**Skin biology video 2 notes: sunburn, DNA and skin cancer**

**Time course of erythema**: after UVR, erythema peaks at around 8-24 hours, although the higher the UVR dose, the earlier you may see erythema, and the longer it may remain.

You can detect **changes in blood flow** in response to UVR, using a laser doppler or a reflectance instrument, and these instruments are more sensitive than the human eye ( you do not need to learn about these machines). You can get 'sunburn' without being able to see it.

**Sunburn:** Strange though it may seem, there is no robust and validated definition of what constitutes sunburn as a binary variable (it is a largely a continuous trait, so it perhaps makes more sense to talk about the degree of sunburn).

**Inhibition of erythema:** Experimentally you can reduce erythema using topical indomethacin applied just before irradiation, although there is a lack of clear evidence for systemic NSAID inhibiting erythema (note: this would **not** be expected to minimise DNA damage, as DNA damage is proximal to erythema in terms of causation).

**Action spectrum:** The term action spectrum is used in many contexts. It is a measure of how some endpoint varies with wavelength (or frequency).

**UVB versus UVA:** In this video I do not introduce the terms UVB and UVA. For the record, UVB is shorter wavelength, than UVA, and causes more DNA damage. We will revisit some of this in a later video.

**Chromophore**: I am using the term chromophore in the sense of a molecule that absorbs / undergoes a conformation change in response to light (in this case UVR). DNA is a chromophore, as is melanin or haemoglobin.

**Type of UVR induced DNA damage:** The development of pyrimidine dimers in DNA is characteristic of UVR induced damage. Antibodies to these dimers can readily detect damage in situ. UVR can however produce many other types of DNA damage.

**Freckles / freckling**. A freckle is a focal overproduction of melanin, and a marker of sun damage from UVR.

**Xeroderma pigmentosum** is an autosomal recessive condition. There are a variety of types of XP which vary in their phenotypes, reflecting mutations of different genes. The clinical severity depends on the type ('complementation group'), but also the amount of UVR the individual is subject too. For completion, but not because you need to know it, different aspects of the repair of UVR DNA damage can be affected. So, in some patients the freckling is more prominent than in others. Similarly the abnormal erythemal response varies between

the different types depending on whether the repair defects affect genes that are being actively transcribed, or not [you do not need to explore this any further].