





TRANSCRIPT

S6 E4: Photosensitivity

Dr Laxmi Iyengar: Welcome to *Spot Diagnosis*. My name is Dr Laxmi Iyengar, and I'm a GP and education fellow at the Skin Health Institute, a world-renowned centre of skin excellence located in Melbourne, Australia.

We would like to acknowledge the traditional custodians of the land on which we are recording this podcast today, the Wurundjeri people of the Eastern Kulin Nation, and pay our respects to Elders past and present, and extend that respect to any Aboriginal or Torres Strait Islander people listening today.

I would like to welcome my co-host, Associate Professor Alvin Chong. Alvin, as you all know, is a consultant dermatologist at St. Vincent's Hospital and Head of the Transplant Clinic at the Skin Health Institute.

Associate Professor Alvin Chong: Thank you, Laxmi. It's wonderful to be here.

Laxmi: Today, we would like to discuss a topic that I'm very curious to learn more about and that is the broad topic of photosensitivity. We have all heard of photosensitive dermatoses, such as lupus, and medications that we use in everyday practice, such as doxycycline, that predispose us to photosensitive reactions. What is photosensitivity, and why does it happen?

Alvin: Good questions, all interesting topics to discuss, Laxmi. We're fortunate enough to have a global expert on photobiology in the studio today to share with us his insight. I would like to introduce everyone to my colleague and friend, Associate Professor Chris Baker. Chris is a Director of the Dermatology Department at St. Vincent's Hospital, Melbourne, so he's, my boss. He has clinical interests in advanced psoriasis therapies, photodermatology, phototherapy, skin cancer, and clinical trials. He undertook postgraduate studies in photobiology at the famed St. Thomas' Hospital in London. Welcome to *Spot Diagnosis*, Chris.

Associate Professor Chris Baker: Thanks for having me here, Alvin and Laxmi, and it's a pleasure to be here.

Laxmi: Chris, I'd like to start from the very basics. What does the term photosensitivity mean?

Chris: Laxmi, photosensitivity refers to sensitivity to light. It's an abnormal reaction to the skin or in the skin from exposure, usually to sunlight.

Laxmi: Chris, could we please discuss the different types of UV radiation and how these affect the skin, specifically with regards to photosensitivity?





Chris: UV radiation coming from the sun is classified into three primary types based on wavelengths. UVC is the shortest wavelength, ranging from 200 to 290 nanometers, and UVC is completely absorbed by the ozone layer, so no UVC reaches the Earth's surface.

UVB is a short wavelength, part of the UV spectrum, ranging from 290 to 320 nanometers, and it's the wavelengths that cause sunburn and tanning but do not penetrate very deeply into the skin.

UVA is a longer wavelength, ranging from 320 to 400 nanometers, and it accounts for 95% of the UV radiation that reaches the Earth's surface, and being a longer wavelength, it can penetrate more deeply into the skin. It's thought that UVA is a significant cause for skin aging and immunosuppression of the skin, and also can lead to some skin tanning.

The wavelengths of light that induce a photosensitive reaction are known as the action spectrum, and this varies from condition to condition. The wavelengths of light that trigger the reaction, we refer to as action spectrum. Generally, the action spectrum is quite specific to the condition that we're considering.

Laxmi: Chris, how does a sunburn differ from a photosensitive reaction?

Chris: A sunburn is an abnormal reaction to excessive UV radiation, and it's really an inflammatory response. It tells you that there's been an injury to the skin, and I think we're all familiar with the signs of sunburn, redness, edema, pain and itch, and subsequently, peeling of the skin. Sunburn will happen in any individual if the level of UV exposure is sufficient.

In photosensitivity, this reaction or the reaction that we see in the skin is happening at a much lower dose because the patient or the person involved has a sensitivity to the wavelengths that we're interested in. This person can be exposed to sunlight but will react at a much lower level than someone who doesn't have photosensitivity.

Alvin: Chris, photosensitivity is when someone can react to much lower levels of UV light. I understand that they're classified as photodermatoses, right? Can you tell us a little bit about the photodermatoses? I know that there's primary and secondary photodermatoses. Is that correct?

