MALIGNANT MELANOMA AND EXPOSURE TO FLUORESCENT LIGHTING AT WORK

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Summary In a study of 274 women with malignant melanoma, aged 18–54 years, and 549 matched controls in New South Wales, Australia, reported exposure to fluorescent light at work was associated with a doubling of melanoma risk (relative risk [RR] = 2·1; 95% confidence limits 1·32–3·32). The risk grew with increasing duration of exposure to fluorescent light and was higher in women who had worked mainly in offices (RR = 2·6) than in women whose main place of work was indoors but not in offices (RR = 1·8). The findings could not be explained by the differences in histories of sunlight exposure, in skin or hair colour, or in any other factor. There was a relative excess of lesions on the trunk in the group exposed to fluorescent light at work. 27 men with melanoma and 35 similarly aged controls were studied, and a significant increase in risk was also found: the RB in those exposed for ⩾10 years compared with those exposed for <10 years was 4·4 (95% confidence limits 1·1–17·5). Such an association has not been reported before, but it is plausible and could explain many of the paradoxical features of the epidemiology of melanoma. Until more data accumulate it must, however, be viewed cautiously.

Introduction

ALTHOUGH sunlight is generally regarded as important in the aetiology of melanoma, evidence proving a causative role remains elusive and contradictory. Despite strong suggestions from geographical studies, most surveys of patients with melanoma have found only a weak relation, if any, between excessive exposure to sunlight and the risk of the disease developing.[1–5] Furthermore, in both Australia and Britain, melanoma rates are high among professional and office workers and are lower in people working outdoors.[6–8] In a case-control study, White women aged 18–54 years in New South Wales, Australia, were asked about exposure to sunlight and to fluorescent light while at work. The latter factor seemed potentially important since the spectral emission from fluorescent bulbs often extends into the ultraviolet range,[9] and cutaneous burns, similar to sunburn, and other photosensitive skin reactions have been reported after exposure to fluorescent light.[10–12]

Subjects and Methods

All patients included in the study attended the melanoma clinic at Sydney Hospital. Diagnosis was made by biopsy and the histological features were classified by Prof. V. J. McGovern. The anatomical sites of the lesions were recorded on a standard form.

The study, which was primarily concerned with exploring the relation between melanoma and oral contraceptive use, began in June, 1978. So that we could assess whether such an association might be spurious, we asked about any other factor which we thought might be linked with melanoma. The 300 most recently diagnosed cases in women aged 18–54 years were used as a starting point. 213 of these 300 women were included in the study: 40 had died; 3 were too ill to be interviewed; 16 lived in very remote areas and it was impracticable to find neighbouring controls; 16 could not be traced; 4 refused interview; and 8 were not interviewed for a variety of other reasons. To the 213 "old cases", 74 "new cases" were added: they were 18–54-year-old women with newly diagnosed melanoma who were treated in the melanoma clinic between June, 1978, and December, 1980. For each case, 2 controls were chosen, matched into 5-year age groups. Old cases were matched also by area of residence: the Australian Bureau of Statistics provided a map of the area in which each patient lived, with randomly selected households, indicating where appropriate controls could be sought. Controls for the new cases were chosen from Sydney Hospital inpatients. Since the main purpose of the study was to compare oral-contraceptive use in cases and controls, we did not choose as controls patients with any chronic illness of more than 2 years' duration, any vascular disease or gynaecological disorder, diabetes, gallbladder or breast disease, rheumatoid arthritis, or mental illness. Although these exclusions would not have been made in a study specifically designed to test the relation between fluorescent light and melanoma, they should not introduce bias into this study. The 148 inpatient controls had been admitted because of orthopaedic disorders (25), gastrointestinal disorders (24), ear, nose, and throat surgery (18), genitourinary disorders (16), respiratory disease (11), neurological symptoms (11), benign or malignant disease (10), and a variety of other conditions (33).

Trained interviewers asked the cases and controls questions, from a standard questionnaire, on demographic factors, occupation, exposure to sunlight, and other factors thought to be important in the aetiology of melanoma. Each job lasting 12 months or longer was recorded, together with information about whether the work was carried out predominantly indoors or outdoors, whether fluorescent lighting was present, and whether the fluorescent lights were switched on most of the time or less frequently. The number of years during which fluorescent lighting was reported to have been on most of the time was calculated and taken as the duration of the woman's exposure. For the old cases, exposure was taken up to the date of diagnosis of the melanoma and for the matched-neighbourhood controls, up to a corresponding date. Inquiry was also made about the presence of fluorescent lights and other possible sources of radiation (such as television) in the home.

