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A quantitative estimate of melanoma mortality from ultraviolet A sunbed use in the U.K.

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Summary

Background Concern has been expressed for many years in the medical and regulatory literature about the adverse health effects, especially melanoma, from the use of sunbeds for cosmetic tanning.

Objectives To estimate the mortality from melanoma as a result of the use of sunbeds for cosmetic tanning in the U.K.

Methods A model using a Monte Carlo random sampling technique was developed to estimate human ultraviolet exposure to both sunlight and sunbeds, and these data were used to predict the contribution of sunbeds to melanoma mortality in the U.K.

Results The mortality from melanoma due to sunbed use each year in the U.K. is estimated to be about 100 deaths.

Conclusions Sunbed use could be regarded as a relatively minor self-imposed detriment to public health compared with other voluntary 'pleasurable' activities associated with significant mortality, such as smoking and drinking alcohol. While cosmetic tanning using sunbeds should be discouraged, prohibition is not warranted especially as exposure to the sun, which cannot be regulated, remains the major contributory factor to the risk of melanoma.

Key words: melanoma, modelling, mortality, sun exposure, sunbeds

The main aetiological factor for melanoma from solar exposure is believed to be intermittent, intense exposure of skin sites not usually exposed in everyday life.¹ A similar pattern occurs in cosmetic tanning using sunbeds, which commonly incorporate lamps emitting primarily ultraviolet (UV) A radiation. Case-control studies linking sunbed use to melanoma raise the possibility of a positive association² and, although the data are presently inconclusive, some studies have found a dose-response relationship between extent of sunbed use and melanoma risk.³

A history of sunburn has been reported as a risk factor in melanoma.¹ As burning is not a common feature when tanning using UVA sunbeds, proponents of cosmetic tanning have taken this to imply that tanning with sunbeds is safer than in sunlight. Marked reddening of the skin ('sunburn') from sunlight occurs

when the skin has received an unweighted UV (290–400 nm) dose of 15 J cm^{-2} (equivalent to about three times the dose required to produce a just perceptible reddening in unacclimatized white skin) or more. A similar exposure is delivered during each UVA sunbed session⁴ but the reason the skin does not generally burn is because of the low UVB component of most UVA sunbeds ($\approx 0.5\text{--}1.5\%$ of total UV emission⁵) relative to sunlight in which the UVB (290–320 nm) component is 4–6% of the total UV energy in the 8-h period around solar noon during the summertime from temperate to tropical latitudes.

The analysis presented here is intended to give a crude estimate of the plausible impact that sunbeds might have on the mortality from melanoma in the U.K. It is acknowledged that several simplifying assumptions are made, but, nevertheless, the analysis does indicate where sunbeds might rank in comparison with other voluntary risk-taking activities as a detriment to public health.

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Analysis

Exposure to sunlight

Consider exposure of the trunk and limbs (normally sun-protected sites) to sunlight. This would generally occur during recreational exposure on holiday (especially overseas) and on sunny summer weekends. It might be reasonable to assume that individuals who are exposed in this way would do so for between 10 and 40 days per year and for 1–5 h per day. The ambient clear sky UV irradiance in summer months during the 6-h period around noon when this type of exposure is likely to occur is between 4 and 6 mW cm⁻² from tropical to temperate latitudes.⁶ Exposed sites will receive between about 10% and 60% of ambient UV, depending upon activity, posture and shade.⁷

By assuming independence between these variables of time outdoors and percentage of ambient UV at exposed sites, a Monte Carlo random sampling technique was used to estimate the annual solar UV exposure to the trunk and limbs. Monte Carlo methods are stochastic techniques, involving the use of random numbers and probability statistics to investigate problems.⁸ The use of such methods allows more complex systems to be examined than might otherwise be possible by conventional mathematical techniques. Basically, a complex system, in this case exposure of people in the sun, can be estimated by combining a large number of random configurations of the elements (days per year and hours per day exposed; ambient UV irradiance; fraction of ambient UV on exposed sites) that contribute to the overall exposure.

