In a new study, biologists report that melanocyte skin cells detect ultraviolet light using a photosensitive receptor previously thought to exist only in the eye. This eye-like ability of skin to sense light triggers the production of melanin within hours, more quickly than previously thought, in an apparent rush to protect against damage to DNA.

PROVIDENCE, R.I. [Brown University] — For most people, tanning seems a simple proposition. A naturally light-skinned person lies in the sun for hours and ends up as bronzed as a Jersey Shore star. To scientists, the reaction of skin to ultraviolet light is more mysterious. A new study demonstrates that skin detects UVA radiation using a light-sensitive receptor previously found only in the eye and that this starts melanin production within a couple of hours. Until now, scientists only knew that melanin production occurred days after UVB radiation had already begun damaging DNA.

“As soon as you step out into the sun, your skin knows that it is exposed to UV radiation,” said senior author Elena Oancea, assistant professor of biology in the Department of Molecular Pharmacology, Physiology, and Biotechnology at Brown University. “This is a very fast process, faster than anything that was known before.”

Credit: Mike Cohea/Brown University

Scientists believe that melanin protects the DNA in skin cells against damage from UVB rays by absorbing the incoming radiation. It isn’t perfect, which is why people must use sun block. But the new study in the journal *Current Biology* shows that the body mounts its defense much sooner, well before it becomes apparent in the form of a tan.

In lab experiments with human melanin-producing skin cells called melanocytes, Oancea, graduate student Nadine Wicks, and their team discovered that the cells contain rhodopsin, a photosensitive receptor used by the eye to detect light. Moreover, they traced the steps of how rhodopsin unleashes calcium ion signals that instigate melanin production.

Eyes on the skin

In the team’s first experiment, the scientists were looking to see whether UV light instigated a calcium signaling response. They found nothing. But guessing that the skin might sense light like the eyes do, they added retinal, a co-factor of opsins receptors including rhodopsin.

“When we did that, we saw an immediate and massive calcium response,” said Wicks, the study’s lead author.

Further investigations found that the cells contained rhodopsin RNA and protein. Under UV light, when the scientists reduced rhodopsin levels in the cells, calcium signaling was reduced. Later, when they starved cells of retinal, they found that melanin production dropped. The authors also determined that long-wavelength UVA light, rather than short-wavelength UVB light, is what stimulates rhodopsin in melanocytes.
A healthy glow Human melanocyte skin cells fluoresce as their calcium signaling spikes after exposure to ultraviolet light and retinal, a key step in producing melanin. This led researchers to discover rhodopsin receptors in skin, which detect UVA light. During several experiments, they were able to trace the following process: When UVA light strikes rhodopsin receptors with retinal, calcium signals are triggered within a few seconds. After an hour, measurable amounts of melanin accumulate, although in relatively small quantities compared to the production that will occur within 24 hours.

As much as they learned, Oancea and Wicks still have some questions. One is whether rhodopsin is acting alone or in concert with another yet undiscovered receptor. Another question is whether melanocytes immediately begin exporting melanin to other kinds of skin cells for protection or whether they keep the early supply for themselves.

Just because scientists are learning more about how the skin responds to and protects itself against UV radiation, Oancea said, that’s no reason for people to change what they do to protect themselves.

“This doesn’t say, ‘Don’t use sunscreen,’” Oancea said. In addition to Oancea and Wicks, other authors were Jason Chan, Julia Najera, and Jonathan Ciriello.

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**Real Uses of UV and Fluorescence**

Fluorescence is used as a diagnostic technique – bacteria can be stained or genetically engineered so they will fluoresce but this is in the realm of proper grown-up labs with specialist techniques and equipment, not some idiot waving a 20W UV lamp bought on the Internet.
Fluorescent biomolecules such as flavins and the amino acid tryptophan do exhibit fluorescence and these are present in bacteria and other living cells but fluorescence is very specific: a particular molecule will absorb at a particular wavelength and re-emit at another, specific wavelength – and the emitted wave needs to be within the visible spectrum for us to see it without specialised equipment. For example tryptophan emits at 280nm and re-emits at 330nm – and the visible spectrum is about 380 to 750nm so you can’t see it. And molecules like tryptophan will often be buried in a cell or its membrane so you still might not spot it even using the really trick microscopic techniques we have in the lab.

BUT Riboflavin (vitamin B2) reliably emits at a bright green 560nm when exposed to a broad spectrum of UV and bacteria are rather partial to flavins – as are we all: they are essential for metabolism. This is the molecule most UV systems are detecting. It is true that you do tend to find bacteria on surfaces where flavins are present but this does not mean that flavin-free or non-fluorescing surfaces are free of bacteria – far from it. This is why the sorts of UV emitters you see on TV programmes are about as much use as tits on a nun as a pukka diagnostic technique.

Evolutionary significance

The evolution of early reproductive proteins and enzymes is attributed in modern models of evolutionary theory to ultraviolet radiation. UVB causes thymine base pairs next to each other in genetic sequences to bond together into thymine dimers, a disruption in the strand that reproductive enzymes cannot copy. This leads to frameshifting during genetic replication and protein synthesis, usually killing the cell. Before formation of the UV-blocking ozone layer, when early prokaryotes approached the surface of the ocean, they almost invariably died out. The few that survived had developed enzymes that monitored the genetic material and removed thymine dimers by nucleotide excision repair enzymes. Many enzymes and proteins involved in modern mitosis and meiosis are similar to repair enzymes, and are believed to be evolved modifications of the enzymes originally used to overcome DNA damages caused by UV.

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