The questionnaire was coded and the data analysed at the London School of Hygiene and Tropical Medicine. Calculation of relative risk (RR), χ^2 tests for trend, and Mantel-Haenszel adjusted RRs were carried out with the calculator programs of Rothman and Boice.[13] Of the 287 cases and 574 controls interviewed, 12 cases and 21 controls had never worked and appropriate occupational data were missing for 1 case and 4 controls; this analysis is confined to the remaining 274 cases and 549 controls. The exclusions were not from the same triplets, and all our analyses further separated indoor and outdoor workers; matched triplet analysis was therefore impossible, since too few triplets were left for a meaningful analysis after the non-matching ones were eliminated. The findings were similar for old and new cases, so the data for the two groups were combined. In the analyses of subgroups (see Results) the matching by age and place of residence may have been broken. More complex statistical techniques than the ones we used are available to allow for this, but we felt that their use would be unlikely to affect the results.

Results

The age distribution of the cases and controls is shown in table I. The reporting of any exposure to fluorescent light at work was associated with a twofold increase in melanoma risk (RR = 2·1; 95% confidence limits 1·32–3·32). Furthermore, the RR grew with increasing duration of exposure (table II; χ^2 for trend = 9·5; $p<0·001$). Women who reported that they had worked outdoors at any time had a lower overall risk of melanoma than did the other women (RR = 0·9); however, when they were compared with women who had worked indoors but had never been exposed to fluorescent light at work RR increased to 1·8 (table II). Among indoor workers, women who reported that they had worked mainly in offices had a slightly, but not significantly.

TABLE I—AGE DISTRIBUTION OF FEMALE CASES AND CONTROLS

Age (yr)	No. cases (%)	No. controls (%)
18–24	40 (*14·6*)	70 (*12·8*)
25–34	89 (*32·5*)	188 (*34·2*)
35–44	97 (*35·4*)	194 (*35·2*)
45–54	48 (*17·5*)	97 (*17·7*)
Total	274	549

TABLE II—REPORTED PLACE OF WORK AND OCCUPATIONAL EXPOSURE TO FLUORESCENT LIGHT IN FEMALE CASES AND CONTROLS

—	Cases	Controls	RR* (95% CI)
Always worked indoors:			
Never exposed	25	94	1·0
1–9 years' exposure	134	269	1·9 (1·2–3·0)
10–19 years' exposure	80	121	2·5 (1·5–4·2)
⩾20 years' exposure	14	20	2·6 (1·2–5·9)
Ever worked outdoors	21	45	1·8 (0·9–3·5)
Total	274	549	..

*Relative to those working indoors and never exposed to fluorescent lights. CI = confidence interval.

higher risk than those who worked mainly in other places (RR = 1·1). In both groups the risk associated with any exposure to fluorescent light remained, but it was stronger for office workers (RR = 2·6) than for the other women (RR = 1·8), and in both groups the risks increased with longer durations of exposure to fluorescent light (for office workers χ^2 for trend = 4·2; $p<0·05$; and for indoor workers elsewhere χ^2 for trend = 5·2; $p<0·05$; table III).

When the distribution of the sites of the lesions was examined according to type of work and exposure to fluorescent light, the most striking finding was a relative excess of lesions on the trunk in women who had been exposed to fluorescent light ($\chi^2 = 5·3$; $p<0·05$; table IV). The relative excess of lesions on the head and neck in outdoor workers is also noteworthy; however, the number of cases in each category was small.

Data on exposure to fluorescent light were examined further to determine whether the relation might be due indirectly to other factors. Various measures of recreational exposure to sunlight—both long-term and intense short-term exposure—showed no consistent relation to melanoma risk. Stratification by the following factors did not diminish the overall association with fluorescent light: amount of time spent outdoors; main outdoor activity and amount of clothing worn in childhood and at ages 20 and 30 years; history of sunburning on various parts of the body; place of birth; hair colour; skin colour; use of oral contraceptives; and reported frequency of naevi on the body. There was, however, evidence that some of them modified the risk (table V). In general, the RRs tended to be lower in women who had apparently been most heavily exposed to sunlight: those born in Australia, those reporting that they spent a great deal of time outdoors in childhood, and those whose main outdoor activity at age 20 years was described as sunbathing. The RR was greater in women who reported that they had more naevi on their bodies than the average. The only group for which fluorescent-light exposure did not persist as a risk factor was those with red hair in childhood, but the numbers were small. The presence of fluorescent lights in the home was not associated with a rise in melanoma risk (RR = 0·9; 95% confidence limits 0·6–1·6), even when the analysis was restricted to women who had never been exposed to fluorescent lights at work and had never worked outdoors. Nor was there a relation between melanoma and the presence of a television in the home or the amount that it was watched.

