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# The photocytotoxicity of different lights on mammalian cells in interior lighting system

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ABSTRACT

In the present paper, two light sources commonly used in interior lighting system: incandescent light and light emitting diode (LED) were chosen to evaluate their influences on three kinds of mammalian cells, together with UVA and UVB, and the mechanism of the photocytotoxicity was investigated in terms of intracellular ROS production, lipid peroxidation, SOD activity and GSH level assays. The results showed that LED and incandescent light both had some photocytotoxicities. In the interior lighting condition (100 lx–250 lx), the cytotoxicities of LED and incandescent lamp on RF/6A cells (rhesus retinal pigment epithelium cell line) were stronger than that on two fibroblast cell lines, while the cytotoxicity of UVA and UVB on HS68 cells (fibroblast cell line) was highest in the tests. The mechanism analysis revealed that the photocytotoxicities of LED and incandescent lamp were both caused by cell lipid peroxidation. LED and incandescent light could promote the production of ROS, raise lipid peroxidation level and lower the activity of the antioxidant key enzymes in mammalian cells, and finally cause a number of cells death. However, the negative function of LED was significantly smaller than incandescent light and ultraviolet in daily interior lighting condition. And the significantly lower photocytotoxicity of LED might be due to the less existence of ultraviolet. Therefore, LED is an efficient and relative safe light source in interior lighting system, which should be widely used instead of traditional light source.



## 1. Introduction

Human life is inseparable from the light, whether outdoors or indoors. However, constantly exposure to potentially harmful lights, like ultraviolet (UV), will make human skin liable to aging process [1,2]. UV irradiation has deleterious effects on human skin, including sunburn, immune suppression, cancer, and photo-aging [3]. UVB, in particular, is the most hazardous environmental carcinogen known with regard to human health through generation of reactive oxygen species (ROS) [4,5]. The ROS results in the subsequent activation of complex signaling pathways, followed by matrix metalloproteinases (MMPs) induction in skin cells and degradation or synthesis inhibition of collagenous extracellular matrix in connective tissues [6].

The light environment, where people live, includes sunlight and artificial. Currently, there are several man-made light sources used for interior lighting, such as incandescent light, fluorescent, light emitting diode (LED), etc. Incandescent light is the first electric light invented by T.A. Edison in 1879, which is culminating in the modern production methods after many improvements during more than a hundred years. It can emit a continuous spectrum, and most of the energy distributes in the visible spectral region. The color rendering of incandescent light is the best in all of light resource [7]. But because of low luminous efficiency, power consumption, etc., governments are introduced their incandescent phase-out plan. Compared with the incandescent light, LED is a new light source. LED is a semiconductor component, which can convert electrical energy into visible light solid-state. LED itself has many advantages, such as high luminous efficiency, long life, dimmable and so on. Because of the unique working principle, LED has the flexibility to produce many required spectra [8].

Do the incandescent light and LED, which people are daily expose to, have no harm for people? This question is closely related to people's health. But, to our best knowledge, there is no systematical study on their photocytotoxicities on mammalian cells in daily interior lighting condition. Therefore, in the present study, two light sources, commonly used in interior lighting system: incandescent light and LED, were chosen to evaluate their influences on three kinds of mammalian cells for the first time, together with UVA and UVB (EB-160C/12, Spectronics Corporation, USA).

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Furthermore, the mechanism of the photocytotoxicity was investigated in terms of intracellular ROS production, lipid peroxidation, SOD activity and GSH level assays.

## 2. Materials and methods

### 2.1. Materials

Incandescent light (Edison 25W/CL/GLS/E27, product number: 70331) was purchased from General Electric Company, with 25-W power, 2800 K ± 500 K color temperature and 25 lm/W luminous efficiency. LED (MASTER LED spotLV MR16, product number: 929000172308) was purchased from Koninklijke Philips Electronics N.V., with 7-W power, 4000 K ± 500 K color temperature and 60 lm/W luminous efficiency. E Series UV Hand-Held Lamps (EA-160/12 and EB-160C/12) were purchased from Spectronics Corporation, USA. EA-160/12, equipped with one 6-W integrally filtered BLB tube, provided UVA condition; EB-160C/12, equipped with one 6-W tube and filter assembly, provided UVB condition.

Commercial kits used for determining lactate dehydrogenase (LDH) and malondialdehyde (MDA) were obtained from the Jiancheng Institute of Biotechnology (Nanjing, China). Other chemicals used in these experiments of analytical grade were obtained from commercial sources.

### 2.2. Cell treatment

RF/6A cells (rhesus retinal pigment epithelium cell line, ATCC® number: CRL-1780™), HS68 cells (normal human foreskin cell line, ATCC® number: CRL-1635™) and 3T3-L1 cells (mouse embryonic fibroblast-adipose like cell line, ATCC® Number: CL-173™) were obtained from American type culture collection (ATCC), USA. The cells were cultivated in Dulbecco's Modified Eagle Medium (DMEM) supplemented with 10% bovine serum (BS), 2 mM glutamine and 1% streptomycin/penicillin on 100 mm plastic culture dishes (BD Falcon, USA), at 37 °C in a humidified atmosphere containing 5% $CO_2$ and the medium was changed every 2–3 days. Cells were incubated up to about 24 h and grown to about 80% confluence before experiments.

