6 months	**LSD**	**P-value**
Control	A,a 48.197±6.765	A,a 48.197±6.765	A,a 48.197±6.765		
T1 4 hrs	B,a 42.302±4.555	B,a 41.929±5.053	B,a 39.787±3.619	2.84	**0.371 Non Sig.**
T2 4 hrs	C,a 27.529±3.057	C,b 21.763±3.530	B,b 21.963±2.963	3.46	**0.0148 Sig.**
T3 8 hrs	C,a 29.063±3.746	C,ab 26.638±2.213	B,b 24.914±2.576	3.58	**0.0106 Sig.**
LSD	5.337	6.528	5.039		
P-value	0.0031 Sign.	0.0027 Sign.	0.0076 Sign.		

Average ± standard error, n = 5

Different capital letters in the vertical direction at the probability level (P < 0.05), show that there are significant differences.

T1 = represents the group of animals exposed to radiation from phones for two months.

T2 = represents Rats in the group exposed to radiation from mobile phones for three months.

T3 = represents Rats in the group exposed to radiation from mobile phones for six months.Table (5) showed:

Table 5: Little breakage in DNA

Parameters / Groups	Low damage % (mean±SD) 2 months	3 months	6 months	LSD	P-value
Control	A,a 33.717±5.047	A,a 33.717±5.047	A,a 33.717±5.047		
T1 2hrs	A,a 32.773±4.235	A,a 31.483±3.095	A,a 30.500±4.030	**2.87**	**0.285 Non Sig.**
T2 4 hrs	B,ab 16.377±2.205	B,a 18.117±2.995	B,b 14.360±1.360	**2.66**	**0.0095 Sig.**
T3 8 hrs	B,a 17.590±1.430	B,a 18.300±1.680	B,a 19.103±2.475	**3.06**	**0.318 Non Sig.**
LSD	**3.940**	**4.366**	**5.327**		
P-value	**0.0062**	**0.0081**	**0..0075**		

Average ± standard error, n = 5

Different capital letters in the vertical direction show that there are differences that are significant at the probability level ($P < 0.05$).

T1 = represents the group of animals exposed to radiation from phone for two months.

T2 = represents Rats in the group exposed to radiation from mobile phones for three months.

T3 = represents Rats in the group exposed to radiation from mobile phones for six months. Table (6) showed

Table 6: average breakage in DNA

Parameters / Groups	Medium damage % (mean±SD) 2 months	3 months	6 months	LSD	P-value
Control	A,a 8.855±0.565	A,a 8.855±0.565	A,a 8.855±0.565		
T1 2hrs	B,a 12.660±0.039	B,a 13.148±0.010	B,a 14.656±1.375	**2.90**	**0.257 Non Sig.**
T2 4 hrs	C,a 29.183±1.161	C,a 30.626±1.858	C,a 29.804±2.196	**3.11**	**0.408 Non Sig.**
T3 8 hrs	D,a 20.092±0.395	D,a 20.807±0.437	D,a 19.837±0.261	**3.07**	**0.396 Non Sig.**
LSD	**2.56**	**3.35**	**3.28**		
P-value	**0.0088 Sig.**	**0.0095 Sig.**	**0.0117 Sig.**		

Average ± standard error, n = 5

Different capital letters in the vertical direction show that there are differences that are significant at the probability level ($P < 0.05$).

T1 = represents the group of animals exposed to radiation from phones for two months.

T2 = represents Rats in the group exposed to radiation from mobile phones for three months.

T3 = represents Rats in the group exposed to radiation from mobile phones for six months. Table (7) showed

Table 7: High breakage in DNA

Parameters / Groups	High damage % (mean±SD) 2 months	3 months	6 months	LSD	P-value
Control	A,a 9.231±1.885	A,a 9.231±1.885	A,a 9.231±1.885		
T1 2hrs	B,a 12.263±0.358	B,ab 13.440±1.160	B,b 15.055±0.212	2.74	**0.0173 Sig.**
T2 4 hrs	C,a 26.910±0.013	C,ab 29.497±3.385	C,b 33.871±0.129	3.85	**0.0096 Sig.**
T3 8 hrs	C,a 33.252±0.081	D,ab 34.260±0.460	C,b 36.146±0.362	2.16	**0.0089 Sig.**
LSD	2.71	3.05	4.12		
P-value	0.0096 Sign.	0.0122 Sign.	0.0085 Sign.		