Having noted the association between melanoma and fluorescent-light exposure in women, we examined some information collected previously for 27 men with melanoma aged 18 to 56 years and 35 male inpatient controls of similar age. They had been interviewed with a questionnaire similar to that used for women. Interviewing began in June, 1978, but because of lack of resources it was abandoned in February, 1979. Despite the small numbers a significant association with exposure to fluorescent light was found (table VI). The RR of 4·4 associated with exposure for ⩾10 years is larger than that found for a comparable exposure in women. Ever having worked outdoors was, as in women, associated with a slightly, but not significantly higher risk than those never exposed (RR = 2·2). 3 of the 5 male cases not exposed to fluorescent light or exposed for <10 years had lesions on the trunk, as had 8 of the 10 exposed for ⩾10 years (date missing for 1 case) and 5 of the 11 who had worked outdoors.

TABLE III—REPORTED OCCUPATIONAL EXPOSURE TO FLUORESCENT LIGHTS IN WOMEN WHO WORKED INDOORS

—	Cases	Controls	RR
Office workers:			
Never exposed	8	34	1·0
1–9 years' exposure	51	89	2·4
10–19 years' exposure	31	47	2·8
⩾20 years' exposure	6	6	4·3
Other indoor workers:			
Never exposed	17	60	1·0
1–9 years' exposure	83	180	1·6
10–19 years' exposure	49	74	2·3
⩾20 years' exposure	8	14	2·0
Total	253	504	..

TABLE IV—SITE OF MELANOMA IN WOMEN BY REPORTED PLACE OF WORK AND EXPOSURE TO FLUORESCENT LIGHT*

—	Site: no. (%)				Total no.
	Head and neck	Arms	Legs	Trunk	
Always worked indoors:					
Never exposed	3 (*12*)	5 (*20*)	16 (*64*)	1 (*4*)	25
Ever exposed	15 (*7*)	39 (*18*)	112 (*51*)	53 (*24*)	219
Ever worked outdoors	*3 (15)*	6 (*30*)	8 (*40*)	3 (*15*)	20

*Site unknown for 10 cases.

TABLE V—RELATIVE RISK OF MELANOMA ASSOCIATED WITH ANY EXPOSURE TO FLUORESCENT LIGHT

—	RR (95% CI)
Place of birth:	
Australia	2·0 (1·2–3·2)
Elsewhere	4·5 (0·7–30·8)
Amount of time spent outdoors in childhood:	
Most*	1·8 (1·1–3·0)
Little or none	7·3 (1·2–46·6)
Main outdoor activity at age 20 years:	
Sunbathing	1·8 (0·5–6·3)
Other	2·1 (1·3–3·5)
Reported frequency of naevi:	
More than average	4·5 (1·6–12·3)
Average or less	1·5 (0·9–2·6)
Hair colour in childhood:	
Red	0·4 (0·1–1·4)
Blonde	2·9 (1·3–6·3)
Brown or black	2·6 (1·2–5·9)

*"A great deal" or "a fair amount".

TABLE VI—PLACE OF WORK AND OCCUPATIONAL EXPOSURE TO FLUORESCENT LIGHT IN MALE CASES AND CONTROLS

—	Cases	Controls	RR* (95% CI)
Always worked indoors:			
Never exposed or exposed for <10 years	5	14	1·0
⩾10 years' exposure	11	7	4·4 (1·1–17·5)
Ever worked outdoors	11	14	2·2 (0·6–8·0)
Total	27	35	..

*Relative to working indoors and being exposed to fluorescent lights for <10 years.