Cells in culture dishes were washed and then covered with 10 mL of Hanks balanced salt solution (1.3 mM $CaCl_2$, 5.4 mM KCl, 0.4 mM $KH_2PO_4$, 0.5 mM $MgCl_2{\cdot}6H_2O$, 0.4 mM $MgSO_4{\cdot}7H_2O$, 136.7 mM NaCl, 4.2 mM $NaHCO_3$ and 0.3 mM $NaH_2PO_4{\cdot}H_2O$). All cells were irradiated with the different lights for 20 min and then cultured for 24 h. There are four kinds of light sources used in the experiment, including LED, incandescent light, UVA and UVB. The illuminations of LED, incandescent light and UVB were 250 lx, and that of UVA was 100 lx. The illumination was controlled by dimmer switch (No. 56101, Simon Electric Co. Ltd., China) and Testo 545 illuminometer (Testo AG, Germany).

### 2.3. Cell viability assay

Cells were seeded onto 24-well plates, 24 h prior to the experiment at a density of $5 \times 10^4$ well$^{-1}$. Cells were exposed to the light sources for 20 min. The photocytotoxicity was evaluated 24 h after light source exposure using the MTT assay whereby the tetrazolium salt (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) is reduced by intracellular dehydrogenases of viable living cells leading to the formation of purple formazan crystals [9].

After 24 h, culture medium was removed and cells were incubated for 2 h at 37 °C with MTT salt solution (0.5 mg/mL) in PBS. The MTT solution was then removed and the crystals were dissolved in 400 mL DMSO. The optical density of each well was read at 550 nm using a microplate reader (Bio-Rad Model 680, USA). Cell viability is expressed as a percentage of live cells compared to unexposed control.

### 2.4. Lactate dehydrogenase (LDH) release assay

Photocytotoxicity induced by different light sources was also assessed by LDH leakage into the culture medium. Following exposure to different lights as described above for the cell viability assay, the cells were harvested and the LDH activity was assayed spectrophotometrically following the decrease in the absorbance of NADH at 340 nm by LDH assay kit.

### 2.5. Intracellular ROS assay

Cells were seeded onto 24-well plates, 24 h prior to the experiment at a density of $5 \times 10^4$ well$^{-1}$. Cells were irradiated with different light source in PBS as described above for the cell viability assay. For detection of photo-induced intracellular ROS formation, the ROS-index probe, carboxy-$H_2$DCFDA, was used. This compound readily diffuses across cell membranes, is hydrolyzed by intracellular esterases, and in the presence of ROS, is oxidized to dichlorofluorescein which is highly fluorescent and whose emission maximum can be monitored at 530 nm [10]. After light exposure, PBS was removed from cells and replaced with a solution of carboxy-$H_2$DCFDA (final concentration 5 mM) in PBS. Cells were incubated in this solution for 30 min at 37 °C before fluorescence measurements, which were carried out using a dual scanning microplate spectrofluorometer (Spectramax Gemini; Molecular Devices, Sunnyvale, CA, USA) with 480 nm excitation and 530 nm emission.

### 2.6. Measurement of intracellular malondialdehyde (MDA)

The thiobarbituric acid assay (TBARS) was used to detect lipid peroxidation [11]. After treatment, cells were washed with PBS and homogenized in 300 μL 0.1% Triton X-100 (PBS, pH 7.4) through sonication on ice for 10 s. After incubation at 4 °C for 10 min, the homogenates were centrifuged at 10,000 rpm for 10 min, and the supernatants were used for assay. MDA content was measured with the MDA kit.

### 2.7. Superoxide dismutase (SOD) activity assay

SOD activity was measured based on the extent inhibition of amino blue tetrazolium formazan formation in the mixture of nicotinamide adenine dinucleotide, phenazine methosulphate and nitroblue tetrazolium (NADH–PMS–NBT) [12]. Assay mixture contained 0.1 mL of cell lysate, 1.2 mL of sodium pyrophosphate buffer (pH 8.3, 0.052 M), 0.1 mL of PMS (186 μM), 0.3 mL of NBT (300 μM) and 0.2 mL of NADH (750 μM). Reaction was started by addition of NADH. After incubation at 30 °C for 90 s, the reaction was stopped by addition of 0.1 mL of glacial acetic acid. Reaction mixture was stirred vigorously with 4.0 mL of *n*-butanol. Color intensity of the chromogen in butanol was measured spectrophotometrically at 560 nm.

