
The Skin Cancer Cover-up

Every summer we're warned that the sun can kill. In fact, most sun-provoked lesions are benign, and not really cancers at all. A clinical scientist writes.

by Professor Sam Shuster

Mankind and the sun have successfully maintained their unequal partnership for some considerable time. We owe our existence to it, and Darwinian genetic and social evolution long ago taught us how to cope with the quiddities of that existence and turn them to our advantage. For example, our bodies have developed the ability to use the sun for the production of vitamin D essential for our bones, and certain immune functions. That ability is passed on by the safe hand of genetic evolution, which is not subject to the vagaries of its social counterpart.

Unfortunately our attitude to sun and ultra-violet (UV) light is subject to much perverse and dubious technical 'advice', which society has passively accepted without questioning its provenance. Whatever the subject, there is always a guru: there must be experts on the best way to tie shoelaces. To test this assertion I asked Google, and found 16,500 sites purporting to give the best way to tie shoelaces! The problem is that there are now so many gurus on the dangers of sunshine that their shadow is obliterating the sun and our long-learned understanding of how to live with it.

What is skin cancer?

We are told that we must severely limit our exposure to the sun and suntan lamps. If we must take a holiday where there is an opportunity to savour the delights of sunshine we should avoid it as much as possible. The middle of the day should be considered dead time to be spent in the shade outdoors or indoors reading improving books. We should wear wide-brimmed hats, long-sleeved shirts or blouses, and cover legs, and we must not forget to cover ourselves with expensive, properly ranked, sun-protective creams and lotions. As for the children: on the few precious occasions when the clouds of a British summer evaporate, we must not allow them out of doors before slapping on sticky sunscreens, bullying them into sweaty hats and clothes made with high sun-protective fabrics. The reasons given for this punitive catalogue of 'don'ts' is that sun exposure ages the skin, and causes cancer. Yet most things we do have risks: what matters is the consequence of that risk, which depends upon the frequency and duration of exposure. Both have been grossly exaggerated for UV and its effect on the skin.

The rejuvenation of ageing skin is a money-spinner. There is no doubt whatsoever that exposure to UV irradiation, particularly by UVB (the shorter wavelength that causes sunburn, but doesn't travel through window glass), gives skin a weather-beaten look, as does smoking. How long this takes and its severity depends on the dose of sun (or smoking) and your genetically determined response to it. The causal damage is to skin collagen, but this is only partly understood. We know that UV promotes molecular cross-links between collagen fibres, making them less elastic, but we do not really know the consequences of this process. While many believe that the weather-beaten 'Marlborough Man' look justifies giving up smoking, sun exposure is different because, as we shall see later, there are trade-off benefits with other bodily functions. However, this particular sun and smoking effect has nothing to do with the ageing process.

The fundamental defect of skin ageing is loss of collagen, the skin's main constituent, which is why ageing skin thins. The loss is one per cent a year throughout adult life and is equal in men and women. The reason female skin appears to age faster than male is that women have less skin collagen. This unfair difference is equivalent to 15 years of ageing! The loss of collagen with age is genetic; it has absolutely nothing to do with UV irradiation and occurs equally in skin that has spent its life covered or exposed. And, contrary to the advertising blurb for anti-ageing creams - which simply irritate the skin producing inflammation that swells the skin and conceals the wrinkles - nothing is known that reverses this loss of collagen. Ageing of the skin is not due to UV and it cannot be overcome by the products of the cosmetic industry.

Skin cancer is the big scare; it is the main plank of the warnings that have come from government bodies. The case that is made is that skin cancer is the commonest of all cancers and its increasing incidence is casually associated with solar irradiation. These facts are correct but they have been mischievously interpreted to scare us into self-inspection, attendance at special skin clinics and a masochistic, oppressive and totally unnecessary, regimen of prophylaxis. Indeed, the very word 'cancer' is being deliberately used to create fear and coerce a public acceptance of these measures. Yet the key fact is that about 95 per cent of skin cancers are basal or squamous cell epitheliomas (in a ratio of about five to one) and although they are called 'cancers' they are functionally benign; they do not spread from the skin and kill. Most are just a centimetre in size; local excision is 95-99 per cent successful; residual microscopic pieces of tumour disappear by themselves and the few recurrences are easily removed. The exceptions are rare and often the consequence of some other diseases.

So while 'skin cancer' is certainly the commonest cancer, the more honest statistic is that skin cancer is the least dangerous cancer; it lies at the very bottom of the mortality table.

