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ORIGINAL ARTICLE

Effect of Artificial Light Sources on DNA Damage in Human Mononuclear Lymphocyte Cells Under In Vitro Conditions

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ABSTRACT

Aim: This study aimed to evaluate the effects of commonly used artificial light sources on DNA damage and oxidative stress parameters in human mononuclear lymphocyte cells. The potential effects of different lighting sources on cellular DNA integrity and oxidative stress levels were comprehensively evaluated. **Methods:** Isolated human mononuclear lymphocyte cells were exposed to artificial light sources at varying intensities and durations. DNA damage was analyzed using the alkaline single-cell gel electrophoresis (comet assay) method. The oxidative status of the cells was assessed using Erel's method. **Results:** The highest DNA damage was observed with white and yellow compact fluorescent lamps, whereas the least damage occurred with yellow incandescent light sources. Additionally, 100-watt fluorescent lamps caused the most DNA damage, while the least damage was detected with 20-watt lamps. Oxidative stress index levels significantly increased with prolonged exposure. **Conclusion:** Our findings indicate that artificial light sources can induce DNA damage by increasing both direct and oxidative stress. Different light types and exposure durations significantly affect cellular genotoxicity and oxidative stress levels. These results provide an important insight into the potential biological risks associated with artificial lighting.

Keywords: Artificial Light Sources, Comet Assay, DNA Damage, Oxidative Stress

ÖZET

Amaç: Bu çalışmanın amacı, günlük yapay ışık kaynaklarının insan mononükleer lenfosit hücrelerinde DNA hasarı ve oksidatif stres parametreleri üzerindeki etkilerini araştırmaktır. Farklı aydınlatma kaynaklarının hücresel DNA bütünlüğü ve oksidatif stres düzeylerine yönelik potansiyel DNA hasarı etkileri kapsamlı bir şekilde değerlendirmektir. **Yöntem:** İzole edilmiş insan mononükleer lenfosit hücreleri çeşitli yoğunluklarda yapay ışık kaynaklarına maruz bırakılmıştır. DNA hasarının analizi için alkalin tek hücreli jel elektroforezi (comet assay) yöntemi kullanılmıştır. Hücrelerin oksidatif durumları ise Erel yöntemiyle değerlendirilmiştir. **Bulgular:** En fazla DNA hasarının, beyaz ve sarı kompakt floresan lambalarda saptanmıştır. En az DNA hasarının ise sarı akkor ışık kaynaklarında meydana geldiği belirlenmiştir. Ayrıca, 100 watt'lık floresan ışık kaynaklarının en fazla DNA hasarına neden olduğu saptanmıştır. En az hasar ise 20 watt'lık lambalarda gözlemlenmiştir. Işığa maruz kalma süresi arttıkça oksidatif stres indeksinin anlamlı derecede yükseldiği tespit edilmiştir. **Sonuç:** Çalışmamız, yapay ışık kaynaklarının hem direk hem de oksidatif stres düzeylerini artırarak DNA hasarına neden olabileceğini ortaya koymuştur. Bulgularımız, farklı yapay ışık kaynaklarının ve maruziyet sürelerinin hücresel genotoksisite ve oksidatif stres üzerinde belirgin etkiler yarattığını göstermektedir. Bu sonuçlar, yapay aydınlatmanın potansiyel biyolojik risklerini göstermektedir.

Anahtar Kelimeler: Yapay Işık Kaynakları, Comet Assay, DNA Hasarı, Oksidatif Stres

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INTRODUCTION

Deoxyribonucleic acid (DNA) is the foundation of genetic information and plays a pivotal role in maintaining cellular integrity (1). Permanent alterations in DNA, such as mutations, can be inherited and contribute to disease and cancer development (1,2). Cells within an organism are constantly exposed to various physical and chemical factors that can damage proteins, lipids, and nucleic acids, ultimately compromising cellular function and stability (2).

DNA damage can result from both external sources, such as ultraviolet (UV) radiation and chemical agents, and internal factors, including errors during DNA replication and recombination and metabolic by-products such as free radicals. These reactive molecules cause oxidative damage to DNA, proteins, and lipids, leading to an increased oxidative stress index (OSI) within cells (3).

Oxidative stress occurs when there is an imbalance between oxidant production and the system that neutralizes them. Excessive accumulation of reactive oxygen species (ROS) and reactive nitrogen species (RNS), which are part of normal cell metabolism, can cause significant molecular damage, especially to DNA.