### 2.8. Glutathione (GSH) level assay

The glutathione content of cell suspensions was determined by the DTNB–GSSG reductase recycling assay as described in Anderson (1985), with some modifications [13]. Briefly, 200 μL of cell suspension was added to 200 μL of 10% (w/v) 5-sulfosalicylic acid for protein precipitation and centrifuged 2 min at 12,000 rpm. Supernatant aliquots were taken out for measurement of total glutathione (GSx) following the DTNB oxidation at 415 nm and compared with a standard curve. The final concentrations of the

assay reagents were 0.6 mM DTNB, 0.2 mM NADPH and 2 U/mL glutathione reductase. For the GSSG determination, 100 μL of supernatant was derivatized with 2 μL of 2-vinylpyridine and 10 μL of 50% (v/v) ethanolamide and mixed continuously for 60 min. GSSG was then measured as described above for total glutathione. The GSH content was calculated by subtracting GSSG content from the total glutathione content. The results are expressed as nmol GSH/mg of protein.

### 2.9. Spectrum analysis

The spectra were recorded using a PMS-80 Sync-Skan High Accuracy Test System (Everfine Photo-E-Info Co. Ltd., Hangzhou, China). The temperature is 25 °C, and the humidity is 43%.

### 2.10. Statistical analysis

All data were expressed as the mean ± SD of six replicates and examined for statistical significance with Student's *t*-tests. A result is considered statistically significant when $p < 0.05$. Two-way ANOVA was conducted to assess significant differences among the treatments as a whole in order to avoid the error inherent in performing multiple *t*-tests.

## 3. Results

### 3.1. Photocytotoxicity of different lights on mammalian cells

In this study, there are three cell lines, including two fibroblast cell lines: HS68 cells (normal human foreskin cell line, ATCC® number: CRL-1635™) and 3T3-L1 cells (mouse embryonic fibroblast-adipose like cell line, ATCC® Number: CL-173™), and one endothelial cell line: RF/6A cells (rhesus retinal pigment epithelium cell line, ATCC® number: CRL-1780™), used to evaluate the possible photocytotoxicity of LED, incandescent light, UVA and UVB on mammalian cells.

The light intensity 100–300 lx is the normal interior illumination which could satisfy human comfort requirements for a long period of time [14]. So, in order to choose suitable light intensity for experiment, photocytotoxicity of LED, incandescent light, UVA and UVB on HS68 cells was assayed in 100, 150 and 250 lx light-intensity and 20 min exposure by MTT method [15]. The results revealed that there was no statistical significant ($p \geqslant 0.05$) of photocytotoxicity of LED or incandescent light among three light-intensity groups. Moreover, survival ratios of cells in 150 lx and 250 lx UVA groups were all below 5%. Therefore, LED (250 lx), incandescent light (250 lx), UVA (100 lx) and UVB (250 lx) were chosen to be studied the photocytotoxicity on mammalian cells.

From the results reported in Fig. 1, the exposure to all four lights lead to significantly decrease in viability of mammalian cells from comparison with the unexposed controls. In the four lights, the photocytotoxicity of LED was weakest, and the survival ratios of cells were respectively 68.0% (RF/6A), 87.2% (HS68) and 89.2% (3T3-L1). Unfortunately, the survival ratios of cells in incandescent light groups were respectively 53.1% (RF/6A), 56.1% (HS68) and 64.8% (3T3-L1), the survival ratios of cells in UVA groups were respectively 21.9% (RF/6A), 9.3% (HS68) and 36.8% (3T3-L1), the survival ratios of cells in UVB groups were respectively 40.1% (RF/6A), 28.6% (HS68) and 44.7% (3T3-L1).

### 3.2. Lactate dehydrogenase (LDH) leakages in mammalian cells induced by different lights

Lactate dehydrogenase (LDH) is an enzyme (EC 1.1.1.27) present in a wide variety of organisms, including plants and animals.

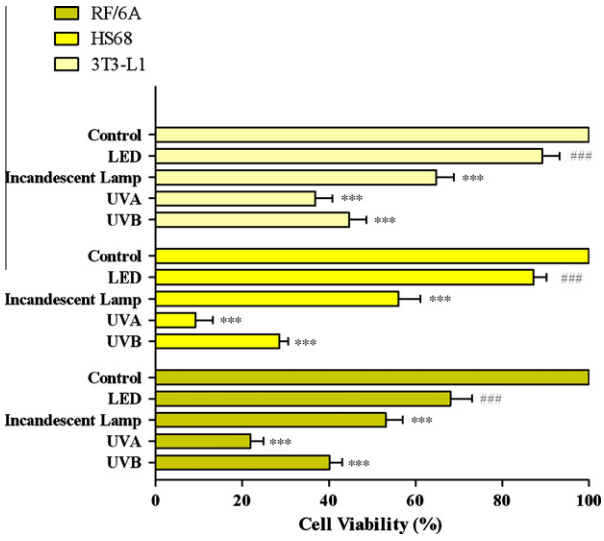


**Fig. 1.** Viability of mammalian cells following different light sources exposure, determined using the MTT assay. Each line represents the mean ± SD of at least three independent experiments. $^{###}p < 0.001$ Denotes significant difference from control group. $^{***}p < 0.001$ Denotes significant difference from LED group.