Chris: Well, it is. We divide photosensitivity into primary and secondary. Primary photosensitivity refers to conditions where there is an endogenous, or the patient has an inherent sensitivity to sunlight. Primary photodermatoses also include sun sensitivity or photosensitivity from exogenous substances like drugs or substances that might touch the skin.

Then there's a group of secondary photodermatoses which, like the systemic disorders, lupus, dermatomyositis, some hereditary disorders, and the porphyrias.

Alvin: Okay. Maybe we should explore the primary photodermatoses first, then Chris. As a dermatologist, I see a fair bit of it, but let's go right back to basics. Can you classify it? What are these diseases?





Chris: The primary photodermatoses are a really interesting group of clinical disorders, Alvin, and they're interesting because they present with quite specific presentations. There's usually a bit of a story to get to behind them. Idiopathic photodermatoses are a group where the person has an inherent reactivity to sunlight, and we can talk more about those individual conditions. Exogenous causation of photosensitivity, such as drugs and external chemicals and chemical exposure is very interesting because that requires some detective work.

Alvin: In my clinical experience, the most common scenario I see is someone coming in from a holiday in Bali and winter in Melbourne, and they go on a holiday, and within days they explode in a rash that's itchy on their face and their arms, and their holiday is more or less ruined. This is, from what I understand, a condition called polymorphous light eruption. Chris let's go through this because that's actually the majority of the photodermatoses that I see. What about you?

Chris: Oh, absolutely, Alvin. It's the most common of the idiopathic photodermatoses. Certainly, in our clinic, where we see a lot of photosensitive patients, it's the most common condition diagnosed. It is a very common condition. It's not, interestingly, it's not always recognised as sun related. Many patients who get polymorphic light eruption will connect it to sun exposure, but some will say, oh, I think it's a reaction to the sunscreen, or it's a heat rash. The reason for that is there's usually a bit of a delay from the sun exposure to the appearance of the rash. In the situation you just described, where a patient goes on holidays and gets the rash, it's usually day two or day three when the rash comes out.

The signs that make us think that it's a sun sensitivity problem is that it will be on exposed skin. Interestingly, it's often on exposed skin that isn't normally exposed. It's where the clothing's come off, forearms, arms, torso, tops of feet, whereas the backs of hands and face are often spared. This is due to a phenomenon called hardening where regular and repeated UV exposure causes those areas to be less reactive.

Alvin: What happens over time? If a patient gets this problem every time, from what I understand, it's really when there's a huge amount of UV at once. A sunny holiday like going to Fiji or going to Bali from winter is the classic trigger. Does this happen if there's actually just gradual, slow exposure of UV?

Chris: It's a really interesting point you raise, Alvin, because you'd think that sun sensitivity is more common in latitudes where there's a lot of sunshine. In fact, for the idiopathic photodermatoses, it's the reverse. We tend to see it more commonly in latitudes that are away from the equator. That's because there's a swing between sun intensity from winter to summer. The skin during the wintertime forgets about the sun exposure, becomes primed, and then with the first exposure of sun during spring or on a sunny holiday during the middle of winter, the skin's ready to react. Whereas in the sunnier climates, there's a degree of low-grade background UV exposure which causes tolerance of the skin.

Alvin: How do you actually treat polymorphous light eruption, Chris?





Chris: Look, fortunately, most cases of polymorphous light eruption are mild and can be treated with a topical steroid, perhaps an antihistamine, some wet dressings. Mild cases, if the person continues to have low levels of sun exposure, can then often just disappear and the skin will become tolerant for the rest of the season. We actually utilise that phenomenon in treatment. We can actually treat patients with phototherapy using low doses of ultraviolet light. It's a bit like desensitising for an allergy.

If patients have a very severe flare of polymorphic light eruption and extensive rash, very itchy, very uncomfortable, it may be necessary to use some oral steroids for short term. Patients who know they're going to go on a holiday and have an episode of polymorphic light eruption will sometimes send these patients away with a script for some oral corticosteroids just to take for a few days to get through this uncomfortable period.

Alvin: What about sun protection and strict sun protection? Would you recommend this for your patients who are prone to polymorphous light eruption?