Artificial light sources are an essential part of modern life. People are exposed to them for long periods due to extended indoor activities and changing lifestyles. These light sources include incandescent bulbs, fluorescent lamps, compact fluorescent lamps, halogen lamps, and high-pressure mercury vapor lamps. The light they emit can have various biological effects.

UV light, particularly UV-C radiation, is known for its strong DNA-damaging effects (4). UV-C can cause covalent bonds to form between adjacent pyrimidines, leading to pyrimidine dimers in DNA (4). These lesions can disrupt replication and transcription, increasing the risk of mutations and cancer. Studies have linked exposure to UV light, γ-radiation, and certain chemical agents with the etiology and pathogenesis of malignancies (5). High levels of oxidative stress play a role in aging, various diseases, and the onset and progression of cancer (6).

While the harmful effects of UV radiation are well-known, the biological impact of other artificial light sources remains unclear. In particular, their role in causing DNA damage and oxidative stress needs further investigations. Given the widespread use of artificial lighting, understanding these effects

is important for public health and safety.

This study aimed to evaluate the effects of artificial light sources, applied at varying intensities and durations, on DNA damage and oxidative stress parameters in vitro in human mononuclear lymphocyte cell cultures. This research will help identify potential biological risks associated with artificial lighting and contribute to safety guidelines for exposure. To the best of our knowledge, this is the first study investigating the relationship between DNA damage and oxidative stress caused by artificial light sources commonly used in daily life.

MATERIAL AND METHODS

Light Sources

This study used four types of artificial light sources: a white compact fluorescent lamp (WCFL), a yellow compact fluorescent lamp (YCFL), a fluorescent lamp, and a yellow incandescent lamp (YIL). These light sources were mounted 72° apart on the perimeter of a circular board (30×30 cm) to ensure uniform exposure. No other light sources were present in the environment where the study was conducted.

Sample Collection and Cell Preparation

Venous whole blood was collected from a healthy volunteer into heparinized tubes to prevent coagulation. Mononuclear cells were isolated using the Histopaque-1077 density gradient centrifugation method. Briefly, blood was layered over the Histopaque solution and centrifuged at 400 × g for 30 minutes at room temperature. The mononuclear cell layer was carefully aspirated, washed twice with phosphate-buffered saline, and resuspended in 20 mL of cell culture medium.

The leukocyte suspension was distributed into flasks containing 10 mL of Dulbecco's modified eagle's medium supplemented with 10% fetal bovine serum, 1% penicillin-streptomycin, and 1% L-glutamine. The flasks were incubated at 37°C in a humidified incubator with 5% CO_2 to maintain physiological conditions and cell viability.

Exposure to Artificial Light

Mononuclear cells were vertically exposed to light from each artificial light source at a standardized distance of 1 meter. The light sources were operated at five different intensities (20W, 40W, 60W, 80W, and 100W), and exposure durations were set at one, two, and three hours to assess time-dependent effects. The incubator was completely shielded from external light to ensure exclusive exposure to the experimental light sources. Each experiment was conducted in triplicate, and standard deviations were calculated. External factors that could potentially cause DNA damage were controlled to minimize confounding effects.

Experimental Controls

Negative and positive control samples were obtained from healthy individuals included in previous studies. Negative controls consisted of cell suspensions incubated in the culture medium without light exposure. No DNA damage was observed in these samples. Positive controls comprised cell suspensions

incubated with 30% hydrogen peroxide (H_2O_2) for 30 minutes to induce maximum DNA damage. After incubation, H_2O_2 was removed by centrifugation, and the cells were resuspended in fresh medium.

Comet Assay for DNA Damage Analysis

DNA damage was assessed using the single-cell gel electrophoresis assay, commonly known as the comet assay, with modifications described by Singh et al. (7). All procedures were performed under minimal illumination to prevent unintended DNA damage. Briefly, 50 µL of cell suspension (~10,000 cells) was mixed with 500 µL of 1% low-melting-point agarose at 37°C and spread onto microscope slides pre-coated with normal melting point agarose. The slides were immersed in a lysis solution (2.5 M NaCl, 100 mM EDTA, 10 mM Tris, pH 10, with 1% freshly added Triton X-100) at 4°C for one hour to lyse cells and remove proteins, leaving nucleoids containing supercoiled DNA.

Following lysis, the slides were placed in a horizontal electrophoresis tank containing fresh alkaline electrophoresis buffer (300 mM NaOH, 1 mM EDTA, pH >13) and left for 20 minutes to allow DNA unwinding and the expression of alkali-labile sites. Electrophoresis was performed at 25 V (0.86 V/cm) and 300 mA for 20 minutes. After electrophoresis, the slides were neutralized with three washes of neutralization buffer 0.4 M Tris (pH 7.5) and stained with ethidium bromide (2 µg/mL).