Cell breakdown releases LDH, and therefore LDH can be measured as a surrogate for cell breakdown. Photocytotoxicity induced by different light sources was also assessed by LDH leakage into the culture medium. As shown in Fig. 2, the LDH leakages induced by the four lights increased in all light groups compared with the non-irradiated control. The increments of LDH leakages in LED group were respectively 18.1% (RF/6A), 16.1% (HS68) and 17.6% (3T3-L1). While, the increments in incandescent light groups were respectively 26.4% (RF/6A), 38.2% (HS68) and 31.9% (3T3-L1), in UVA groups were respectively 51.8% (RF/6A), 65.0% (HS68) and 81.2% (3T3-L1), and in UVB groups were respectively 32.6% (RF/6A), 52.1% (HS68) and 60.0% (3T3-L1). This result was consistent with photocytotoxicities of the four lights, which indicated the cell damage induced by LED the weakest of the four lights. Moreover, the above two results revealed that the four lights all resulted in cell breakdown, and then caused the cell death. So, the photocytotoxicities of the four lights might occur at cell membrane to make cells ruptured.

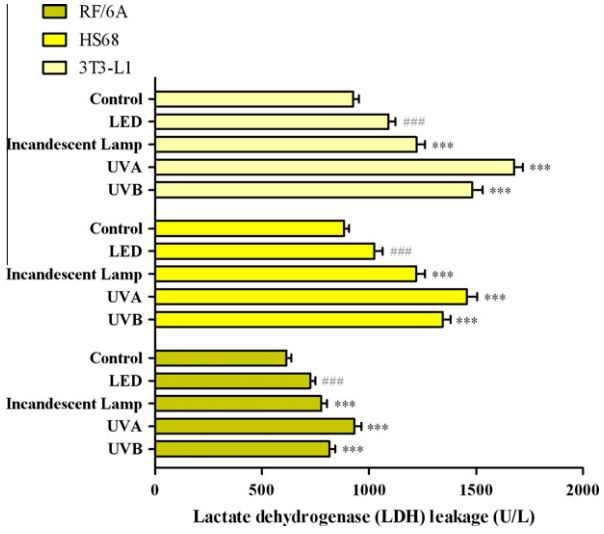


**Fig. 2.** LDH leakages in mammalian cells following different light sources exposure, Each line represents the mean ± SD of at least three independent experiments. $^{###}p < 0.001$ Denotes significant difference from control group. $^{***}p < 0.001$ Denotes significant difference from LED group.

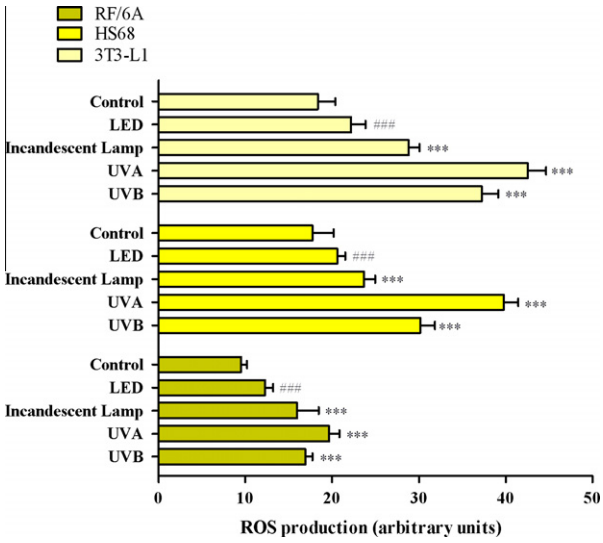


**Fig. 3.** ROS production in mammalian cells following different light sources exposure, Each line represents the mean ± SD of at least three independent experiments. $^{###}p < 0.001$ Denotes significant difference from control group. $^{***}p < 0.001$ Denotes significant difference from LED group.

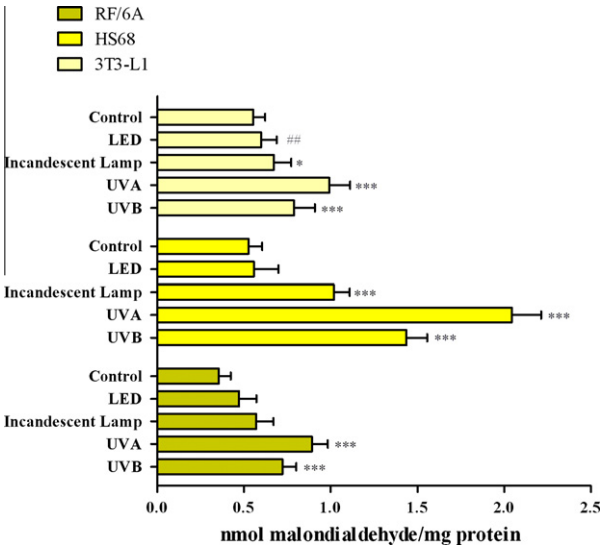


**Fig. 4.** Lipid peroxidation in mammalian cells following different light sources exposure, Each line represents the mean ± SD of at least three independent experiments. $^{##}p < 0.01$ Denotes significant difference from control group.$^{*}p < 0.05$ and $^{***}p < 0.001$ Denotes significant difference from LED group.