Chris: Yes, look, the use of sunscreens in polymorphic or polymorphous light eruption, it's always worth emphasizing to the patient that sunscreen and sun protection are useful. However, many people want to just get out and enjoy the outside, and it's not always practical. The problem with polymorphous light eruption is that UVA is one of the triggers for the eruption, and sunscreens, although they do have some cover into the UVA spectrum, are usually not sufficient to stop it happening. It's a very much individual thing, and some people will benefit from using a sunscreen, whereas with others, they'll still react through the sunscreen.

Alvin: Difficult problem and something we need to pay attention to next time we have a patient with polymorphous light eruption who wants to have a sunny holiday somewhere. My personal preference is actually to give them a small supply of prednisolone as a crisis buster. They go where they go, when the rash comes up, a small dose of prednisolone for a week or two and topical steroid, and it just settles them down.

Chris: Yes. Look, that's not unreasonable, Alvin, and I think it's very much patient-focused. You really want to understand the pattern of reactivity for an individual patient, how predictable is their rash when it's going to occur. With some patients who, for example, are going to get polymorphous light eruption in their hometown over spring and summer, we often start phototherapy prior to the time they expect to have the rash. Phototherapy given over six weeks prior to the spring period can be very helpful and give them tolerance during the summer period. You might also read that hydroxychloroquine has been used and that's another preventative way of reducing incidence.

Laxmi: Chris, how about the other primary photodermatoses?

Chris: Laxmi, the other photodermatoses that we talk about are juvenile spring eruption, solar urticaria, and chronic actinic dermatitis. There's another very rare one called hydroa vacciniforme. Juvenile spring eruption is quite an interesting one. It's very classic if you see it. It's usually young children or teenagers, and it's little itchy blisters, particularly on the helices of the ears, mainly in





young boys, probably related to hairstyle more than anything. Juvenile spring eruption, some feel, is a form of polymorphic light eruption.

Solar urticaria is a very interesting condition, which if you have a patient with this condition, it's very interesting because their history is quite specific. With solar urticaria, the rash or the reactivity to the sun happens very soon after exposure, usually within minutes to an hour or so. Patients described feeling their skin getting very itchy. They'll see redness. They'll see swelling, hives will develop, so urticarial wheels, and these usually last a day and then disappear if sun exposure is discontinued.

The interesting thing about solar urticaria is the wavelengths that trigger this eruption, and they can be quite variable. It can be in the UVB, UVA, or even visible light. A very good question to ask patients is, does this happen with sunlight through glass? If they describe the rash coming up through glass, it suggests that UVA or visible light are relevant in their presentation.

Alvin: That's because glass filters out most UVB.

Chris: Most UVB. If there's plastic film on the glass or plastic in the lamination, that will tend to knock out UVA as well.

Alvin: One of the problems we do in prescribing medicines is occasionally, some of the medications we prescribe are very photosensitizing. We all use doxycycline, minocycline in acne, isotretinoin. Can we discuss, Chris, how do these photosensitizing medications, what happens? Why do they create photosensitivity, and what do we do about it?

Chris: Photosensitivity to medication is a bit of a complex area, Alvin because the mechanisms vary according to the medication. Sometimes it's not the medication causing the problem, but it's a metabolite. You've got differences in tissue binding, dose effects. To simplify it, I think there are two main causes of drug-induced photosensitivity. One is what we refer to as phototoxic reaction, where there's direct interaction between the drug, UV light, and the generation of substances that are very irritant to the skin. It's usually free radicals and oxygen-dependent inflammation. You mentioned tetracyclines, they're a classic for this, and non-steroidal anti-inflammatory agents similarly can do it this way. Medications that tend to be photosensitisers tend to have a number of ring structures, and that explains the sort of photochemical interaction and why they absorb light. The other mechanism is photoallergy, where, again, either the drug or a metabolite, after interaction with sunlight becomes allergenic and sets off an immune reaction in the skin. This may be specifically the chemical involved, or it may mean that the drug or a metabolite is connected to a protein, which the body's immune system then recognises as an allergen. We see this, for example, in the phenothiazines.

Alvin: The way I usually work, if we prescribe doxycycline, for example, in a patient for their acne, I talk to them about the importance of avoiding direct sunlight and using sun protection and using sunscreen. Often, I think that in most female patients they're very compliant. They don't get burnt. Interestingly, I think the compliance is a lot less in male patients, particularly in the summer





months. Doxycycline or isotretinoin almost guaranteed sunburn during the summer. It's interesting, isn't it?

