J Cancer Res Clin Oncol (1986) 112: 141–143

Cancer Research
Clinical Oncology

Fluorescent lighting enhances chemically induced papilloma formation and increases susceptibility to tumor challenge in mice

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Summary. To study whether fluorescent lighting at work might increase carcinogenesis, hairless mice were exposed to a bank of six 36 W standard fluorescent lamps (neutral-white) every workday for 8 h at an illuminance level of 1 000 lx. For comparison, other mice were exposed to UVB radiation or to simulated solar radiation.

In experiment A the animals were irradiated for 6 weeks prior to the application of 7,12-dimethylbenzanthracene once and – following an interval of 2 days – for 10 weeks after DMBA application. The number of blue nevi and papillomas was enhanced by exposure to all spectra 10 weeks after chemical tumor induction.

In experiment B the animals were irradiated for 6 weeks prior to the transplantation of UV-induced fibrosarcoma cells from syngeneic mice into the dorsal and ventral skin. Within the following 4 months fibrosarcoma developed in the dorsal skin exposed to the fluorescent lighting and to the UVB radiation, as well as in the non-irradiated ventral skin of 10–20% of the mice.

The results suggest that fluorescent lighting as used in certain work environments may increase carcinogenesis caused by other factors.

Key words: Skin cancer – Fluorescent lighting – Immune surveillance

Introduction

In 1982 Beral et al. reported the association of the exposure to fluorescent lighting at work with a doubling of melanoma risk in man. This study provoked a vehement discussion on fluorescent lighting as a risk factor for developing melanoma (Pasternack et al. 1983; Rigel et al. 1983). Ronchi and Bodmann (1983) concluded that there is no evidence of risk of skin cancer whatever associated with fluorescent light, and that as there is no clear understanding of the causes and mechanisms involved in melanoma, fluorescent lighting cannot be implicated.

The first aim of our study was to examine whether fluorescent lighting, simulating the conditions at work, is able to modify chemical carcinogenesis in mice. In a second experiment we tested the UV-induced susceptibility to tumor callenge to see whether immune surveillance against tumor cell growth can be impaired by fluorescent light.

In comparison to the UVB spectrum of fluorescent lamps (mainly λ313 nm) the mice were treated with equal UVB exposures from a continuous spectrum λ295–330 nm and from a simulated solar radiation λ290–2 500 nm.

Materials and methods

A total of 165 female hairless pigmented mice of the inbred strain hr hr, obtained from Bomholtgard, Ry, Denmark, was divided into 11 groups (Tables 2 and 3). The backs of the animals of groups 1 and 8 were exposed to a bank of six Osram standard fluorescent lamps 36 W/25 (neutral-white) without diffusers at an illuminance level of 1 000 lx. According to the manufacturer's measurements, the irradiance of the UVB range was 12 mW/m^2 and of the UVA range 78 mW/m^2 (mean values), related to 1 000 lx. The distribution of spectral irradiance within the UVB waveband is shown in Table 1. Within 8 h, the mice were exposed to 35 mJ/cm^2 of the UVB radiation from the fluorescent lamps.

Table 1. Spectral irradiance $E_{e\lambda}$ of a standard fluorescent lamp (Osram neutral-white L 36 W/25) after 100 h ageing, at an illuminance level of 1000 lx.

Wavelength λ/nm	<295	297	302	313
Irradiance E_e/mW · m^{-2}	0	0.1	0.4	11.5

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In comparison with fluorescent lighting, the mice of groups 2 and 9 were exposed to 35 mJ/cm² of UVB radiation $\lambda = 295–330$ nm, produced by Philips TL 12 fluorescent sunlamps and filtered through Schott glasses UG 11 and GG 19, each 1 mm thick. The mice of groups 3 and 10 were exposed to 35 mJ/cm² of the UVB portion of a simulated solar radiation $\lambda = 290–2.500$ nm, produced by two unfiltered Osram metal halide high pressure lamps HMI 575 W., the total irradiance amounting to 753 W/cm². The irradiances were measured with a Gamma Scientific Spectroradiometer model 2900 at 6.5 nm band widths. All animals were irradiated on workdays for 6 weeks.

Experiment A. Immediately after the last irradiation, the backs of the animals of groups 1 to 4 were painted once with 0.5% 7,12-dimethylbenzanthracene (15 µg DMBA), dissolved in acetone. The irradiation of the mice of groups 1 to 3 was continued 2 days later up to a total irradiation period of 10 weeks. The mice of groups 5 to 7 were irradiated without painting the backs. All animals were observed weekly for tumors > 0.5 mm in diameter.

Experiment B: At 24 h after the last irradiation 0.1 ml of a suspension containing 7×10^5 UV-induced, cultured fibrosarcoma cells from syngeneic mice, was transplanted into the dorsal and ventral skin of the mice of groups 8 to 11. The animals of group 11 served as nonirradiated controls. All mice were observed weekly.

Results

The results are summarized in the Tables 2 and 3. Fluorescent lighting, 6 weeks before and 10 weeks after DMBA painting, enhanced the growth of DMBA-induced blue nevi and papillomas. This also applied to equal exposures of UVB radiation and the UVB portion of the simulated solar radiation.