### 3.3. Intracellular ROS productions in mammalian cells induced by different lights

Reactive oxygen species (ROS) are chemically reactive molecules containing oxygen. ROS are produced as a normal product of cellular metabolism. Under normal circumstances, cells are able to defend themselves against ROS damage with enzymes. However, during times of environmental stress (e.g., UV or heat exposure), ROS levels can increase dramatically. This may result in significant damage to structures of cell membrane and induce cell apoptosis [16].

ROS production in mammalian cells was initially established using moderate light doses by measuring the fluorescence emitted from the oxidized carboxy-H2DCFDA ROS index probe. The results reported in Fig. 3 clearly demonstrated that irradiations generated the increase of intracellular ROS production. The productions of intracellular ROS in three cell lines induced by lights were all following the sequence: LED < incandescent light < UVB < UVA. Compared with the non-irradiated groups, the intracellular ROS productions induced by LED was increased 29.4% (RF/6A), 16.0% (HS68) and 20.7% (3T3-L1), while the productions induced by incandescent light was increased 68.2% (RF/6A), 33.3% (HS68) and 57.0% (3T3-L1).

### 3.4. Lipid peroxidation in mammalian cells induced by different lights

In order to evaluate the oxidative potency of the four lights on mammalian cells, we measured TBARS levels. Among these reactive substances, MDA is a unique end-product of lipid peroxidation which usually represents the extent of lipid peroxidation.

The four lights exposure increased MDA levels of cells (Fig. 4). Compared with the non-irradiated control groups, the MDA levels increments in LED group were respectively 35.9% (RF/6A), 5.9% (HS68) and 8.2% (3T3-L1). While, the increments in incandescent lamp groups were respectively 60.5% (RF/6A), 93.7% (HS68) and 21.9% (3T3-L1), in UVA groups were respectively 151.9% (RF/6A), 288.6% (HS68) and 79.7% (3T3-L1), and in UVB groups were respectively 103.7% (RF/6A), 173.0% (HS68) and 42.9% (3T3-L1).

### 3.5. Influence of different lights on SOD activity in mammalian cells

Super oxide dismutase (known as SOD) is an enzyme which acts as a catalyst in the process of dismutation of superoxide into oxygen and hydrogen peroxide. It is therefore a critical antioxidant defense which is present in nearly all cells which are exposed to oxygen [17].

In order to evaluate the oxidative potency of the four lights on mammalian cells, the SOD activity was measured in irradiated mammalian cells. As shown in Fig. 5, SOD activity in control group and tested groups differed significantly. Moreover, SOD activity in LED group was markedly ($p < 0.01$) higher than that in other tested groups. Compared with the non-irradiated control groups, the decline rate of SOD activity in LED group were respectively 17.8% (RF/6A), 9.5% (HS68) and 8.0% (3T3-L1). While that in incandescent lamp groups were respectively 23.9% (RF/6A), 25.2% (HS68) and 31.3% (3T3-L1), in UVA groups were respectively 40.3% (RF/6A), 44.5% (HS68) and 41.3% (3T3-L1), in UVB groups were respectively 28.6% (RF/6A), 37.7% (HS68) and 37.6% (3T3-L1).

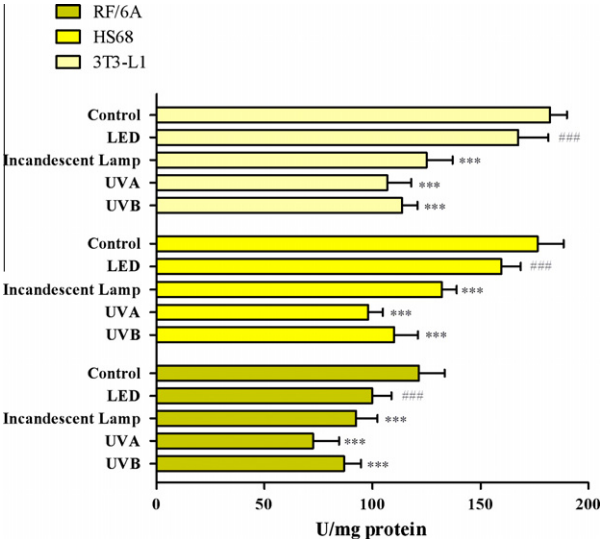


**Fig. 5.** SOD activities in mammalian cells following different light sources exposure, Each line represents the mean ± SD of at least three independent experiments. $^{###}p < 0.001$ Denotes significant difference from control group. $^{***}p < 0.001$ Denotes significant difference from LED group.