Fluorescence microscopy (Olympus BX51, Japan) at 200× magnification was used for analysis. DNA damage was visually classified based on tail formation in 100 cells per slide, categorized into five classes from 0 (no damage) to 4 (maximum damage) (Figure 1). An arbitrary unit system was used for quantification (8).

Assessment of Oxidative Status

Total oxidant status (TOS) and total antioxidant status (TAS) in the cell culture medium were determined using commercially available assay kits. These values were measured via colorimetric methods developed by Erel (9) on a fully automated analyzer (Architect CI16200; Abbott Laboratories, Abbott Park, IL, USA).

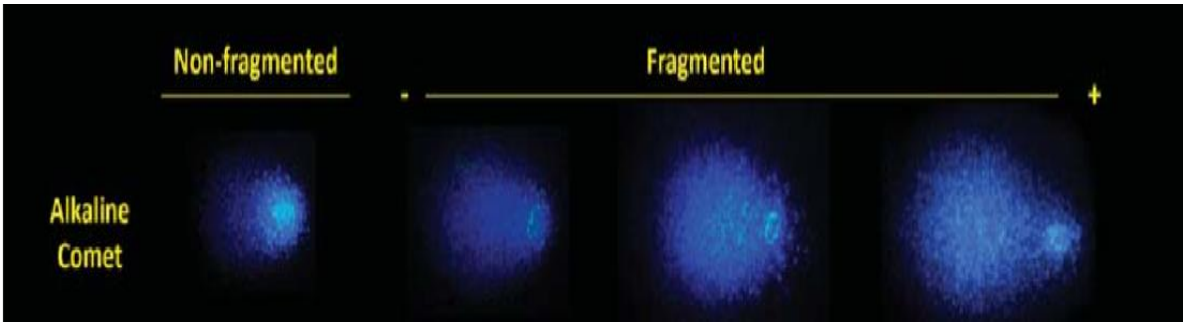

Figure 1. Non-fragmented and fragmented human mononuclear lymphocyte cells in the alkaline comet assays.

•TOS assay: This assay measures oxidation of ferrous ions to ferric ions in the presence of oxidants, forming a colored complex with xylenol orange in an acidic medium. The color intensity is proportional to the total oxidant content.

•TAS assay: This assay evaluates the antioxidative effect of the sample against a potent free radical reaction initiated by hydroxyl radical production. The absorbance change is inversely proportional to total antioxidant levels.

OSI was calculated as the percentage ratio of TOS to TAS (OSI = TOS/TAS) to quantify oxidative stress. TOS and TAS values were recorded at multiple time points during exposure.

Statistical Analysis

Data were analyzed using SPSS software (version 25.0; IBM Corp., Armonk, NY, USA). Results were presented as mean ± standard deviation or frequency (percentage). The association between DNA strand break values and malondialdehyde levels was evaluated using the Pearson correlation coefficient. Differences between groups were analyzed using one-way analysis of variance followed by Tukey's post hoc test. A p-value of <0.05 was considered statistically significant.

RESULTS

In experiments with the WCFL, the highest DNA damage was observed at 100 W after three hours, while the lowest DNA damage occurred at 20 W after one hour. A significant positive correlation was identified between light intensity and DNA damage, with higher intensities causing greater damage. DNA damage also increased proportionally with exposure, indicating a cumulative effect on DNA integrity. Specifically, DNA damage was significantly higher at three hours than that at two hours, and higher at two hours than at one hour (Table 1).

Table 1. DNA damage (arbitrary units) in relation to increasing light exposure duration in experiments with a white compact fluorescent lamp

	Hour 1	Hour 2	Hour 3	Positive control	Negative control	p
20 watt*	0 ± 0	2.66 ± 0.57[a]	9.33 ± 0.57[b.c]	80.33 ± 0.57	0 ± 0	<0.05
40 watt*	0.33 ± 0.57	5 ± 0[a]	10.66 ± 1[b.c]	77.33 ± 0.57	0 ± 0	<0.05
60 watt*	1.66 ± 1.52	7.66 ± 0.5773	15.33 ± 0.57[b.c]	77.33 ± 1.15	0 ± 0	<0.05
80 watt*	6.33 ± 1.15	10 ± 1[a]	21 ± 2[b.c]	81.67 ± 1.52	0 ± 0	<0.05
100 watt*	13.33 ± 1.52	17.66 ± 0.57	33.66 ± 3.51[b.c]	80.33 ± 2.51	0 ± 0	<0.05

[a,b,c]denote statistically significant differences between hours 1 and 2, hours 1 and 3, and hours 2 and 3, respectively. Note: Data presented as mean ± standard deviation

Similarly, in all experiments with the WCFL, OSI values increased significantly with longer exposure durations ($p<0.05$), indicating a direct relationship between exposure time and oxidative stress levels. These results suggest that prolonged exposure to WCFL exacerbates oxidative stress in mononuclear cells.

