Continued from p. 361

primarily UVB rays. The first sunscreen developed, para-aminobenzoic acid (PABA), fell out of use because it stained clothing and was found to cause allergic reactions in some people. Last year, FDA approved avobenzone, also known as Parsol 1789, as a UVA absorber.

Other substances, such as titanium dioxide and zinc oxide, can scatter both types of UV light. So-called chemicalfree sunblocks often contain these compounds, as did the white paste that decorated the noses of lifeguards years ago. Sunscreens today use smaller titanium dioxide particles, which are invisible.

Most sunscreen formulations contain a mix of these compounds to provide broad-spectrum protection over UVA and UVB wavelengths.

If sunscreens absorb light, they must also re-emit it. "They cannot destroy that energy, they can only convert it to some other form," says Knowland. Moreover, scattering compounds that reflect light off the skin also redirect some of it onto the skin.

When exposed to UV light, a sunscreen's active compounds interact with inert ingredients and with each other, as well as with the skin. The first step in deciphering this complex interplay is to conduct test-tube studies of the chemicals involved.

The results may not reveal what sunscreens actually do on the skin, but they do indicate what sunscreens are capable of doing—"so that if you want to examine what these chemicals might do in a realistic situation, then at least you know what to look for," Knowland explains.

In the early 1980s, researchers demonstrated that PABA increases the formation of a particular DNA defect in human cells. This defect occurs when two adjacent molecules of thymine, one of the four bases of DNA, link together chemically to form a dimer. People who lack the mechanism to repair these defects are more susceptible to skin cancer, says Knowland. "Thymine dimers in your DNA are bad news."

Last year, Knowland and Oxford colleague P.J. McHugh found in test tubes and in laboratory-grown human cells that Padimate O, a derivative of PABA, does not generate such thymine defects. However, it does oxidize DNA, and it produces free radicals that break DNA strands.

Titanium dioxide and zinc oxide create similar strand breaks. Aware of the compounds' potency, manufacturers coat the sunscreen particles to make them less active. "These treatments do indeed reduce the activity," Knowland notes, "but they don't seem to eliminate DNA damage altogether."

Experiments have shown that sunscreen-protected skin seems to suffer less DNA damage than unscreened skin, notes Frank Gasparro of Thomas Jefferson University in Philadelphia. "However, DNA damage isn't the only thing that contributes to skin cancer." In recent years, dermatologists have also become concerned about sunlight's ability to suppress the immune system, but little is known about this effect.

Knowland says that hydroxyl radicals probably caused the DNA strand breaks he observed in his laboratory experiments. Allen adds that oxygen radicals, while not as reactive, can also harm DNA and other cell components. In collaboration with Sandra K. Allen, he used a filtered lamp that simulates sunlight to illuminate various sunscreens and gauge their ability to produce oxygen radicals.

That ability varied widely. PABA generated oxygen radicals most readily, whereas benzophenones, such as oxybenzone, and salicylates appeared to produce none. "They are not equal," Allen says. Based on these results, however, Allen hesitates to recommend any one sunscreen over another.

The picture gets even more complicated when one considers how the sunscreens interact with the radicals they have generated. "The sunscreen actually forms oxygen radicals that we would like to protect the skin against, but sunscreen also reacts with and traps them," mitigating harmful effects, Allen speculates. Some scientists argue that it is by trapping radicals that sunscreen blends offer their protection, he notes.

No one knows whether sunscreens form oxygen radicals under real-world conditions, nor, if such radicals do form, whether they would damage living cells, Allen cautions.

Much of the debate rests on whether a given sunscreen penetrates the skin. If it stays on the outermost layer, the epidermis, the effects of free radicals may not matter, since the epidermis is made of dead skin cells, says Allen. If a sunscreen penetrates the epidermis, enters the underlying cells, and is then excited by UV light, the picture becomes more disconcerting.

Studies have shown that skin does absorb certain sunscreens. The breakdown products of PABA, for example, can be detected in urine, Knowland says. The same is true of the UVA absorber oxybenzone, researchers from the University of Queensland in Australia reported in the Sept. 20, 1997 LANCET. Schering-Plough Health Care Products in Memphis, Tenn., manufacturer of several brands of sunscreen, countered in the Feb. 14 LANCET that the amount of absorbed compound the Australian team detected was too small to be harmful.

UV-scattering compounds don't sidestep the question of absorption either. Reducing the size of titanium dioxide particles to make them invisible could also enable them to enter cells more easily, Knowland suggests. "As far as published literature is concerned, my own personal view is that this question has not been adequately addressed yet."

"The bottom line is, are sunscreens a good thing or not?" Most experts would say yes, perhaps dismissing as irrelevant the effects observed in the laboratory, Knowland continues. "They may turn out to be right, but my own view is that you have to continue to explore that before making an absolutely definitive pronouncement."

"Wishing for a result isn't going to get that result," Gasparro remarks. "More research and understanding of basic science and biology in the skin—that's going to tell us what's going on." □

Letters continued from p. 355

Why platelets are stupid

"Genetic makeup can boost aspirin's benefit" (SN: 5/2/98, p. 278) states, "Platelets, the smallest blood cells, are indispensable." Technically, platelets are not cells—they lack nuclei and stem from cytoplasmic fragments of large bone-marrow cells.

This lack of genetic information explains why "'platelet cells are pretty stupid.'"

James G. Losser
Evanston, Ill.

Inserted genes and survival

For an inserted gene to spread to most mosquitoes in the wild ("Colorful gene marks mosquito manipulation," SN: 4/4/98, p. 213), wouldn't it have to provide some survival advantage to the insect? Doesn't the fact that mosquitoes are carriers of diseases such as malaria suggest that these pathogens themselves provide some survival advantage and that eliminating them from the mosquito would be a detriment to its survival?

Ted Toal
Nevada City, Calif.

Not necessarily. As for your second question, the malaria-causing parasites offer no known benefit to the mosquitoes that host them. Unlike genes, the parasites aren't inherited by a mosquito's offspring and therefore play no role in natural selection. —J. Travis

Early work on mutualism

"Mutualisms seen as partnerships for barter" (SN: 4/11/98, p. 230) is an excellent model, and I believe it applies to much more than fungi and plants.

In 1902, P.A. Kropotkin published an extended scientific study of animals and of human societies, looking for validation of the "standard" interpretation of Darwin's law of the survival of the fittest. What he and others found was that, as interpreted then, survival of the fittest operated primarily against nature.

The second great law of survival most prominent among animals, within or without the same species, was mutual aid. See *Mutual Aid: A Factor of Evolution* by Kropotkin, published originally in 1902 and still available.

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