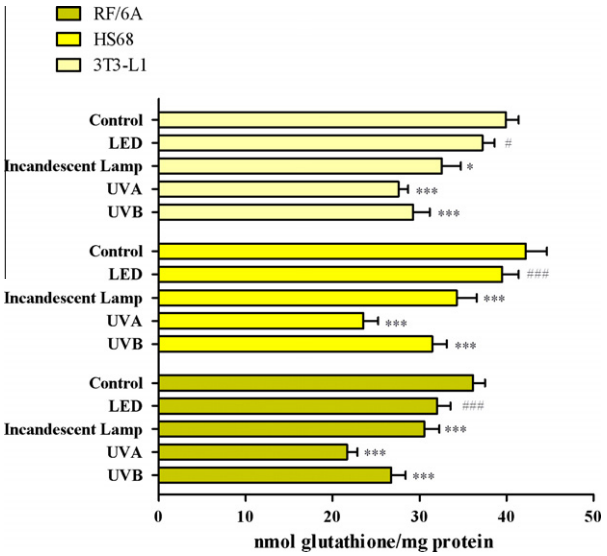


**Fig. 6.** Glutathione (GSH) content in mammalian cells following different light sources exposure, each line represents the mean ± SD of at least three independent experiments. $^{\#}p < 0.05$ and $^{\#\#\#}p < 0.001$ Denotes significant difference from control group. $^{*}p < 0.05$ and $^{***}p < 0.001$ Denotes significant difference from LED group.

### *3.6. Influence of different lights on glutathione (GSH) level in mammalian cells*

Glutathione, L-γ-glutamyl–L-cysteinylglycine (GSx), is the most important nonprotein thiol present in mammalian cells. This tripeptide is involved in many diverse biological processes such as protection against reactive electrophiles and peroxides, maintenance of the sulfhydryl status of proteins, modulation of enzyme activity by disulfide interchange and transport processes [18]. Measurement of GSx and its reduction state (GSH) may thus provide information about cellular responses to xenobiotics as well as about mechanisms of toxicity.

As shown in Fig. 6, the GSH levels in mammalian cells were all decreased by irradiation of four lights. In the four lights, the influence of LED was smallest, the GSH level in LED group was markedly ($p < 0.01$) higher than that in other tested groups. Compared with the non-irradiated control groups, the decline rates of GSH level in LED group were respectively 11.3% (RF/6A), 9.5% (HS68) and 6.7% (3T3-L1). While, that in incandescent lamp groups were respectively 15.4% (RF/6A), 18.9% (HS68) and 18.4% (3T3-L1), in UVA groups were respectively 40.1% (RF/6A), 44.3% (HS68) and 30.9% (3T3-L1), in UVB groups were respectively 20.7% (RF/6A), 25.6% (HS68) and 26.8% (3T3-L1).

## 4. Discussion

Exposure of human skin to light radiations over a period of years induces chronic photodamage, also called photoaging [19]. Photoaging is the result of morphological changes such as wrinkling and sagging due to general alteration of all the epidermal and dermal components of skin including the cutaneous cells. The human eye is exposed to ambient radiation that serves the fundamental biological functions of directing vision and circadian rhythm [20]. Although, the cornea cuts off all light below 295 nm, any substance that absorbs light above 295 nm and produces reactive oxygen species has the potential to damage the human eyes. Therefore, this is of importance to search an efficient and safe light source for human life.

Light-emitting diode (LED), a semiconductor light source, is used as indicator lamps in many devices and are increasingly used for other lighting [21]. Introduced as a practical electronic component in 1962, early LEDs emitted low-intensity red light, but modern versions are available across the visible, ultraviolet and infrared wavelengths, with very high brightness [22]. Over the last years, LEDs have been employed in diverse industrial products and have recently become popular due to the increased demand for energy-saving TVs. However, to our best knowledge, there is no study on the photocytotoxicity of interior lighting used LED on mammalian cells in interior lighting system and its mechanism by now. In the present paper, the influence of LED on mammalian cells was comprehensively studied. For comparison with the LED influence, a traditional light source (incandescent light) and two known harmful light sources (UVA and UVB) were studied together.

The photocytotoxicity of lights on mammalian cells was determined by cell viabilities and LDH leakages in mammalian cells. The results were consistent to reveal that LED and incandescent light could also result in mammalian cell apoptosis, like UVA and UVB. Furthermore, in the interior lighting condition, the cytotoxicities of LED and incandescent lamp on RF/6A cells (rhesus retinal pigment epithelium cell line) were stronger than that on two fibroblast cell lines, while the cytotoxicitiy of UVA and UVB on HS68 cells (fibroblast cell line) was highest in the tests.