Average ± standard error, n = 5

Different capital letters in the vertical direction show that there are differences that are significant at the probability level ($P < 0.05$).

T1 = represents the group of animals exposed to radiation from phones for two months.

T2 = represents Rats in the group exposed to radiation from mobile phones for three months.

T3 = represents Rats in the group exposed to radiation from mobile phones for six months.

Here are four Figure that illustrate DNA(1,2,3,4)

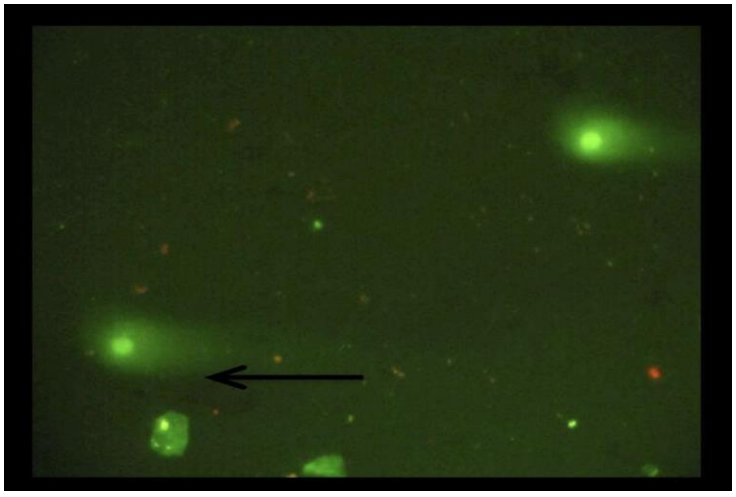

Figure (1) shows normal, unbreakable DNA

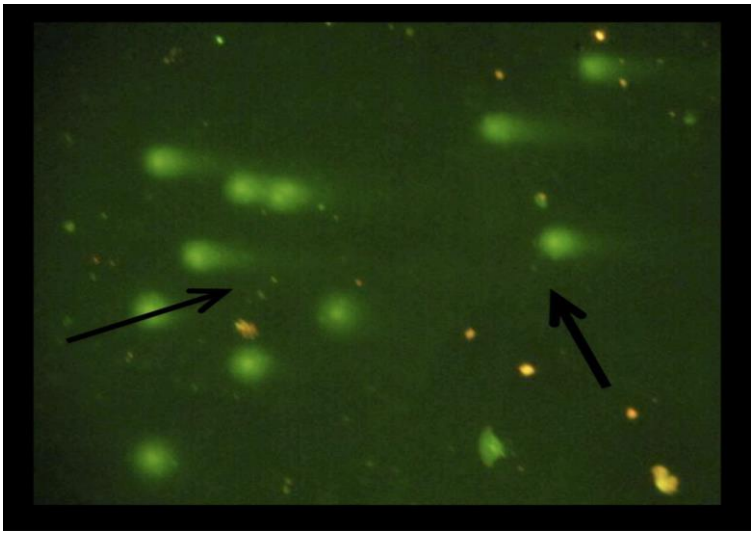

Figure (2) shows a small break in DNA

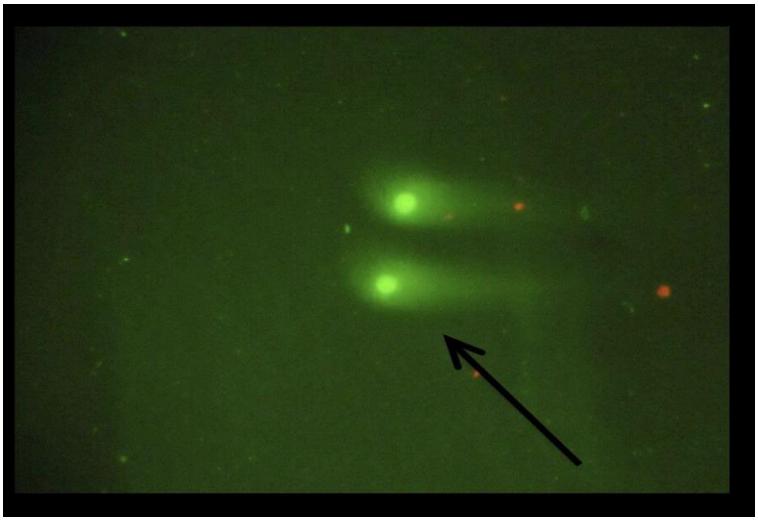

Figure (3) shows a moderate break in DNA

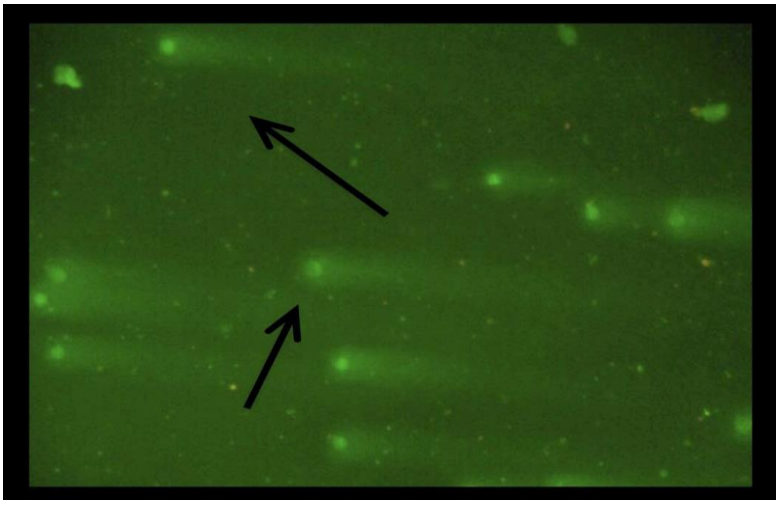

Figure (4) shows a high breakdown in DNA

The results of the current study showed that exposure to mobile phone radiation caused a notable rise in the percentage of DNA degradation in male albino rats' blood, and this study was in agreement with [24] And the study, which was collected from medical professionals, showed that because cell phones emit radio waves, they have a negative impact on people's health. Our DNA is destroyed by these radiations as they enter our bodies. [25]. Cell phones emit electromagnetic fields which are another form of non-ionizing radiation in our environmental surroundings. These studies have indicated that exposure to electromagnetic radiation leads to DNA damage [26; 27] In addition, two studies [28; 29] on electromagnetic radiation's impacts on the mechanisms of DNA repair, free radicals, interaction with transition metals (such as iron) and how damage occurs [30; 31].

It showed that DNA damage occurred in different types of cells after exposure to cell phone frequency fields. [9] Exposed human fibroblasts and granulosa cells. the comet COMET ASSAY method was used to detect different types of DNA, such as double and single strand breaks of DNA, sites of alkaline impurities, Cross-links, unfinished