By taking 5000 random samples of each of the above factors that contribute to exposure, it was found that the annual solar UV exposure to the trunk and limbs might vary between 30 and 2000 J cm⁻², depending on an individual's propensity for exposure, with a median value (defined as the exposure exceeded by 50% of the 5000 individual estimates) of about 380 J cm⁻². The median daily dose on those days

when this type of exposure takes place is about 15 J cm⁻², of the order of the UV exposure from a single UVA sunbed session (see below). While a solar UV exposure of this magnitude on unacclimatized skin could result in sunburn, the hyperplasia and tanning that develop during repeated sun exposure would permit this and higher doses to be tolerated without noticeable erythema in individuals who adapt well to sun exposure.

Exposure to sunbeds

Surveys⁴ of the UV emissions from the most common type of sunbed incorporating UVA fluorescent lamps have found UV irradiances varying between 5 and 25 mW cm⁻², with exposure times per session varying from 10 to 30 min. This results in a UV exposure dose per session of between about 4 and 40 J cm⁻², with a median value of about 15 J cm⁻². However, tanning with UVA confers less protection against sunburn induced by subsequent exposure to sunlight than an equivalent tan induced by UVB, which is equivalent to a sunlight-induced tan.⁹

A U.K. survey of just over 6000 adults carried out in 1996 by The Sunbed Association showed that about 7% of the U.K. population use sunbeds each year,¹⁰ with a pattern according to that shown in Table 1. This pattern of usage can be closely modelled by a lognormal distribution with a mean number of annual sessions of 11, and mode of 3. The same survey gave a breakdown of the self-reported sun-reactive skin types of users (Table 2). The data in the final column of Table 2 are from a survey of a representative sample of

Table 1. Sunbed usage in the U.K. from a survey of users carried out in 1996¹⁰

No. sunbed sessions in past 12 months	%
1–10	54
11–20	25
21–50	14
> 50	7

Table 2. The distribution of self-reported sun-reactive skin types in sunbed users¹⁰ and the general U.K. population¹¹

Skin type	No. respondents (%)	
	Sunbed users	General population
White skin that always burns and never tans	798 (12)	220 (12)
White skin that burns at first and tans with difficulty	2027 (29)	600 (32)
White skin that burns rarely and tans easily	2518 (36)	620 (33)
White skin that never burns and always tans	1351 (20)	320 (17)
Brown/black skin	183 (3)	120 (6)
Total	6877 (100)	1880 (100)

almost 2000 U.K. adults¹¹ and show that there is no appreciable difference in the distribution of skin types (and presumably susceptibility to melanoma) between the general population and sunbed users.

By combining sunbed exposure data with patterns of usage, it can be estimated that UVA sunbed users receive an annual exposure from this activity of between 10 and 3000 J cm⁻², with a median annual exposure from sunbeds of about 150 J cm⁻².

Melanoma mortality from sunbeds

If we assume that somewhere between 30% and 80% of the U.K. population engage in sun exposure of their trunk and limbs each summer, and that between 5% and 9% of the population use sunbeds, then the annual population UV exposure (sunlight and sunbeds) for each random sample is the product of the fraction of the population using sunbeds with the average annual sunbed exposure, plus the product of the fraction of the population exposing their trunk and limbs to sunlight with the average annual solar exposure resulting from this. Repeating this calculation many hundreds of times indicates that the annual population exposure from sunbeds as a percentage of the total UV exposure is between 3% and 12%.

The U.K. mortality from melanoma in 1999 was 1640.¹² It has been estimated that 80% of melanomas in Europe are related to sun (UV) exposure.¹³ It can be estimated that of the 1312 (80% × 1640) deaths resulting as a consequence of UV exposure, the mortality due to UVA sunbed use each year in the UK is about 100, with a range of about 50–200, which is based on the assumptions that: (i) the patterns of exposure from sunbeds and sunlight are equally carcinogenic; (ii) the melanomas from these two sources are equally fatal; and (iii) the fraction of deaths due to sunbed use is equal to the population exposure from sunbeds expressed as a fraction of the total population exposure from sunlight and sunbeds. It follows that there is an annual mortality risk of about 0.0025% for the 7% of the U.K. population of 59 million who use sunbeds. A *pro rata* estimate of the attributable incidence of melanoma from sunbed use in the U.K. would be about 370 cases per year of the 6000 or so reported.¹²

Discussion

This analysis has indicated that UVA sunbeds might account for about 6% of deaths due to melanoma each year in the U.K. While it is recognized that some of the

assumptions leading to this estimate are based upon limited data, it is not obvious how the approach could be made significantly more robust. Concern has been expressed for many years in the medical¹⁴ and regulatory¹⁵ literature about the adverse health effects of sunbeds from a qualitative perspective; this analysis is intended to provide a quantitative insight into the magnitude of harm.

