Spectrum

Sun, skin, and solaria

Sitting in the sun has complicated results as far as our skin is concerned. Work by Dr Adriana Scheibner of the Melanoma Unit at the Royal Prince Alfred Hospital, Sydney, and Mr David Hollis of CSIRO's Division of Animal Production confirms that ultraviolet light affects the skin in many ways — doing much more than simply giving the Caucasians among us a bronze glow!

The unusual collaboration between a dermatologist and an animal scientist came about because Mr Hollis had for years been studying the ultrastructure of sheep skin — in relation to wool growth, and the biological de-fleecing of sheep — and so was an expert on its structural aspects. He contributed his skills in the preparation of skin tissue for electron microscopy and in the examination and interpretation of the results.

By now most Australians know that too much exposure to the sun can lead to skin cancer in those of European origin, especially in the more fair-skinned among us. But the notion that a bronzed skin is always healthy still persists; many people therefore want to carry a year-round tan, and to acquire it without increasing their risk of skin cancer.

Such a person may visit solaria, or tanning centres, which advertise that they provide a 'safe' type of UV light. What in fact they mean is light with only very small quantities of UVB (wavelength 280–320 nm), the type known to cause cancer by direct damage to DNA (see *Ecos* 48, p. 19). Light in the UVA band (wavelength 320–400 nm) had been thought of as relatively harmless.

Sunlight is predominantly UVA, but contains sufficient UVB (about 1%) to cause sunburn and cell mutations in unprotected skin. Solaria provide light with far less UVB, hence their claim of safety.

It so happens that the molecule of DNA strongly absorbs light of the UVB band, and the absorption of this energy can damage the DNA, leading to mutations. Being absorbed to a far lesser extent, UVA light causes fewer mutations in cells than an equivalent amount of UVB light, but it does seem to penetrate more deeply into the skin and may cause damage of its own.

Dr Scheibner and Mr Hollis, in collaboration with Professor William McCarthy and Professor Gerald Milton, also of the Prince Alfred Hospital, examined the effects that both sunlight and the UV light of a commercial solarium have on two types of cell in the epidermis of the human skin. Their findings take us into the frontier realms of the immune system and cancer.

The main purpose of our skin is to protect us from a variable and often hostile environment. It's also involved in other functions such as perception, temperature control, and signalling. (Blushing, blanching, and certain types of sweat are all signals to other individuals.)

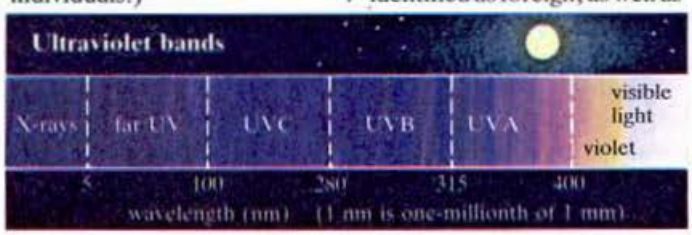
Apart from lying on beaches, many other Australian pastimes involve exposure to the summer sun.

The skin's outer part, or epidermis, comprises several layers including a surface one of dead cells, which constantly flake off, to be renewed from the living cells beneath. But the epidermis as a barrier is far from perfect. Bacteria try to gain entry through hair follicles and sweat glands, and will also establish a foothold — albeit a limited one — any time our skin is cut.

So it's quite clear that the skin needs to be well supplied with defences against marauding microbes in order to deal with any invasion of the body before this can reach vital internal organs. The body's immune system is responsible for defence against everything identified as foreign, as well as for internal surveillance against its own cells that might have mutated into a different and often damaging form — usually cancerous.

In the epidermis, the immune system is represented mainly by the Langerhans cells, named after the man who first observed them. (He also gave his name to the islets of insulin-producing tissue in the pancreas.) These cells, about 30–50 μm across, are born in the bone marrow and migrate into the skin from the blood. They form the outer barrier of the immune system and their job is to recognise foreign antigens — that is, compounds that are not normally part of an individual.