Table 2. Numbers of blue nevi and papillomas 0,5 mm in diameter/group of 15 pre- and postiradiated mice, 10 weeks after DMBA induction

Group		Number of blue nevi	Papillomas
1	Fluorescent lighting	150	11
2	UVB irradiation	102	25
3	Solar irradiation (simulated)	321	81
4	DMBA without irradiation	78	0
5–7	Irradiation without DMBA (all radiation sources)	0	0

Table 3. Fibrosarcoma incidences observed 4 months after transplantation of UV-induced fibrosarcoma cells in preirradiated hairless mice

Group		Fibrosarcoma incidences	
		Dorsal skin	Ventral skin
8	Fluoresent lighting	1/15	3/15
9	UVB irradiation	2/15	3/15
10	Solar irradiation (simulated)	0/15	0/15
11	No irradiation	0/15	0/15

Preirradiation with fluorescent light and UVB radiation for 6 weeks, corresponding to a total UVB-exposure of 1.05 J/cm², rendered 10%–20% of the mice immunologically nonresponsive to the transplanted UV-induced fibrosarcoma cells from syngeneic animals. The tumor growth in the nonirradiated ventral skin demonstrated the systemic character of this specific alteration of the immune response. Preirradiation with the simulated solar radiation, also corresponding to a total UVB exposure of 1.05 J/cm², failed to induce nonresponsiveness to the transplanted fibrosarcoma cells.

Discussion

Experiment A showed the enhancing effect of 6 weeks fluorescent lighting "at work" on the number of DMBA-induced papillomas in mice. Different effects of equal UVB exposures from the three modes of radiation may have been due to the different spectral power distributions within the UVB wavebands and to additional heat effects of the "solar radiation" (Freeman and Knox 1964).

The design of experiment A was influenced by the findings of Epstein and Epstein (1962) on the increase of DMBA-induced skin tumors by subsequent exposure to UV radiation as well as by the paper of Stenbäck (1975) on the enhancing effect of UVB preirradiation on the number of grossly observed DMBA-induced tumors. Photodegradation of DMBA (Davies 1978) or activation of the DMBA action by UVA-radiation was avoided by an interval of 2 days between DMBA application and subsequent irradiation.

Experiment A gave no information about the mechanism of tumor enhancement by fluorescent lighting. Stenbäck (1975) discussed the specific effect of UV pretreatment on the immunologic defence mechanisms of the host. This hypothesis is supported by the findings of Roberts and Daynes (1980) on benzpyrene-induced tumors. Three weeks of UV treatment before benzpyrene application to the nonUV-exposed ventral side of each mouse reduced the latency period for tumour induction, indicating a systemic effect of the UV irradiation.

De Gruijl and van der Leun (1983) reported similar results involving daily ventral UV exposure for 4 weeks before tumor induction by dorsal UV exposure. They concluded that a systemic effect impaired immunologic defence against the UV-induced tumor cells, thus increasing the likelihood of tumor initiation.

Experiment B was designed to examine the effect of chronic fluorescent lighting "at work" on immune surveillance (Burnet 1970). Using the technique of Kripke and Fischer (1976), the mice were irradiated for

6 weeks prior to the transplantation of UV-induced fibrosarcoma cells from syngeneic mice. Some 20% of the animals failed to reject the cell transplants from the nonirradiated ventral skin. Instead of this, fibrosarcomas appeared within 4 months. This was also the case after equal exposure to the UVB waveband but not to the UVB portion of the "solar radiation".

In the above cited experiments on the influence of preirradiation on chemically or UV-induced carcinogenesis, fluorescent sunlamps have been used. The major peak of spectral energy of these lamps corresponds to $\lambda = 313$ nm as the predominant wavelength within the UVB portion of the Osram L 36W/25 neutral-white fluorescent lamps (Table 1). Nevertheless the ratio of short wavelength UVB ($\lambda = 295$–305 nm) to long wavelength UVB ($\lambda = 310$–315 nm) is much higher for fluorescent light than for sunlight (Maxwell and Elwood 1983).

The total UVB exposure from preirradiation with fluorescent sunlamps, delivered by Stenback (1975) and by de Gruijl and van der Leun (1983) was in the order of 3 J/cm^2. In the experiments of de Fabo and Kripke (1979, 1980), the susceptibility to tumor challenge was directly proportional to the total UV exposure in the range of 1 to 8 J cm^2 and independent of the dose rate. In the present experiment B, 6 weeks of fluorescent lighting, at the rather extreme illuminance level of 1 000 lx, corresponding to a total UVB exposure of 1.05 J/cm^2, altered the immunologic response to UV-induced tumor cells in mice. Considering the findings of Kripke (1977) and Bowen and Brody (1983), this may also apply for some nonUV-induced tumor cells, particularly to melanoma cells.

The total UVA exposure of 6 weeks fluorescent lighting amounted to 6.75 J/cm^2. This dose is by far beyond the UVA exposure, that produces tumor susceptibility (Morison 1985).

Direct conclusions on the risk of skin cancer in man, especially of melanomas in connection with fluorescent lighting at work are not possible. Nevertheless the findings support the speculation that fluorescent lighting might enhance the growth of nascent melanoma cells arising from other unrelated causes (Kripke 1979).

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Received December 20, 1985/Accepted Juli 7, 1986