Chris: Oh, yes. Despite advising the patient of it, until they actually experience it often, they don't appreciate what you're talking about. I guess also important to emphasise that with drug photosensitivity, usually it's the UVA spectrum that's triggering the reaction, and there's a lot of UVA around on bright, cloudy days. Patients may not recognise that they're in direct sunlight, that they're still at risk of photosensitivity in those conditions.

The other problem, of course, is that not all patients react the same, and it is interesting. I find, Alvin, you can have two people on doxycycline doing exactly the same thing. One person will get photosensitivity and the other won't.

Alvin: Maybe I can wear my transplant dermatology hat and chime in. There are some medicines which are actually associated with both not only photosensitivity but actually carcinogenicity in azathioprine or Imuran is the classic one. We know that it actually sensitises you to UVA, and you get toxic metabolites from the UVA, which are carcinogenics. Sun protection in these patients is absolutely crucial. More recently, there's some data that thiazide diuretics may be sensitising as well to the sun and can increase potentially slightly the risk of skin cancers in susceptible patients. Is that correct, Chris?

Chris: Yes, it is. There has been some literature on the thiazides and increased skin cancer risk. It's a hard one to understand because most patients on thiazides, well, not most, but a number of patients are in the older age group where there is more of a skin cancer signature anyway. You're absolutely right about drugs like azathioprine which create a DNA lesion. That, in itself, is of concern in terms of skin cancer formation.

Also they're immunosuppressant. They're suppressing the body's immune response which is very important in fighting and dealing with precancerous change in the skin. It's probably a dual action there.

Alvin: Chris, I think the last primary form of dermatosis that we probably should touch on is chronic actinic dermatitis, known as CAD. I've got a couple of cases of CAD, and they can be very challenging to diagnose and manage. What do you think, Chris?

Chris: CAD, or chronic actinic dermatitis, is a really interesting condition, Alvin, and I think certainly worth talking about because the presentation is very classic. The typical patient who gets CAD is the middle-aged to elderly male often spends a lot of time outside, may have had a history of allergic contact dermatitis previously, often to airborne allergens like pollens and plant resins that are in the atmosphere, or rubber allergy, for example. In this group, they tend to have a history of eczema that becomes more chronic and very definitely related to sun exposure. Summertime flare-ups after being out in the sun, they might notice the skin becomes quite red and irritated in the short term.





The other interesting presentation of chronic actinic dermatitis is what we call actinic reticuloid, where if the dermatitis is chronic, the skin becomes quite thickened and infiltrated, and it almost looks like or resembles a skin lymphoma. On biopsy, there's a lot of lymphocytic cells present, but it's not a cancer, it's just a chronic dermatitis.

CAD can be quite disabling, and patients with CAD, if they become very sensitive to light, their action spectrum goes from UVB into UVA into visible light, to the point where they're reacting to very, very low doses of visible light. They can become erythrodermic, their skin can become totally involved and they can be reacting to light through clothing.

Alvin: We usually immunosuppress these patients.

Chris: Very much. These patients, when severe, will need oral corticosteroids and often non-steroid immunosuppressants.

Laxmi: How is photosensitivity clinically diagnosed?

Chris: Like most medical conditions, Laxmi, it's a combination of history and examination findings. The history is very important with photosensitivity for a couple of reasons. One is that not all patients will recognise that sunlight triggered their rash. By asking what the patient was doing in the day or days before the eruption, were they outside, were they exposing skin that wasn't previously exposed to sunlight? The next thing would be things like drug history. We talked about certain medications being associated with photosensitivity. A thorough drug history is very important.

The other sorts of things to take in history would be exposure to external substances, have they been in the garden, for example? There are some plants that produce a chemical called psoralen, which if that touches the skin, sensitises the skin. The sorts of plants that do that are fig trees, celery, limes. It's really quite interesting. Very common things contain psoralens and can cause sun sensitivity.

Then it's really looking at the presentation. How is the rash distributed? Does it affect areas that have been exposed to the sun, cheeks, V of the neck, back of neck? Are there areas of sparing? Are the upper eyelids spared, under the chin, behind the ears? They're very key clinical findings to look for that would help provide information or confirmation of photosensitivity.

