In This Issue . . .

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UVA and UVB Have Nearly Identical Biochemical Effects on Melanocytes

Although ultraviolet A radiation causes immediate tanning that can persist and ultraviolet B causes tanning only after a delay, the two types of radiation may have the same effects on melanocytes, according to Cheryl Rosen, Yoshihito Seki, William Farinelli, Robert Stern, Thomas Fitzpatrick, Madhu Pathak, and R. William Gange of Massachusetts General Hospital. The group reports its results in this issue.

The investigators decided to use monochromatic UVA and UVB of narrow bandwidths in order to avoid the middle wave lengths, around 320 nm of radiation, where the two types of radiation co-exist in varying proportion. They then looked at the specific effects of UVA and UVB on human melanocytes in vivo.

Both UVA and UVB caused similar changes in the morphology of human melanocytes—the melanocytes became more dendritic and the cell bodies increased in size—and both caused the melanocytes to proliferate. The enzyme tyrosinase, an indicator of increased melanin production, was not active at all at day 1 after exposure to UVA or UVB. But tyrosinase was active at day 7 and its activity diminished by day 14, the researchers found.

Rosen points out that, "until recently, people thought UVA was harmless, but now many groups are beginning to see harmful effects. We've shown that UVA *can* be harmful. It stimulates melanocytes—it doesn't just darken preexisting melanin." When the investigators analyzed their results, says Rosen, "we were really struck by the similarities between the effects of UVA and UVB." The explanation of the clinical differences between the two radiations may lie in the fact that UVB immediately induces melanocytes to proliferate, whereas UVA stimulates them only after a delay. But esssentially, says Rosen, "UVA and UVB have the same biochemical effects."

How Langerhans Cells Reappear After Injury

For several years, researchers have noticed that Langerhans cells in the skin appear to decrease in number after the skin is injured by ultraviolet light. But it has not been clear how these UV-damaged Langerhans cells repopulate the skin. In this issue, Sunji Miyauchi and Ken Hashimoto of Wayne State University School of Medicine provide an explanation.

By measuring ATPase activity, a cell-surface marker for Langerhans cells, Miyauchi and Hashimoto found previously that Langerhans cells decreased in number after a high dose of ultraviolet B irradiation. Then, within 60 days, the Langerhans cell population recovered. It was possible, according to Hashimoto, that the cells could recover by repairing their damaged cell membranes, or new Langerhans cells could migrate from the bone marrow to repopulate the epidermis. Alternatively, the affected Langerhans cells could undergo mitosis.

To resolve the issue, Miyauchi and Hasimoto irradiated mouse ear epidermal sheets and followed the recovery of Langerhans cells using electron microscopy. The Langerhans cells that survived the UV irradiation first grew very large—the cell volume almost doubled. Then these cells underwent mitosis. Moreover, Hashimoto says, "when we looked at the dermis, we saw no migration of Langerhans cells from the blood through the dermal blood vessels." The new Langerhans cells in the UV-treated epidermis, in other words, did not come from the bone marrow.

The significance of this work, according to Hashimoto, is that mitosis in Langerhans cells of the epidermis has seldom been reported, although it is clearly a rapid and efficient mechanism for repopulating the cells. "I am not sure if the bone marrow is capable of responding in adult life to repopulate UV-damaged epidermis as they do in fetal skill," Hashimoto says.

Investigating a Possible Fibroblast Defect in Epidermolysis Bullosa

A few years ago, it was reported that cultured dermal fibroblasts of patients with recessive dystrophic epidermolysis bullosa have a biochemical defect that renders them unable to effectively contract a collagen lattice. This finding intrigued a group of researchers at Washington University School of Medicine who wanted to know if this defect was a general finding in cells from patients with epidermolysis bullosa, and, if so, what abnormality in the cells from patients with erythema bullosa causes this defect. The group, which consists of Arthur Eisen, Alice Pentland, Eugene Bauer, and Gregory Goldberg, reports its results in this issue.

"We were interested in studying fibroblast-populated collagen lattices," says Pentland, because these collagen lattices appear to be involved in such things as wound repair, clot retraction, and perhaps tumor invasion. It is known that fibroblasts contract collagen gels in vitro, and may contract them as much as 50-fold. So it was feasible to test the hypothesis that fibroblasts from patients with all types of epidermolysis bullosa do not contract these lattices very well due to the presence of a common genetic defect.

The investigators found, however, that the ability of the epidermolysis bullosa fibroblasts to contract the lattice was inconsistent. "We checked all the EB cells we had," says Pentland. "Some cells contracted the lattices poorly and some contracted normally." Poor contraction of a collagen lattice "was not a specific or consistent finding associated with EB," she says.

Pentland and her colleagues suggest that there may be a defect

0022-202X/87/$03.50

in lattice contraction in patients with EB who have had repeated skin injury. If so, their finding, although not as general as they had hoped it would be, nonetheless may help them to understand more about how injury and trauma affect cell interactions with the surrounding matrix.

Which Patients Benefit From Tunable (577 nm) Pulsed Dye Lasers?

Tunable pulsed dye lasers, which have a wavelength of 577 nm, cause highly specific damage to the microvasculature. For this reason, they are now being used to treat various vascular abnormalities and tumors, such as portwine lesions and telangiectases. But no one had established whether the effects of yellow pulsed dye lasers vary depending on the amount of pigment in a patient's skin. Arthur Tong, Oon Tan, James Boll, John Parrish, and George Murphy of Boston University School of Medicine, Brigham and Women's Hospital, and Massachusetts General Hospital therefore decided to determine if or how pigment may change the effects of this type of laser. They report their results in this issue.

The investigators used a pulsed dye laser to irradiate the buttocks of persons with very fair (type I) skin or persons with dark (type V) skin. In fair skin they found that most of the laser light was absorbed by the superficial blood vessels and there was very little damage to the epidermis. In contrast, this type of laser induced considerable damage to the epidermis of persons with dark (type V) skin—so much so that the irradiated skin actually blistered. The researchers suggest that red blood cells are the preferred target for the pulsed dye laser in fair skin and that melanin is the preferred target in dark skin.

Tong concludes that since the pulsed dye laser, when irradiating dark skin not only damages the epidermis but also the blood dermal vessels, it provides no advantages over other types of laser for these patients. It is useful, however, in the treatment of appropriate skin lesions in patients with fair or hypopigmented skin.