Discussion

Although a link between exposure to fluorescent lighting and melanoma has not been suggested before, it seemed worthwhile to investigate the link. First, emissions from fluorescent light extend into the potentially carcinogenic range. The actual wavelengths and the intensities emitted vary with the type of lamp, its glass envelope, and other covers; and defects in the tube may increase ultraviolet emissions.[9,14] As well as being associated with cutaneous burns and photosensitive skin reactions,[11,12] fluorescent light has been shown to cause mutations in cultures of mouse embryo cells.[15] Secondly, the incidence of melanoma in office workers, who are likely to be exposed to fluorescent lights for long periods each day, is high.[7,8] This study was primarily designed to elucidate the relation between oral-contraceptive use and melanoma in women, but we had also enquired about a number of factors which might affect melanoma risk. Having noted the link with exposure to fluorescent lights, we tested the hypothesis further using data collected earlier from men. Even with the small numbers, melanoma in men was more strongly related to fluorescent-light exposure than it was in women.

The findings should not be biased. Neither cases and controls nor interviewers knew of the hypothesis, and exposure to fluorescent light had not previously been linked with any disease. Although subjects may not be able to recall exactly whether they worked under fluorescent lighting during their various employments, there is no reason why the cases and controls should differ in their recall and reporting. It is possible that our measure of exposure to fluorescent light actually reflected exposure to some related causal factor. While we cannot exclude such a possibility, we should point out that no factor known to be associated with melanoma could account for the observation; also the various unknown factors which have been suggested, such as air-conditioning, seem more unlikely than fluorescent light to account for it.

Certain observations need clarification, however. First, why did fluorescent light emerge as a stronger risk factor than sunlight? Our failure to incriminate outdoor work or other sunlight exposure in simple analyses is compatible with the findings of others.[2–4] Nevertheless, sunlight exposure is important; working outdoors was associated with an increase in melanoma risk, but only after exposure to fluorescent light had been taken in to account (tables II and VI). It is curious, however, that fluorescent lights appear to be so important, especially since they emit much smaller amounts of "erythemal" ultraviolet light (UV-B, wavelength 280–315 nm) than solar radiation.[9,12,14] Solar radiation produces a smooth spectrum of emissions with a sharp cut-off of wavelengths below 297 nm,[16] whereas fluorescent lights emit a jagged spectrum with peaks at 298, 302, and 313 nm in the UV-B range.[9,12] This difference in the quality of emissions may be important. Another possibility which needs to be considered is that the longer-wavelength UV-A (315-400 nm) is carcinogenic. It is present in larger quantities than is UV-B in the emissions from fluorescent lamps, and it is carcinogenic in animals when used together with chemical photosensitisers.[17] It is not possible, however, even to speculate about the likely quantity of ultraviolet emissions from fluorescent lamps to which the people in our survey were exposed, since it is so strongly determined by the type of lamp, the presence or absence of a plastic cover, and the distance from the lamp. Another inconsistent finding was that the presence of fluorescent lights at home was not associated with melanoma; if exposure to fluorescent lights at work is important, exposure at home would be expected to emerge as a risk factor also. The type of light or the use of plastic diffusers may differ; also fluorescent lights at home may not be left on for long periods of time, but the women were not asked specifically about these points. Clearly they need to be investigated further.

Regular exposure to sunlight induces tanning and thickening of the skin which almost certainly protects the skin from the carcinogenic effects of solar radiation.[16,18] The precise mix of wavelengths in the ultraviolet range may well be important in determining how much tanning and skin thickening, and thus how much protection, occurs. If fluorescent lights were less efficient than sunlight at inducing such habituation responses they might, despite their low energy production, leave the skin more vulnerable to their ultraviolet emissions. Our data hint that this may be so: women who, according to their reports, would have been most heavily exposed to sunlight—those born in Australia, those who spent a great deal of time out-of-doors in childhood, and those whose main outdoor activity was sunbathing—tended to have lower RRs associated with fluorescent-light exposure than did other women (table V). Moreover, the stronger relation of fluorescent-light exposure and melanoma in men than women may also reflect the fact that men's skin is less habituated to sunlight exposure than is women's. In both sexes the relation with fluorescent lights was strongest for lesions of the trunk. For this site to be predominantly affected means that clothing must be permeable to the carcinogenic wavelengths of fluorescent light. Little relevant information is available on this question, but Robertson[16] reported that "Light, summery weaves favoured by the ladies often transmit 50% of the sunburning ultraviolet radiation . . . A businessman's white shirt may transmit 20%". Even if clothing were only slightly permeable to ultraviolet light, it is not entirely implausible that the effects might be greatest on the trunk: it and other covered parts of the body are more sensitive than the exposed parts in their erythema response to ultraviolet light,[19] presumably because the usually covered parts of the body are less tanned and the skin is thinner. Thus, just as people who are most heavily exposed to sunlight seem to be less affected by fluorescent light, those parts of the body most exposed to sunlight might also be least vulnerable.