So the problem of 'skin cancer' shrivels as soon as you start to examine it, because the vast majority of these lesions are benign. The problem is technical: these benign epitheliomas are classified as cancers from a particular appearance under the microscope, not from their behaviour. The public, for whom the word cancer creates fear, does not understand this. While it may be technically correct to say that skin 'cancer' is related to sun exposure, this is meaningless, because these sun-provoked lesions are not really cancers: they are just small, local, slow-growing and above all benign. These trivial benign lesions cannot possibly justify the aggressive hue and cry about avoidance of UV exposure.

The misunderstanding has been inappropriately talked up by the Australian experience. The high incidence of skin cancer in Australia is the product of a high UV exposure in a population whose ancestors included many with pale, freckled skin and red hair. It should not be extrapolated to different populations living in sun-deprived climates.

But if 'skin cancer' is the bait, melanoma is the hook. Melanoma is the least common of the three skin cancers. There is an alleged increase in its incidence and this is blamed on UV. People have been terrified into inspecting their skin regularly, even though it is of doubtful value. Most of us have simple moles and even more have seborrhoeic warts, which enlarge, get darker, itch and bleed in the same way as melanomas. Dermatological clinics are overfilled with patients worried about these totally innocent spots. Malignant melanomas are not found often enough to justify the hoo-ha about early screening and there is no good evidence that screening saves lives.

Changing diagnosis

We need to have definite answers to two questions: is the increase in melanoma real, and what is its relationship to UV? Sadly, the answer to both questions is uncertain.

Certainly, there has been a big increase in reports of melanoma; the problem is that what is now being called melanoma may be nothing of the sort: it seems to be due to a reclassification of what constitutes malignancy. The diagnosis of malignancy in a melanoma is subjective; it's in the eye of the histopathologist looking down a microscope. In the past it was commonplace for histologists to report borderline, minimal or dubiously suspicious histological appearances of moles. Experience of outcome of these cases taught us that it was not alarming; we did nothing and nothing untoward happened to the patients.

Later, as compensation claims began to dictate a more defensive practice, this led to the very same lesions being labelled suspicious, without the qualification of dubious. The process moved on, and it didn't take long before brown spots previously labelled benign acquired a new label indicating the possibility of early malignant change. In time this moved on again to probability and finally to certainty. The moles have not changed but the diagnosis has.

Having seen the process evolve, I have no doubt that the re-labelling of benign lesions as malignant is a major, if not the main cause of the increased incidence of reported malignant melanoma. I had confirmation of this from well-known clinicians who had observed the same development in other countries. But an idea

is nothing without testing, and to put it to the test I proposed to send copies of the histology slides of moles that were labelled benign years ago, from patients found by follow-up not to have had a malignant melanoma, to a panel of histopathologists for their diagnosis by today's criteria. No laboratory would agree to take part in the study; although they agreed with its design they appeared fearful of its outcome.

Support for this thesis comes from a variety of sources. The most important is that while the incidence of melanoma has increased it has not been accompanied by a corresponding change in mortality. In the UK the annual number of melanomas in women increased by 250 per cent between 1980 and 2002, but mortality increased by just under 30 per cent and is decreasing. The reason for the apparent improvement is not that we have more effective therapy, but that the number of cancers has been swollen by the new wave melanomas. These have a cure rate of 100 per cent because they were never malignant in the first place; they are paper malignancies, benign moles reclassified!

There are other explanations for the diagnostic confusion: for example, it is possible that UV, which is known to increase the number of moles, also induces changes that lead to them being classified as atypical, the jargon name for the features on which the histological diagnosis of malignancy may be based. It has been found that death from melanoma is lower in the higher social classes. Does this mean that the genetic defect that causes the cancer is class-related? This is obvious nonsense; the more likely reason is that the middle classes always turn up first and flock to the clinics with their benign moles which they have been frightened into having removed, and some of these are labelled malignant when in practice they are really benign. Until we have better diagnostic criteria it is impossible to determine if the reported increase of malignant melanoma is genuine. The case for an increase in the prevalence of truly malignant melanoma remains unproven.

The role of UV

Even more doubtful is the role of UV as a causal agent. The evidence is fragile and certainly does not justify the present anti-solar terror campaign. What we might expect if UV really caused melanomas is illustrated by the skin epitheliomas. These cancers are caused by UV. They can be easily induced by UV in laboratory animals, and in the case of epitheliomas there is an excellent correlation between their prevalence in patients, the latitude at which they live and between the site at which they occur and areas of the body exposed to UV.