It is well known that UVA is a long-wave ultraviolet light with strong penetration, and UVB is a wave medium-wave ultraviolet light with medium penetration. The wavelength of UVA is between 320 and 420 nm, and UVB is between 275 and 320 nm. More than 98% of UVA in sunlight can through the ozone layer and penetrate the clouds reach the Earth's surface, and then reach the dermis layer of skin to destruction of elastic fibers and collagen fibers, tanning of our skin. While, most of UVB in sunlight is absorbed by the ozone layer, only less than 2% can reach earth's surface [23]. UVB can promote the formation of vitamin D and mineral metabolism in human body, but if exposed to UVB for too long time, skin will be tanned, then swelled and peeled [24,25]. The incandescent light contains the whole spectrum and without any intermittent. Except for major of energy located in visible region, the incandescent light is composed of nearly 20% of energy located in ultraviolet and infrared region (Fig. 7a). While, the energy of tested LED is almost completely located in visible region, in other words, nearly no energy is located in ultraviolet and infrared region (Fig. 7b) [26]. So, we deduced that the visible-light might be more harmful to eyes than skin, while the ultraviolet light is opposite. Moreover, the significantly lower photocytotoxicity of LED than incandescent light might be due to the less ultraviolet and infrared light existed in LED than incandescent light in the same illumination.

It is commonly accepted that the formation of free radicals or their reaction products with oxygen and the production of reactive oxygen species (ROS) are responsible for the biologic changes observed in light-exposed tissues [27]. Under normal circumstances, cells are able to defend themselves against ROS damage with enzymes such as superoxide dismutases, catalases, lactoperoxidases, glutathione peroxidases and peroxiredoxins. But, if the balance of ROS generation and elimination is broken, human being will get injured. In the present paper, LED and incandescent light induced oxidation in mammalian cells were firstly confirmed. Like ultraviolet, LED and incandescent light could promote the production of ROS, raise lipid peroxidation level and lower the activity of the antioxidant key enzymes in mammalian cells, and finally cause a number of cells death. However, in the same illumination, the negative function of LED was significantly smaller than incandescent lamp and ultraviolet.

In this experiment, the luminous efficiency of incandescent light is 25 lm/W, while the luminous efficiency of LED is 60 lm/W. Therefore, compared with the traditional light source, LED was much efficient and safer in interior lighting system, whatever for mammalian skin or eyes, and should be widely used instead of traditional light source.

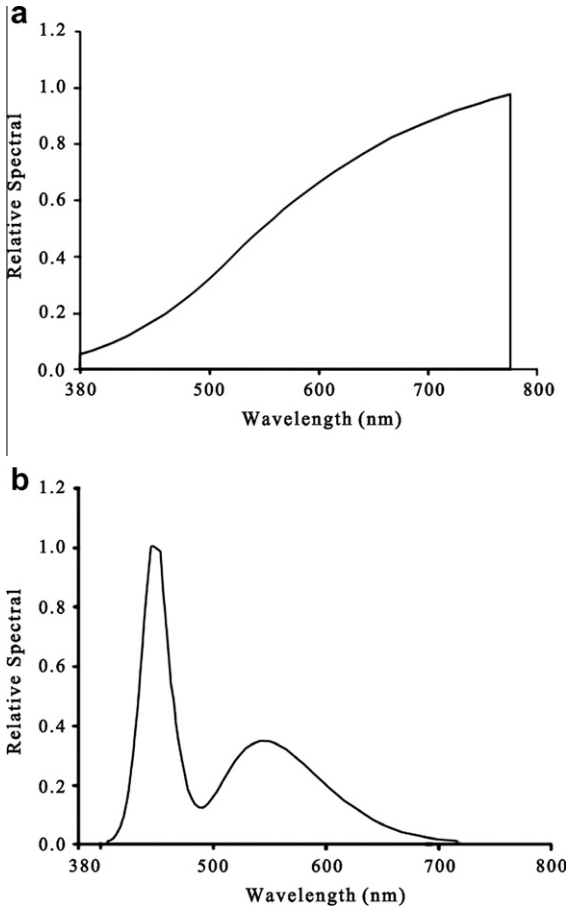


**Fig. 7.** The spectra of tested lights: (a) incandescent light; (b) LED.

## Acknowledgment

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## Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jphotobiol.2012.08.007.