Experiments with the YCFL demonstrated a similar trend, where DNA damage was positively correlated with light intensity. The highest DNA damage was observed at 100 W, with the order of damage by light intensity being 100 W > 80 W > 60 W > 40 W > 20 W ($p<0.05$, Table 2). Exposure duration also played a significant role in DNA damage, with damage at three hours being higher than at two hours and damage at two hours being higher than at one hour ($p<0.05$, Table 2). These findings highlight the additive effects of light intensity and exposure time on DNA damage.

Similar to WCFL, OSI values in YCFL experiments increased significantly with longer exposure times ($p<0.05$). This suggests that prolonged exposure to YCFL leads to increased oxidative stress, emphasizing the importance of understanding the long-term biological effects of these light sources.

Experiments with FL revealed significant DNA damage under all conditions. DNA damage levels were lower than in the positive control but higher than in the negative control in all experiments, confirming the genotoxic potential of FL. A significant increase in DNA damage was observed with increasing light intensity, with the highest damage recorded at 100 W. The order of DNA damage based on light intensity was 100 W > 80 W > 60 W > 40 W > 20 W ($p<0.05$, Table3). Similarly, OSI values increased significantly with longer exposure durations ($p<0.05$). This consistent increase indicates a cumulative effect of prolonged FL exposure on oxidative stress, further highlighting the potential risks of long-term exposure.

Table 2. DNA damage (arbitrary units) in relation to increasing light exposure duration in experiments with a yellow compact fluorescent lamp

	Hour 1	Hour 2	Hour 3	Positive control	Negative control	p
20 watt	0 ± 0	2.33 ± 1.15	7.66 ± 1.15[b]	83 ± 3	0 ± 0	<0.05
40 watt	0.66 ± 1.15	3.66 ± 0.57	9.33 ± 0.57[b.c]	82 ± 1	0 ± 0	<0.05
60 watt	2.33 ± 0.57	7 ± 0[a]	15.33 ± 0.57[b.c]	74.3 ± 2.51	0 ± 0	<0.05
80 watt	6 ± 1	9.66 ± 0.57	22.33 ± 0.57[b.c]	81.3 ± 1.52	0 ± 0	<0.05
100 watt	11.33 ± 0.57	15.66 ± 0.57[a]	31.33 ± 2.08[b.c]	79.67 ± 1.53	0 ± 0	<0.05

[a,b,c]denote statistically significant differences between hours 1 and 2, hours 1 and 3, and hours 2 and 3, respectively. Note: Data presented as mean ± standard deviation

Table 3. DNA damage (arbitrary units) in relation to increasing light exposure duration in experiments conducted with a fluorescent lamp

	Hour 1	Hour 1	Hour 3	Positive control	Negative control	p
20 watt	0 ± 0	2.33 ± 1.15[a]	7 ± 0[b.c]	83 ± 1	0 ± 0	<0.05
40 watt	0.33 ± 0.57	3 ± 0[a]	7.66 ± 1.15[b.c]	76.7 ± 1.53	0 ± 0	<0.05
60 watt	1.33 ± 0.57	5.66 ± 0.57	10 ± 1[b.c]	81 ± 2	0 ± 0	<0.05
80 watt	5.66 ± 0.57	7 ± 0[a]	15 ± 2.64[b.c]	82.67 ± 1.53	0 ± 0	<0.05
100 watt	8.33 ± 0.57	12.33 ± 0.57	25.66 ± 1.15[b.c]	81.3 ± 0.58	0 ± 0	<0.05

[a,b,c]denote statistically significant differences between hours 1 and 2, hours 1 and 3, and hours 2 and 3, respectively. Note: Data presented as mean ± standard deviation

In YIL experiments, DNA damage increased with light intensity, with the highest damage recorded at 100 W. The order of DNA damage by light intensity at the 2nd hours was 100 W > 80 W > 60 W = 40 W = 20 W. Although DNA damage was observed in all YIL experiments, it was consistently lower than in the positive control. The highest DNA damage occurred after three hours at 100 W, but this increase was not statistically significant across all conditions (Table 4).