repair sites, and single-cell repair. This method has been Used by several researchers to track DNA defects and quantify DNA by measuring the exchanges between the nucleus and tail's genetic material, leading to noticeably greater genomic damage in healthy individuals without a history of of exposure (past or representative). [32]. Where a study revealed that the locations' DNA levels had increased near the mobile phone station compared to the control group. This significantly increased in the length of the larger DNA tail involves genomic damage in white blood cells [33]. Differences in electromagnetic radiation frequencies are almost certainly to blame for genetic harm. Several studies have also revealed that microwave radiation causes interference in DNA resulting in more double tension in the DNA strands) [34] (RF-induced genotoxicity is persistent, and in particular causes Chromosome instability [35], altered gene expression [36], genetic mutations [37] and DNA structure breaks [20]. the non-ionizing energy electromagnetic radiation is insufficient to directly break the chemical bonds of DNA [3], however, it can act through direct directing mechanisms, thus leading to the generation of radicals [38]. which are powerful, inert chemicals that are essential to cells [20], where free radicals have many effects by inducing mutagenic responses depending on the concentration, duration of exposure and cell type [39] Significant in the level of DNA in lymphocytes [40] Oxidative stress plays an important role in DNA damage, general and specific gene expression and programmed cell death [41]. The effects of electromagnetic radiation depend on its characteristics such as frequency, intensity and duration of exposure. Both internal and external forces continuously damage DNA, which is subsequently restored by DNA repair enzymes. DNA damage and/or faulty repair can lead to an accumulation of DNA neutralizers that can eventually lead to changes in cellular function, cell death or cancer [42; 43].