## References

[1] L. Rittie, G.J. Fisher, UV-light-induced signal cascades and skin aging, Ageing Res. Rev. 1 (2002) 705–720.
[2] P.U. Giacomoni, P. D'Alessio, Open questions in photobiology IV. Photoaging of the skin, J. Photochem. Photobiol. B: Biol. 33 (1996) 267–272.
[3] S.A. Jones, F. McArdle, C.I. Jack, M.J. Jackson, Effect of antioxidant supplementation on the adaptive response of human skin fibroblasts to UV-induced oxidative stress, Redox Rep. 4 (1999) 291–299.
[4] P.S. Peres, V.A. Terra, F.A. Guarnier, R. Cecchini, A.L. Cecchini, Photoaging and chronological aging profile: understanding oxidation of the skin, J. Photochem. Photobiol. B: Biol. 103 (2011) 93–97.
[5] G.J. Fisher, S. Kang, J. Varani, Z. Bata-Csorgo, Y. Wan, S. Datta, J.J. Voorhees, Mechanisms of photoaging and chronological skin aging, Arch. Dermatol. 138 (2002) 1462–1470.
[6] J.H. Oh, A.S. Chung, H. Steinbrenner, H. Sies, P. Brenneisen, Thioredoxin secreted upon ultraviolet A irradiation modulates activities of matrix metalloproteinase-2 and tissue inhibitor of metalloproteinase-2 in human dermal fibroblasts, Arch. Biochem. Biophys. 423 (2004) 218–226.
[7] D.C. Agrawal, H.S. Leff, V.J. Menon, Efficiency and efficacy of incandescent lamps, Am. J. Phys. 64 (1996) 649–654.
[8] Y. Uchida, T. Taguchi, Lighting theory and luminous characteristics of white light-emitting diodes, Opt. Eng. 44 (2005) 124003-1–124003-9.
[9] V. Martinez, V. Galbiati, M.P. Vinardell, E. Corsini, M. Mitjans, Establishment of an in vitro photoassay test using THP-1 cells and IL-8 to discriminate photoirritants from photoallergens, Toxicol. Lett. 205 (2011) S166.
[10] S.L. Hempel, G.R. Buettner, Y.Q. O'Malley, D.A. Wessels, D.M. Flaherty, Dihydrofluorescein diacetate is superior for detecting intracellular oxidants: comparison with 2′,7′-dichlorodihydrofluorescein diacetate, 5(and 6)-carboxy-2′,7′-dichlorodihydrofluorescein diacetate, and dihydrorhodamine 123, Free Radical Biol. Med. 27 (1999) 146–159.
[11] H.H. Draper, M. Hadley, Malondialdehyde determination as index of lipid peroxidation, Methods Enzymol. 186 (1990) 421–431.
[12] B. Eftekharzadeh, M. Ramin, F. Khodagholi, S. Moradi, K. Tabrizian, R. Sharif, K. Azami, C. Beyer, M. Sharifzadeh, Inhibition of PKA attenuates memory deficits induced by β-amyloid (1–42), and decreases oxidative stress and NF-κB transcription factors, Behav. Brain Res. 226 (2012) 301–308.
[13] M.E. Anderson, Determination of glutathione and glutathione disulfide in biological samples, Methods Enzymol. 113 (1985) 548–555.
[14] C.H. Tsuei, J.W. Pen, W.S. Sun, Simulating the luminance and the efficiency of the LED and fluorescent lights used in indoor lighting design, Opt. Express 16 (2008) 18692–18701.
[15] I. Seotsanyana-Mokhosi, T. Kresfelder, H. Abrahamse, T. Nyokong, The effect of Ge, Si and Sn phthalocyanine photosensitizers on cell proliferation and viability of human oesophageal carcinoma cells, J. Photochem. Photobiol. B: Biol. 83 (2006) 55–62.
[16] R. Kohen, A. Nyska, Oxidation of biological systems: oxidative stress phenomena, antioxidants, redox reactions, and methods for their quantification, Toxicol. Pathol. 30 (2002) 620–650.
[17] M. Messarah, A. Boumendjel, A. Chouabia, F. Klibet, C. Abdennour, M.S. Boulakoud, A.E. Feki, Influence of thyroid dysfunction on liver lipid peroxidation and antioxidant status in experimental rats, Exp. Toxicol. Pathol. 62 (2010) 301–310.
[18] C. Vandeputte, I. Guizon, I. Genestie-Denis, B. Vannier, G. Lorenzon, A microtiter plate assay for total glutathione and glutathione disulfide contents in cultured/isolated cells: performance study of a new miniaturized protocol, Cell Biol. Toxicol. 10 (1994) 415–421.
[19] I.M. Braverman, E. Fonferko, Studies in cutaneous aging. 1. The elastic fiber network, J. Invest. Dermatol. 78 (1982) 434–443.
[20] J.E. Roberts, Update on the positive effects of light in humans, Photochem. Photobiol. 81 (2005) 490–492.
[21] LED, The American Heritage Science Dictionary, Houghton Mifflin Company, 2005.
[22] I. Moreno, C. Sun, Modeling the radiation pattern of LEDs, Opt. Express 16 (2008) 1808–1819.
[23] S.D. Lamore, S. Azimian, D. Horn, B.L. Anglin, K. Uchida, C.M. Cabello, G.T. Wondrak, The malondialdehyde-derived fluorophore DHP-lysine is a potent sensitizer of UVA-induced photooxidative stress in human skin cells, J. Photochem. Photobiol. B: Biol. 101 (2010) 251–264.
[24] U.P. Kappes, D. Luo, M. Potter, K. Schulmeister, T.M. Runger, Short- and long-wave UV light (UVB and UVA) induce similar mutations in human skin cells, J. Invest. Dermatol. 126 (2006) 667–675.
[25] A. Petrova, L.M. Davids, F. Rautenbach, J.L. Marnewick, Photoprotection by honeybush extracts, hesperidin and mangiferin against UVB-induced skin damage in SKH-1 mice, J. Photochem. Photobiol. B: Biol. 103 (2011) 126–129.
[26] W. Van Bommel, Lighting quality and energy efficiency, a critical review, Light Eng. 19 (2011) 5–11.
[27] E. Kohen, R. Santus, J.G. Hirschberg, Photobiology, Academic Press, San Diego, CA, 1995.