Despite the generally lower DNA damage, OSI values increased significantly with longer exposure durations, indicating that even incandescent light, which is often considered less harmful, can induce oxidative stress with prolonged exposure.

Table 4. DNA damage (arbitrary units) in relation to increasing light exposure duration in experiments with an incandescent lamp

	Hour 1	Hour 2	Hour 3	Positive control	Negative control	p
20 watt*	0 ± 0	0 ± 0	0 ± 0	81.67 ± 1.53	0 ± 0	>0.05
40 watt*	0 ± 0	0 ± 0	0 ± 0	78 ± 1	0 ± 0	>0.05
60 watt*	0 ± 0	0 ± 0	0 ± 0	81.67 ± 1.15	0 ± 0	>0.05
80 watt*	0 ± 0	0.33 ± 0.57	0.66 ± 1.15	79.67 ± 1.53	0 ± 0	>0.05
100 watt*	0 ± 0	1.33 ± 0.57	2.33 ± 1.15	78.3 ± 2.3	0 ± 0	<0.05

[a,b,c]denote statistically significant differences between hours 1 and 2, hours 1 and 3, and hours 2 and 3, respectively. Note: Data presented as mean ± standard deviation

DISCUSSION

Previous studies have demonstrated that, in addition to UV radiation, both solar rays and artificial light sources can affect mutagenesis, carcinogenesis, and immune system functions (10).

Our study revealed that incandescent light sources induced DNA damage when mononuclear cells were exposed to high intensities for prolonged durations under in vitro conditions. In contrast, compact fluorescent and fluorescent light sources caused DNA damage at lower intensities and shorter durations. Furthermore, we observed a correlation between increased oxidative stress and DNA damage induced by these artificial light sources. However, our findings did not establish a significant correlation between the magnitude of DNA damage and OSI levels.

Previous research has identified multiple mechanisms by which light exposure can damage DNA (11). Pflaum et al. (12) reported that visible light inhibited antioxidants and induced oxidative DNA damage in mammalian cell cultures. Similarly, Kielbassa et al. (11) found that UV and visible light caused DNA damage via different mechanisms depending on the wavelength. Specifically, UV-B and UV-C (250–315 nm) induced direct DNA damage by forming pyrimidine dimers, whereas UV-A and visible light (320–740 nm) caused indirect DNA damage by generating oxidative radicals such as singlet oxygen, superoxide, and hydroxyl radicals. Additionally, Botta et al. (13) reported UV-A (320–400 nm) and visible light (400–800 nm) could trigger oxidative DNA damage by photoactivating polyaromatic hydrocarbons and benzopyrones.

Solar radiation causes both acute and chronic effects on human and animal skin cells (14). Chronic exposure leads to the development of benign and malignant skin tumors, particularly malignant melanoma. Animal studies have shown that UV-B (290–320 nm) is more mutagenic and carcinogenic than UV-A (320–400 nm) (15). Furthermore, epidemiological studies have demonstrated that UV radiation contributes to gene mutations and immunosuppression, increasing the risk of skin tumors. UV radiation has been reported to cause both direct and indirect DNA damage (16).

Since the skin is the primary organ exposed to light, skin cancers are among the most common malignancies in Western countries (17). Godley et al. reported that blue visible light (390–550 nm) increased free radical production and induced mitochondrial DNA damage in primary retinal epithelial cells (18). Consistent with these findings, our study also observed increased OSI levels and DNA damage in mononuclear cells following exposure to artificial light sources. However, it is important to note that our study was conducted in vivo using cell cultures. Further in vivo research involving living organisms is necessary to provide a better understanding of the biological relevance of these findings.

CONCLUSION

Our findings suggest that fluorescent and compact fluorescent lights induce greater DNA damage and oxidative stress than incandescent lights. A possible explanation is that incandescent lights primarily emit UV-A and visible light, which can indirectly cause DNA damage through oxidative radicals produced by photoreactions. In contrast, fluorescent and compact fluorescent lamps emit not only visible light but also UV-B and UV-C rays, which can directly damage DNA by forming thymine dimers.

This in vitro study indicates that artificial light sources commonly used in daily life are not entirely harmless and can cause significant DNA damage and oxidative stress when used at high intensities and for prolonged durations. Therefore, limiting light intensity and exposure time may help mitigate potential damage. Additionally, incandescent lights, which resulted in lower DNA damage, may be a safer alternative for reducing the risk of genotoxicity. However, given the in vitro nature of this study, further in vivo research is required to confirm these findings and assess their implications for human health.

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Ethics approval: This study was approved by the Clinical Research Ethics Committee of Harran University (HRÜ23.06.14).

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