Damage can be in the form of single and double rope breaks. The genotoxic effects of exposure to electromagnetic radiation for 30 and 60 days have been studied in vitro using the most widely used method known as the comet test, showing that In rats' brains, modest levels of electromagnetic radiation can cause DNA damage [24; 44] He also said that being exposed to low-intensity electromagnetic radiation for 30 days is able to

interact with DNA and cause changes in it [42]. They attribute DNA damage to oxidative stress through ROS, or reactive oxygen species [45]. ROS could contribute to the mechanism of the biological effects of electromagnetic radiation [21]. So one of the possibilities for how DNA damage occurs is that free radicals that develop inside cells harm DNA. Cell damage is the effect of free radicals. Big compounds like proteins and DNA, and lipid membranes. Several studies have revealed that electromagnetic radiation enhances the activity of free radicals in cells [46;47]. Especially by the Fenton reaction is an iron-catalyzed process whereby hydroxyl free radicals, which are extremely powerful and cytotoxic, are created from hydrogen peroxide, a byproduct of oxidized oxygen in mitochondria [20].

Conclusions

The comet test is an application of genetics in genotoxicity. It is a simple method for measuring double-stranded DNA degradation in eukaryotic cells. This test is widely used to evaluate the effects of chemicals and radiation on DNA and its repair mechanisms, as well as to analyze the impact of environmental factors on it and thus determine its genotoxicity. It is the easiest way to detect DNA damage. DNA damage appears as a tail in a microscopic image of a cell. This tail consists of broken DNA fragments far from the nucleus, hence the name of the test. The results showed an increase in DNA damage with increased exposure to mobile phone radiation, representing a significant increase compared to the control group.

References

[1] electromagnetic fields (EMF) on human health-opinion of the scientific committee on emerging and newly identified health risks (SCENIHR). J. Toxicology. 246(2-3): pp. 248-250.

[2] Marino A, Becker R. (1977/9) Biological effects of extremely low frequency electric and magnetic fields: a review. Physiological Chemistry and Physics (2), 131-147.

[3] Foletti A, Lisi A, Ledda M et al. (2009). Cellular ELF signals as a possible tool in informative medicine. Electromagnetic Biology and Medicine. 28 (1), 71-79.

[4] Tian F, Nakahara T, Yoshida M, Honda N, Hirose H, Miyakoshi J. (2002). Exposure topower frequency magnetic fields suppresses X-ray-induced apoptosis transiently inKu80-deficient xrs5cells. Biocemical and Biophysical Research Communications 292(2), 355-361.

[5] Takahashi K, Kaneko I, Date M, Fukada E. (1986). Effect of pulsing electromagnetic fields on DNA syntesis in mammalian cells in culture. Cellular and Molecular Life Sciences 42 (2), 185-186.

[6] Lai H 2012. Genetic Effects of Non-Ionizing Electromagnetic Fields. BioInitiative Working Group, Section.

[7] Lai H 2012. Genetic Effects of Non-Ionizing Electromagnetic Fields. BioInitiative Working Group, Section.

[8] Singh S, Kapoor N 2014. Health implications of electromagnetic fields, mechanisms of action, and research needs. Advances in Biology, 2014: 1-24.

[9] Diem E, Schwarz C, Adlkofer F, Jahn O, Rudiger H: Non-thermal DNA breakage by mobile-phone radiation (1800 MHz) in human

fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro. Mutat Res 2005, 583(2):178-183

[10] Franzellitti S, Valbonesi P, Ciancaglini N, Biondi C، Contin A, Bersani F, Fabbri E 2010. Transient DNA damage induced by high-frequency electromagnetic fields (GSM 1.8 GHz) in the human trophoblast HTR-8/SVneo cell line evaluated with the alkaline.

[11] Mihai CT, Rotinberg P, Brinza F, Vochita G 2014. Extremely low-frequency electromagnetic fields cause DNA strand breaks in normal cells. J Environ Health Sci Eng, 12: 15.