Antigens may enter on a micro-organism, or may be on one of the body's own cells that has mutated into a potentially cancerous state, and so needs to be 'dealt with'. When Langerhans cells recognise a non-self antigen, they 'present' it to T-lymphocytes — a class of white blood cell involved in rejection of foreign



Ultraviolet light extends in wavelength from 100 nm to 400 nm and lies between X-rays and the violet end of the visible light spectrum.

antigens and destruction of any cells bearing them.

Many studies have shown the importance of the Langerhans cells for the functioning of the immune reponse in the skin; if these cells' activity is depressed, animals will not respond to various highly antigenic substances put onto or introduced through their skin.

Dr Scheibner's studies with human volunteers have shown that exposure to UV light actually decreases the number of Langerhans cells in the skin. Her subjects received half an hour of solarium light or sunlight daily for 10 or 12 consecutive days. (Those participating in the solarium study did not sunbathe during that period.)

Using local anaesthetic, Dr Scheibner took skin shaves before exposure, after the period of exposure, and again after 2 weeks. The scientists examined the epidermis (upper layer) of the skin under a microscope, and subjected melanocytes (the cells that make the tanning pigment melanin) and Langerhans cells to biochemical reactions, to make them visible so that they could be counted. Additional studies with the electron microscope revealed the internal structure of these cells.

In the experiments, UV light affected the functioning of remaining Langerhans cells. Each immunologically competent Langerhans cell has a special chemical compound attached to its outer membrane; if this receptor (termed HLA-DR) is missing, the cell can no longer present antigen to T-lymphocytes and thereby initiate the process of antibody production against an offending antigen.

In the subjects studied, UV light caused quite a reduction in the number of Langerhans cells that possessed HLA-DR. Dr Scheibner and Mr Hollis found this in all the volunteers — both those who sunbathed and those given the solarium type of UV light.

Of course, absolute comparisons between the two results would be artificial because a dose of sunlight cannot be as accurately controlled as light from a solarium. However, the solarium light, despite its predominantly 'safe' UVA wavelength, caused a reduction in Langerhans cell numbers of the same order of magnitude as did sunlight.

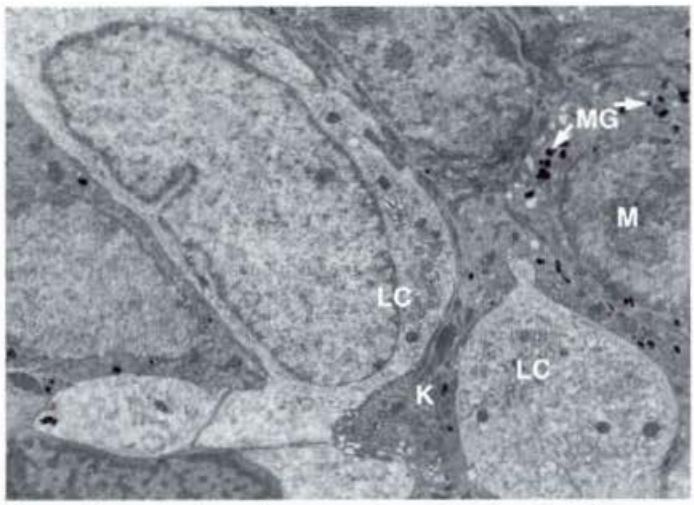
The significance of all this is quite clear: the immune response is vital in, among other things, helping to prevent cancer developing anywhere in the body. It does this by 'secret-police' methods of quick and efficient elimination of any cells that become slightly 'deviant' because of a mutation or viral infection changing not only their behaviour (cancer cells multiply when they shouldn't), but also the chemicals on the membrane that identify that cell as belonging to your body.

Now, if the Langerhans cells are depleted or unable to work efficiently, then potentially cancerous or pre-cancerous cells will not be identified or removed. And of course, exposure to light in the UVB band itself can cause cells to change into a possibly cancerous state by damaging their DNA.