Alvin: I might just jump in. I've seen two really amazing cases of plant-induced dermatosis. One is in a gentleman who was cutting down his fig tree. They cut the branches down, and he had gravelled the branches in his bare forearms on a sunny day. He presented with explosive dermatosis of his forearms and only his forearms.

The other one is a little kid who, you know how in summer they make drinks, and they try to sell drinks? While his drink was actually a limeade. He was out in the open, squeezing limes. He's getting the lime juice on his hands. He had this streaky dermatitis on his hands. I said, oh, you've been... It was really quite fascinating. Take a good history.





Chris: Take a good history.

Alvin: Take a good history, yes.

Laxmi: Chris, what are the specific investigations for photosensitivity?

Chris: Laxmi, often diagnosis is based on the history and clinical findings. The investigations are more to exclude other things rather than to confirm photosensitivity. We would normally do a full blood count, do the connective tissue screen, ANA, ENAs in appropriate cases. This is because cases of lupus can present as a sun sensitivity. This is not really diagnosing a primary dermatosis, which, as I said, really boils down to really thorough detective work as a clinician.

We often perform a skin biopsy on inflammatory skin conditions to confirm diagnosis. In the idiopathic photodermatoses, we'll see changes of urticaria or eczema, or dermatitis. It won't be specific or tell us that this was a photosensitivity issue.

Alvin: There is one more test that we potentially can do, which is phototesting, Chris. I'll let you take us through it.

Chris: Phototesting is, if you like, the gold standard for proving photosensitivity. It's also termed provocation testing. This is where you use wavelengths of light that you think might be causing the problem, shining it on the patient's skin in a small amount and demonstrating an abnormal reaction. The sources of light that you can use, it can be natural sunlight. For example, just having the patient go outside for a few minutes might confirm solar urticaria, for example. Most dermatologists who provide phototherapy services have UV equipment in their offices. You can do testing of the skin using the phototherapy equipment. This is usually UVB. It's a fairly narrow spectrum.

The use of sources of light that break down the spectrum into individual wavelengths or provide a source that approximates sunlight, called a solar simulator, also very useful in testing the skin. However, these are really restricted to very specialised centres. Really, for most patients and most doctors, unfortunately, aren't an available testing regime. I think if you can show that a patient is reacting to light and you can provoke a small amount of the rash, then that does help with diagnosis.

Alvin: Ever wondered what the Skin Health Institute does? At the Skin Health Institute based in Melbourne, we aim to improve skin health for all our patients. The research we conduct shapes clinical treatment and practice. We provide over 30,000 patient treatments each year and also deliver exceptional education programs for dermatologists, registrars and healthcare workers. We provide specialist training for visiting international medical graduates, workshops to upskill GPs and medical students, and public education programs aimed at improving skin health in the community.

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Laxmi: Let's segue to the secondary photodermatoses now. We talked about lupus earlier. Chris, can you give me some insights into how photosensitive rashes can present in lupus?

Chris: Yes, Laxmi, the connective tissue diseases and autoimmune disorders have in many patients photosensitivity as one of the presentations. Patients may notice that after short periods of sun exposure, their skin becomes red, irritable, and that can be seen in lupus and dermatomyositis. Also the rash itself can start to come out and be provoked in the area of sun exposure.

In lupus, for example, and particularly subacute cutaneous lupus, which is with the Rh-positive antibody, we'll often see patches of lupus and erythematous and scaly patches appearing in a very sun-exposed area, so the V of the chest, the upper arms, for example. I think the mechanism why it occurs is poorly understood, but it is one of the diagnostic features of these autoimmune connective tissue disorders.

Laxmi: Can we talk about dermatomyositis, which can also present with that classic V-shaped photodistributed rash over the anterior chest and face?

Chris: Yes, I think, Laxmi, this is another autoimmune connective tissue disorder where sun sensitivity can be one of the presenting features. When diagnosing dermatomyositis, you look for other features such as the Gottron's papules over the knuckles and the heliotrope erythema around the eyelids. Now, they're not specifically photo-induced signs, but they are occurring on sun-exposed areas. You may see in dermatomyositis erythema and changes in the skin in the V of the neck or the back of the neck area.