[12] Maes A, Gorp UV, Verschaeve L 2006. Cytogenetic investigation of subjects professionally exposed to radiofrequency radiation. Mutagenesis, 21: 139–142.

[13] Winker R, Ivancsits S, Pilger A, Adlkofer F, Rüdiger HW 2005. Chromosomal damage in human diploid fibroblasts by intermittent exposure to extremely low-frequency electromagnetic fields. Mutat Res.49-43 :585 .

[14] Blank M. Do electromagnetic fields interact with electrons in the Na, K-ATPase?[J]. Bioelectromagnetics , 2005, 26 :677–83. doi:10.1002/(ISSN)1521-186X.

[15] Belyaev I. (2005). Nonthermal biological effects of microwaves: current knowledge، further perspective, and urgent needs. Electromagnetic Biology and Medicine .403-24,375.

[16] Behari J, Paulraj R: Biomarkers of induced electromagnetic field and cancer. Indian J Exp Biol 2007, 45(1):77-85. Marino A, Becker R. (1977/9) Biological effects of extremely low frequency electric and magnetic fields: a review. Physiological Chemistry and Physics (2), 131-147.

[17] Sannino A, Di Costanzo G, Brescia F, Sarti M, Zeni O, Juutilainen J، Scarfi MR: Human fibroblasts and 900 MHz radiofrequency radiation: evaluation of DNA damage after exposure and coexposure to 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5 h-(furanone (MX). Radiat Res 2009, 171(6):743-751.

[18] Aitken, R. J. and Baker, M. A. (2004). Oxidative stress and malereproductive biology .Vertebrata Reproductive science and technology., 16(5):581-588.

[19] Remondini D, Nylund R, Reivinen J, et al. Gene expression changes in human cells after exposure to mobile phone microwaves. Proteomic. 4754–6:4745؛2006.

[20] Lai H, Singh NP 2004. Magnetic-field-induced DNA strand breaks in brain cells of the rat. Environ Health Perspect, 112: 687-694.

[21] Nisarg R Desai1,2, Kavindra K Kesari3 and Ashok Agarwal*1Pathophysiology of cell phone radiation: oxidative stress and carcinogenesis with focus on male reproductive system

a. Reproductive Biology and Endocrinology 2009,

[22] Zhao TY, Zou SP, Knapp PE: Exposure to cell phone radiation up-regulates apoptosis genes in primary cultures of neurons and astrocytes. Neurosci Lett 2007, 412(1):34-38.

[23] Capri M, Scarcella E, Fumelli C, Bianchi E, Salvioli S, Mesirca P, Agostini C, Antolini A, Schiavoni A, Castellani G, Bersani F, Franceschi C: In vitro exposure of human lymphocytes to 900 MHz CW and GSM modulated radiofrequency: studies of proliferation، apoptosis and mitochondrial

membrane potential. Radiat Res 2004, 162(2):211-218.

[24] Deshmukh PS, Megha K, Banerjee BD, et al. Detection of low level microwave radiation induced DNA damage vis-a-vis genotoxicity in brain of Fischer rats[J]. Toxicol Int , 2013, 20 :19–24. doi:10.4103/0971-6580.111549.

[25] Keller, A., Rackwitz, J., Cauët, E., Liévin, J., Körzdörfer, T., Rotaru, A., ...& Bald, I. (2014). Sequence dependence of electron-induced DNA strand breakage revealed by DNA nanoarrays. Scientific reports, 4.

[26] Vijayalaxmi, B.Z. Leal, M. Szilagyi, T.J. Prihoda, M.L. Meltz, Primary DNA damage in human blood lymphocytes exposed in vitro to 2450MHz radiofrequency radiation, Radiat. Res. 153 (2000) 486–479.

[27] M. Zmyslony, J. Palus, J. Jajte, E. Dziubaltowska, E. Rajkowska، DNA damage in rat lymphocytes treated in vitro with iron cations and exposed to 7 mT magnetic fields (static or 50 Hz), Mutat. Res–89 (2000) 453 . 96

[28] K. Chow, W.L. Tung, Magnetic field exposure enhances DNA repair through the induction of DnaK/J synthesis, FEBS Lett. 478 (2000).136–133 .