None of this is true of melanomas. Melanomas are difficult to produce experimentally, the correlation with the latitude at which the patients live is marginal, and their site of occurrence does not correspond to the intensity of its UV exposure. They are commonest on the trunk of men, the legs of women, and the soles of the feet of Africans, a phenomenon not to be explained by exposure to the sun's rays. Their reported increase has been much less than the UV-related skin cancers and, unlike epitheliomas, there is no evidence that sun screens prevent them from occurring.

The problem with melanoma, as with many other branches of contemporary clinical research, is that it is based on circumstantial evidence obtained from epidemiological studies rather than an understanding of the pathology. Melanoma is an illustration of the muddle introduced by uncritical acceptance of epidemiology with its almost random generation of unhelpful numbers. A preoccupation with epidemiology has distracted us from the essential biology. For example, we still need to establish the melanoma's cell of origin. Many think it starts in the pigment cell, the melanocyte, but it may start in the 'naevus' cell of the ordinary 'mole'. Establishing this is vital to our understanding because we know the distribution of moles but not naevus cells over the skin surface, let alone what makes them go malignant. It is well established that UV damage to DNA can produce cancer; but the only sensible conclusion from all the studies to date has to be that while this effect plays a major role in producing epitheliomas, at worst it can only be marginal for melanomas.

The evidence on the effect of UV on the skin is surprisingly clear: it has no effect on skin ageing, which is due to thinning of the skin and loss of collagen, although UV does give the same weather-beaten appearance that is caused by smoking. While UV is the main cause of epitheliomatous skin cancers, which are functionally benign, there is no hard evidence that UV is the principal cause of malignant melanomas.

Nature's own sunblock

What then should we do about UV exposure and sunscreens? The short answer is that in moderate climates like the UK, apart from avoiding sunburn and staring at the sun, it doesn't matter what we do, because the risk of exposure is trivial. Of course, children have to learn how much sun they can take without burning, and their parents need to ensure they get a gradual UV exposure in order to achieve a protective tan (that is more important in children with ginger hair and freckles, most of whom will need to take care not to burn throughout adult life). In the UK, there is no point in trying to minimise sun exposure to avoid skin cancer because our sun is usually too weak to be a danger. Although sunscreens will reduce epithelioma formation they have not been shown to prevent melanomas. The use of a sun blocker in countries such as the UK could be harmful, by impairing Vitamin D synthesis in the skin, causing a risk of osteoporosis.

We still have a lot to learn about what may be the silent benefits of sun exposure. We do not know the significance and purpose of the profound changes in immune mechanisms, the extraordinary improvement in mood and the alleged decreased risk in bowel and prostatic cancer experienced after sun exposure. We may do more harm avoiding these advantages than anything we might gain from the uncertain benefits of sun avoidance.

But not all of the sun's benefits are uncertain, particularly the protective effect of a suntan. Since there is some epidemiological evidence to suggest that sunburn in children may be more harmful later in life, parents have been told that sun exposure must be avoided in childhood. However, if you take a close look at people who were sunburnt as children, you will see areas of white skin that doesn't tan because the pigment cells have been lost by the sunburning. Such skin will always be oversensitive to sun. It is evident that the original sunburn, and subsequent damage, would have been less had there already been a protective tan.

Excessive avoidance and UV screening is a danger because it does not allow a tan, nature's own sun block, to develop and as a result exposure is likely to cause sun-burn. The dogma, now fossilised in print, is that any tan is a sign of skin damage. Tell that to Darwin. Pigmented melanocytes in the skin are a system that protects it from excessive UV, which evolved long before the advent of sunscreens. Even if there was hard evidence that melanoma was UV-induced it would be all the more important to keep a protective tan.

It must now be evident that the effect of the sun on the skin is in desperate need of illumination, and that the prophylactic message, particularly on melanoma, is unreliable. By presenting the fragility of the case against the dangers of UV I hope I will provoke consideration of real cause of melanoma.

Sam Shuster is Emeritus Professor of Dermatology at the University of Newcastle Upon Tyne, and Honorary Consultant to the Department of Dermatology, Norfolk and Norwich University Hospital. This is an edited version of a chapter in "Panic Nation? Unpicking the myths we're told about food and health" edited by Stanley Feldman and Vincent Marks. This excerpt appears on www.spiked-online.com. Buy this book from www.amazon.com.