Alvin: The other component of dermatomyositis is myositis. Patients will have very often sore muscles, inability to get up from a chair, can't raise their arms. The concern we have in dermatomyositis is that it's a multi-systems disease that can, in some cases, your respiratory muscles get affected. You can die of respiratory failure. The other association is that of malignancy. Dermatomyositis is something we always keep our eyes open, particularly if someone presents with photosensitivity.

Laxmi: I'd like to discuss with you how common skin conditions respond to sunlight exposure. Why is UV used as a therapy for some diseases, such as psoriasis and eczema?

Chris: There's a paradox there, isn't there, Laxmi, that we've got conditions that respond to low doses of UV. As you mentioned, psoriasis, atopic dermatitis, but there are a group within the patients who suffer psoriasis and eczema who are actually exquisitely sensitive to light, and their condition actually can be exacerbated by sun exposure. There's really no way of predicting who will do well and who will be exacerbated unless you get some clues in the history of past experience.





There are other skin disorders which are exacerbated by sunlight, so rosacea is a very classic common skin condition which is made worse by UV exposure. Some less common conditions, Darier's disease, some of the autoimmune blistering disorders can be made worse by sunlight. A very common condition which is often triggered by sunlight is reactivation of herpes simplex.

Alvin: Well, we see very rarely some genetic conditions that can be associated with photosensitivity, Chris. Perhaps we can discuss xeroderma pigmentosum.

Chris: It's interesting there are a group of disorders which are inherited, and unfortunately, children that are affected by these conditions will, as part of their presentation, have some sensitivity. Xeroderma pigmentosum, where there is a defect in the DNA repair system, is a classic example. Not only do these patients in early life demonstrate sun sensitivity, so their skin becomes reddened and irritated after short-term sun exposure, but the DNA damage that's done and not repaired then leads to premature development of skin cancer. Often, if not recognised, not well dealt with sun protection can lead to premature death of the individual. This is interesting also that this condition seems to happen in many patients around the world, different racial groups, different skin types, so skin pigmentation is not protective.

Laxmi: There are some metabolic conditions that are associated with photosensitivity. Porphyria cutanea tarda, otherwise known as PCT, is probably the best known. Chris, could we please discuss how defects in metabolism could contribute to photosensitivity?

Chris: Well, the porphyria group are a really interesting group of disorders because there is this biochemical explanation as to what's going on, and the genetic enzyme deficiencies are well understood. I think, as you say, Porphyria cutanea tarda, or PCT, is the most common that we see.

It is interesting though, Laxmi, that PCT patients often don't associate their sun sensitivity issue with the condition. What happens in PCT is that with sunlight and the buildup of porphyrin in the skin, it leads to skin fragility and so patients start to get blisters and erosions on the backs of the hands, sites where there is sun exposure but also trauma. Although it is a photodermatosis in the classification, patients are not presenting with acute photosensitivity.

There are other forms of porphyria where there is acute photosensitivity, and there's erythropoietic protoporphyria and some of the very rare forms of porphyria where there are congenital deficiencies in the enzyme system. These patients will present with acute photosensitivity. As children, if they're left in the sun, will often cry because they're getting stinging within the skin, and it will be noted that their skin becomes red and inflamed. That's the less common group of porphyrias when we compare it to PCT.

The other metabolic condition to discuss is pellagra, which is due to a deficiency of niacin. This is a very interesting condition because patients present with a dermatitis which is restricted or most evident in sun-exposed areas, so backs of the hands, forearms, the V of the neck. It gives a very particular type of appearance of dermatitis with this sort of glazed, pigmented change in the skin, and they use the term Casals necklaces used to describe the changes around the neck.





In addition to dermatitis, these patients often have GI disturbance, diarrhoea, diminished cognitive ability, and dementia, and so the three Ds for pellagra. This can occur in patients who have nutritional deficiencies, those that, for example, have alcoholism. It's also seen in some medications, so patients that have been on isoniazid for a long period of time or are in an environment where there's very little tryptophan in the diet. Pellagra a very classic presentation and one that's always worth thinking about when we're seeing an elderly patient with a photosensitive dermatosis or a patient who is subject to neglect who's presenting with dermatitis.