Although the relative effect of different wavebands of UV radiation in the aetiology of melanoma remains unknown, there are accumulating data that suggest a potential role for UVA in the pathogenesis of melanoma.¹⁶ An assumption inherent in the analysis is that the action spectrum, or causative UV wavelengths, for melanoma is equal at all wavelengths throughout the UV spectrum. The only data that exist on an action spectrum for melanoma induction are those obtained from irradiating hybrids of a small tropical fish with different wavelengths of UV irradiation.¹⁷ The action spectrum obtained in this study showed that all wavelengths of UV radiation may be important in melanoma, unlike nonmelanoma skin cancer in which the causative wavelengths are largely within the UVB waveband. In estimating the relative effectiveness of different light sources in causing a biological effect, the usual practice is to weight the spectral emission of each light source by the action spectrum for the effect. If this is done using the action spectrum for melanoma induction in fish¹⁷ as a surrogate for human melanoma, then joule-for-joule, UVA sunbeds would be 15% or so more effective than sunlight. This small difference, coupled with the uncertainty of extrapolating an action spectrum obtained in a small tropical fish to humans, does not warrant a dosimetric approach different from that used here.

It was estimated above that the median daily dose to normally sun-protected sites from solar exposure on those days when this takes place is about 15 J cm⁻². This may well be an overestimate as it does not take into account the use of topical sunscreens during recreational sun exposure. Most modern sunscreens provide balanced protection across the UVB and UVA wavebands and so the actual exposure of the skin would be appreciably less. Sunscreens are not used during cosmetic tanning using sunbeds and as a consequence the contribution of sunbeds to the population UV burden, and by implication to melanoma mortality, could be considerably greater than estimated here.

The mechanism for the induction of melanoma by UV exposure is not well understood. Present evidence

suggests that there are at least two, and possibly three, periods that are relevant.¹⁸ UV exposure early in life may give rise to the first mutational step in the development of melanoma and occurrence of benign naevi. There is evidence that exposures within the age range 10–24 years are important in respect of later melanoma development as such exposure may relate to the interaction of further UV exposure with the existence of acquired naevi from childhood exposure. This may be particularly important in respect of sunbed exposure as most users of sunbeds are young women aged between 16 and 30 years.⁵ Finally, further exposure at any age stimulates proliferation of melanocytes. This proliferative stimulus in already mutated melanocytes may promote the late stages in the development of melanoma. It has been argued that UV exposure near to the time of diagnosis may influence the risk of melanoma¹⁹ and, as such, underlines the importance that sunbed use may have as a promotional factor.

That sunbed use will almost certainly result in morbidity and mortality has finally been acknowledged by the sunbed industry.²⁰ However, the social desirability of a tanned skin remains apparent and people will indulge in cosmetic tanning for a number of reasons, notably to acquire a preholiday tan, to improve their perceived attractiveness and to feel healthier.^{4,5} In a similar way, people will indulge in other voluntary and 'pleasurable' activities that are associated with a detrimental effect on health. Of the 3–4 million Britons who use a sunbed, this analysis has suggested that possibly 100 or so might die each year as a direct result. Compared with smoking, which claims over 120 000 lives each year in the U.K.,²¹ and alcohol consumption, where there are estimated to be between 5000 and 40 000 alcohol-related deaths in England and Wales each year,²² sunbed use could be regarded as a relatively minor, self-imposed, but occasionally fatal, detriment to public health. So while cosmetic tanning using sunbeds should be discouraged, prohibition is not warranted especially as exposure to the sun, which cannot be regulated, remains the major contributory factor to the risk of melanoma.

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