[29] .G. Robison, A.R. Pendleton, K.O. Monson, B.K. Murray, K.L. O'Neil DecreasedDNArepair rates and protection from heat induced apoptosis mediated by electromagnetic field exposure, Bioelectromagnetics112–106 (2002) 23

[30] R. Lourencini da Silva, F. Albano, L.R. Lopes dos Santos, A.D Tavares Jr., I. Felzenszwalb, The effect of electromagnetic field exposure on the formation ofDNAlesions, Redox. Rep. 5 (2000) 299–301.

[31] J. Jajte, M. Zmyslony, J. Palus, E. Dziubaltowska, E. Rajkowska، Protective effect of melatonin against in vitro iron ions and 7mT50Hz magnetic field-induced DNA damage in rat lymphocytes, Mutat. Res 483 . .64–57 (2001)

[32] Collins AR 2004. The comet assay for DNA damage and repair: Principles, applications, and limitations Mol Biotechnol, 26: 249-261

[33] Fenech M 2002. Biomarkers of genetic damage for cancer epidemiology. Toxicology, 181: 411-416.

[34] Ruiz-Gómez MJ, Martínez-Morillo M 2009. Electromagnetic fields and the induction of DNA strand breaks. Electromagn Biol Med, 28: 201-214.

[35] Mashevich M, Folkman D, Kesar A, Barbul A, Korenstein R, Jerby E, Avivi L 2003. Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal instability. Bioelectromagnetics, 24: 82-90.

[36] Huang TQ, Lee MS, Oh E, Zhang BT, Seo JS, Park WY: Molecular responses of Jurkat T-cells to 1763 MHz radiofrequency radiation. Int J Radiat Biol 2008, 84(9):734-741.

[37] Koyama S, Takashima Y, Sakurai T, Suzuki Y, Taki M، Miyakoshi J 2007. Effects of 2.45 GHz electromagnetic fields with a wide range of SARs on bacterial and HPRT gene mutations. J Radiat Res, 48.75–69 :

[38] Phillips JL, Singh NP, Lai H 2009. Electromagnetic fields and DNA damage. Pathophysiology, 16: 79–88.

[39] Wolf FI, Torsello A, Tedesco B, Fasanella S, Boninsegna A, D'Ascenzo M, Grassi C, Azzera GB, Cittadini A 2005. 50-Hz

extremely low frequency electromagnetic fields enhance cell proliferation and DNA damage: Possible involvement of a redox mechanism Biochim Biophys Acta, 174: 120–129.

[40] El-Abd SF, Eltoweissy MY 2012. Cytogenetic alterations in human lymphocyte culture following exposure to radiofrequency field of mobile phone. JAPS, 2: 16-20.

[41] Ozmen I, Naziroglu M, Alici HA, Sahin F, Cengiz M, Eren I. Spinal morphineadministration reduces the fatty acid contents in spinal cord and brain byincreasing oxidative stress. Neurochem Res 2007;32:19–25

[42] Helleday T, Loc J, van Gentd DC, et al. DNA double strand break repair:from mechanistic understanding to cancer treatment[J]. DNA Repair , 2007, 6 :923–35. doi:10.1016/j.dnarep.2007.02.00

[43] Schindowski K, Leutner S, Muller WE, et al. Age related changes of apoptotic cell death in human lymphocytes[J]. Neurobiol Aging , 2000, 21 :661–70. doi:10.1016/S0197-4580(00)00171-8.

[44] Megha K, Deshmukh PS, Banerjee BD, et al. Low intensity microwave radiation induces genotoxicity in rat brain[J]. Neurotoxicology , 2015, 51 :158–65. doi:10.1016/j.neuro.2015.10.009.

[45] Simko M. Cell type specific redox status is responsible for diverse electromagnetic field effect[J]. Curr Med Chem , 2007, 14 :1141–52. doi:10.2174/092986707780362835.

[46] B. Oral, M. Guney, F. Ozguner, N. Karahan, T. Mungan, S. Comlekci‹ G. Cesur, Endometrial apoptosis induced by a 900-MHz mobile phone: preventive effects of vitamins E and C, Adv. Ther. 23 (2006) .973–957

[47] M. Simkó, Cell type specific redox status is responsible for diverse electromagnetic field effects, Curr. Med. Chem. 14 (2007) 1141–1152.