So sunlight mounts a two-pronged attack on skin — both causing mutations and reducing the efficiency of the defence against them — which is why cancer of the skin is so common in Australians of European descent.

Sunlight is one thing; UV from a solarium is a little different. Is it safer? More work needs to be done before we can answer the question fully.

The fact that Dr Scheibner and Mr Hollis have found a reduction in the number of competent Langerhans cells in the skin suggests that solarium light is not quite as innocent as had been presumed. Also worrying is their finding that the number of HLA-DR Langerhans cells remained below normal for 2 weeks after the exposures. Whether or not this represents serious damage will, it is hoped, emerge from further studies.



Seen with an electron microscope (magnification × 6500), a Langerhans cell (LC) from human skin weaves behind a keratin-producing cell (K). A melanocyte (M) on the right contains dark granules of melanin (MG) produced in response to exposure to UV light.

The acquisition of a sun-tan involves the production of melanin, a dark pigment that absorbs much of the UV. It seems probable that this would protect against many of the damaging effects of sunlight or solarium light.

Dr Scheibner found that, among the European subjects in her study, those who had had some recent previous exposure to the sun (sufficient to give a light tan) still recorded damage to their Langerhans cells. In fact, after the exposures they had even fewer epidermal Langerhans cells than did untanned subjects.

However, Dr Scheibner argues that this does not necessarily mean you should visit a solarium in a raw white state, because such subjects showed a rapid multiplication of, and a great increase in, the number of melanocytes — the cells that produce the pigment melanin. (These changes were manifestations of the skin's attempt to tan quickly.)

The importance of this is that the faster cells multiply, the greater is the chance of a cancer cell popping up in error. Untanned people exposed to the solarium light may therefore run a greater risk of a melanoma — or cancer of the melanocytes — than previously tanned people.

In tanned subjects, Langerhans cells dropped to lower levels than those in the untanned because of the detrimental effects of their recent previous exposure to the sun, after which their skin had not had time to recover before receiving the solarium light in the experiment.

It remains true that most white people can protect themselves against some of the sun's effects by acquiring a tan, and going about it in the correct way, although there is no doubt that the more often white people are exposed to the sun, the greater their chance of developing skin cancer becomes.

If you nonetheless still wish or perhaps need to acquire a tan, Dr Scheibner recommends gentle exposure to the sun for, say, 15–20 minutes per day for 3 or 4 days in a row, followed by at least 3 days of no exposure before venturing out again. The 'rest' period gives time for the Langerhans cells to start recovering, and so minimises the time when the skin

immune system is in disarray.

This slow intermittent way is akin to the natural process of sun-tanning that would have occurred in pre-historic European peoples as the summer began.

European skin is designed to afford its wearer protection against the quantity and strength of sunlight found in Europe. A white person's tan, no matter how deep, is inadequate protection against long exposure to tropical sun, and such people must recognise their biological limitations. The many Australians of European descent (unless they are Tasmanians) must admit to themselves that they were not designed for the latitudes in which they are now living.

Europeans have fewer melanocytes than Aborigines, and produce a type of melanin that is probably less efficient. Full-blooded Aborigines can spend many hours in sunlight (although even they may eventually burn), and skin cancer is virtually unknown among them, apart from on the soles of the feet.

For many Australians, maintaining a life-long sun-tan — however acquired — is dangerous. Perhaps we should admit that the so-called glamour of a sun-tan is only skin deep.

Roger Beckmann

Effects of exposure to ultraviolet light in a commercial solarium on Langerhans cells and melanocytes in human epidermis. A. Scheibner, D.E. Hollis, W.H. McCarthy, and G.W. Milton. *Australian Journal of Dermatology*, 1986, **27**, 35–41.

Effects of sunlight exposure on Langerhans cells and melanocytes in human epidermis. A. Scheibner, D.E. Hollis, W.H. McCarthy, and G.W. Milton. *Photodermatology*, 1986, **3**, 15–25.