Laxmi: Chris, I've noticed that skincare products that we use every day, such as retinol, can cause photosensitivity in some patients. Is that something you are increasingly encountering in your clinical practice?

Chris: I think this is a very important clinical point, Laxmi because vitamin A and vitamin A analogues are used frequently in cosmetics. We also prescribe retinoids or vitamin A analogues in treatment of acne, can be used to help reverse sun damage and help with the changes of fine wrinkling and aging. People are using topical products that contain retinoids very frequently.

Retinoids are chemicals that cause sun sensitivity or sun sensitisation. I think it's important to remind patients of this fact and perhaps use them at night, make sure they're using a sunscreen during the day. When patients come in and say, look, I think this product is causing skin irritation, do think about photosensitivity and ask them about, well, what are you using? Are you using the product and then going outside? The association of photosensitivity with the retinoids is well understood. We see it in patients who take high doses of vitamin A, but also we see it in patients who are on treatment for acne with isotretinoin, the vitamin A analogue.

Laxmi: Chris, may I ask whether photosensitive dermatoses can be more challenging to recognise in patients of skin of colour?

Chris: That's a great point because the natural thinking would be that patients who have higher skin pigmentation may be at less risk of photosensitivity, but we have to keep a high index of suspicion because, as we discussed earlier, a number of the idiopathic photodermatoses have an immune basis and skin pigmentation and Fitzpatrick skin type, so the more heavily pigmented the skin is by no means protective. We see PLE, and we see CAD in all skin types and all racial groups.

The other issue to consider is that the presentation or the appearance of the rash can be different in patients with skin of colour. The distribution is still of a sun sensitivity exposure pattern, but there may be more heavily pigmentation than you would see in, say a type 1 or type 2 skin of paler colour. I think the suspicion needs to be kept because there is this natural inclination to think maybe these patients are not likely to get photosensitivity.

Patients with skin of colour are less likely to get sunburn because there is some resistance to total dose, but the idiopathic photodermatoses don't really require much light to trigger them. Hence, skin pigmentation isn't protective necessarily.





Alvin: One last question, Chris. We have patients who sometimes come and they tell us that when they use a sunscreen, they get a rash from the sunscreen. What exactly is happening there? Is this truly sunscreen allergy or is there something else at play?

Chris: Yes, look, it's a good question, Alvin, because sunscreens are used so frequently, and we do have patients who, for various reasons, get a reaction or feel intolerant to a sunscreen, and I suppose if we unpack it, first of all, patients who have an idiopathic photodermatoses like polymorphous light eruption put their sunscreen on, go in the sun, still get the rash, and they think, I've reacted to the sunscreen, but in fact, it's the condition that's reacting to small amounts of UVA that have got through. That's probably situation number one.

Number two would be patients who have a true allergy to a component in the sunscreen, and sunscreens often have preservatives, fragrances, and the sunscreen absorbing chemicals. I think it's fair to say that reaction to preservatives and fragrance occurs a lot more frequently than allergy to a sunscreen chemical, but because these chemicals are used so frequently in such a high amount of use, we are going to see allergies to them.

The interesting thing about sunscreen allergy is that some of the sunscreen allergens require sun exposure to trigger the allergy. What I mean by that is the patient could put the sunscreen on and keep their skin covered and have no reaction, but when they put the sunscreen on and go into the sun, the sun interacts with the sunscreen chemical and triggers an allergic response, and they get the rash. I'm sorry that it's not simple, but these are the sorts of issues you need to think about when you're talking to your patients about reacting to their sunscreen.

Alvin: That's a good point, and it's very complicated.

Chris: Despite that and the reactions that some people get to sunscreens, nevertheless, the benefits of using a sunscreen for sun protection far outweigh these small risks, and we would certainly encourage patients to adopt strict sun protection in the interest of preventing skin cancer.

Laxmi: That concludes today's episode on photosensitivity, and I personally learnt a lot from today's discussion. Chris, thank you for being here and sharing your insights, and Alvin, thank you also for sharing your expertise.

We would like to thank the education team at the Skin Health Institute and Balloon Tree Productions. Remember, these podcasts are not meant to replace medical advice. If you have a skin condition that requires attention, we strongly encourage you to see your medical practitioner.

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