Perhaps one of the most intriguing aspects of our observations is that the many paradoxes and unanswered questions about melanoma could be explained if fluorescent lights were a risk factor. A major question, which has been asked repeatedly, is why melanoma incidence has more than doubled in the past 30 years throughout the world.[20] Despite many attempts to answer this question in terms of an increase in exposure to solar radiation, the explanation has generally been unsatisfactory.[5] The frequently reported high rate of melanoma among office workers has always been puzzling. It

has been speculated that office workers may be more likely than other workers to sunbathe, but there has never been evidence to support this assumption. Finally, why has the increase in incidence been largely in lesions on the usually covered parts of the body? If fluorescent lighting were an important aetiological factor, many of these issues would be resolved. Fluorescent lighting is being used more commonly throughout the world, especially since the second world war. The frequency of use of many other substances has risen at the same time; nevertheless, the higher frequency of fluorescent-light use coincides with the increase in melanoma incidence. There are other similarities between the two: for example, that fluorescent lighting is associated most of all with lesions of the trunk and least with lesions of the head and neck, is consistent with the trends—the incidence of lesions of the head and neck is rising slowly and that of lesions of other sites is increasing most rapidly.[21] Furthermore, fluorescent lamps are widely used in offices, often as the only source of illumination. According to our data the effects of fluorescent lights are stronger for office workers than others, which might explain the especially high rate of melanoma in office workers[7,8] and their excess of lesions of the trunk and limbs.[8] This is, however, the first report of an association between melanoma and exposure to fluorescent light. The findings should be interpreted cautiously until further relevant data accumulate.

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IMPROVEMENT OF CELLULAR IMMUNITY AND IgA PRODUCTION IN IMMUNODEFICIENT CHILDREN AFTER TREATMENT WITH SYNTHETIC SERUM THYMIC FACTOR (FTS)

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Summary Three children with IgA and IgE deficiency and T-cell defects (two related patients with ataxia telangiectasia and one with common variable immune deficiency) were treated with synthetic serum thymic factor (FTS) intravenously. A reduction in frequency and severity of infection was noted concomitantly with improvement in cell-mediated-immunity tests. Serum IgA, which was absent in two patients, appeared within 4 weeks of treatment and increased significantly in the third patient. Specific antibodies against vaccination antigens appeared for the first time or increased to titres higher than ever before. In two patients, transient interruption of FTS administration was followed by a regression of the immunological improvement, but this disappeared after the treatment was started again.

Introduction

THYMIC hormones are known for their ability to induce the expression of T-cell markers and eventually T-cell functions in lymphoid precursors of athymic subjects.[1] In 1975 Wara et al.[2] reported the effect of a relatively crude thymic extract, thymosin fraction 5, on the lymphoid cells of children with disorders of cell-mediated immunity. Thymosin induced a significant increase in E-rosette formation with sheep erythrocytes both in vivo and in vitro. Advances in the knowledge of thymic-hormone chemistry have made available for clinical use several well-defined peptides including thymosin alpha-1,[3] thymopoietin and its pentapeptide fragment TP-5,[4] and the serum thymic factor (FTS).[5] This last peptide was initially characterised as a circulating thymic hormone and called *facteur thymique sérique* (FTS), but there is now direct evidence of its presence in the thymic epithelium.[6,7] The aminoacid sequence of FTS has been reported,[8] and synthetic FTS has been shown to display the same activity as natural FTS. The potential application of FTS therapy in the treatment of immune deficiencies is suggested by the observation that children with various types of immune deficiency have low levels of circulating FTS.[9–11] We report here the cases of three children with immunodeficiency syndromes who showed significant immunological improvement, including increased IgA synthesis, after receiving synthetic FTS.

Methods

Serum immunoglobulins (IgA, IgG, IgM) were measured by immunonephelometry with specific monovalent antisera (Behring, Marburg, Germany). The lower limit of detection of IgA was 3 μg/ml. Serum IgE was evaluated by radioimmunoassay (lower limit of detection 2 IU/ml). A quantitative immunodiffusion test (Mancini) was used to assay serum IgD (Partigen, Behring). IgG subclasses were assayed in some instances with haemagglutination inhibition.[12]