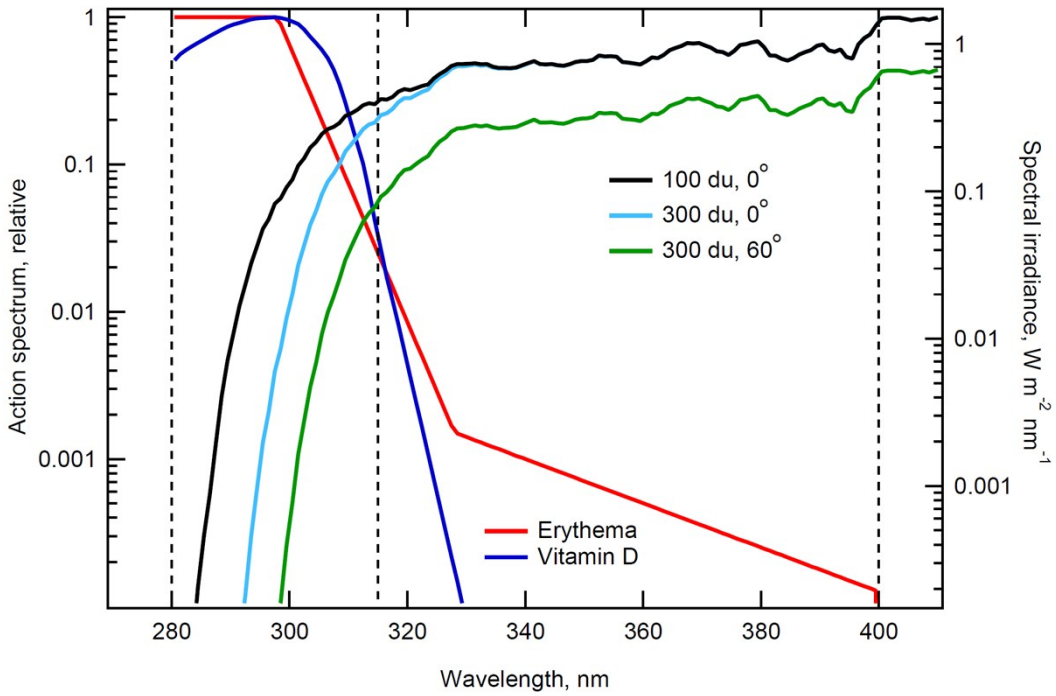
Supplementary data

Spectral data

The UV spectral region of interest at Earth's surface is shown in Figure S1, including the UVA (315-400nm) and UVB (280-315nm) bands delimited by the vertical dashed lines. Action spectra for erythema induction[1] and vitamin D production[2] are also shown, and the substantial UVA "tail" is clearly evident for erythema. Also shown are typical clear-sky spectral irradiances, for several solar zenith angles and ozone columns (integrated UVA and UVB values are given in Table 1). Note that changes in ozone affect mostly UVB wavelengths, while variations of solar zenith angle affect all wavelengths.

Figure S1: Left (logarithmic) scale shows action spectra for (red) human erythema induction[1] and (blue) vitamin D production,[2] and their decrease toward longer wavelengths. The right (logarithmic) scale shows the spectral irradiance at Earth's surface (cloud-free and unpolluted), in decreasing order for (black) overhead sun and low ozone column (in Dobson units, du), (cyan) overhead sun and moderate ozone column, and (green) 60° solar zenith angle and moderate ozone column. Spectral irradiances were computed with the TUV model (version 5.3) and smoothed with a running 5 nm filter, boxcar (rectangular) average, i.e. simple average of central point and the two on each side of it; value is assigned to the wavelength of the central point. Vertical dashed lines show the defining wavelengths for UVB (280-315 nm) and UVA (315-400 nm). Higher resolution is available at http://cprm.acom.ucar.edu/Models/TUV/Interactive_TUV/



UVA Immunosuppression

In the study by Damian and colleagues, the cessation of the allergic reaction after UV irradiation indicates the amount of UV radiation required to induce immunosuppression in these volunteers.[3] The Fitzpatrick skin classifications[4] were mainly Type II (55%) and Type III (37%) and exposure protocols included UVA only, UVB only or solar simulated UV (SSUV) which includes both UVA and UVB, ranging from once only to daily for 5 days (see Table S1).

Immunosuppression was quantified[3] as ΔEI = EI (unirradiated control) – EI (irradiated test site), where EI is the erythema index, determined by the skin reddening measured by reflectance spectroscopy. Prior to any irradiation, there will be some allergic skin reddening, with no immunosuppressive reduction of the skin reflectance (i.e., $\Delta EI = 0$). As exposure increases, the reddening diminishes (i.e., ΔEI increases) until an exposure level is reached where no further improvement is noted, and ΔEI starts to decrease. The exposure at which this occurs is the target irradiance for optimal immunosuppression. Results are shown in Table S2 and Figure S3.

The largest immunosuppression occurred for irradiation from the solar simulator; however this contained both UVA and UVB wavelengths. To investigate the effects of UVA and UVB separately we use the data from the waveband specific exposures.

For UVA exposure, maximum effects were seen after 3 days.

Exposure dose = 3 x 19420 Jm^{-2}

= 58260 Jm^{-2}

$\approx 6 \times 10^4$ Jm^{-2}

The maximum UVA from Figure 1a for a UVI of 3 is 45 Wm^{-2}. At this level, the time to reach this highest immunosuppressive UVA exposure dose is 6×10^4 Jm^{-2} /45Wm^{-2} = 1333 seconds, or 22 minutes. We note, however, that the uncertainties in these estimates are substantial. Sources of uncertainty include the scatter in the data (shown in Figure S3), uncertainty in estimating the time evolution of the effects, and differences between the spectral distribution of the artificial sources and sunlight.

UVB Immunosuppression

For UVB exposure, maximum immune suppression was seen after 4 days.

Exposure = 4 x 1430 Jm-2

= 5720 Jm-2

At noon in summer, the UVB irradiance (to 320 nm) is approximately 3.5 Wm^{-2} (or 2 Wm^{-2} (to 315 nm), or UVI=11).

Therefore, at noon in summer for UVI=11, the time to reach highest immunosuppressive UVB dose is 5720 Jm^{-2} /3.5Wm^{-2} = 1600 seconds (about 27 minutes). This is much longer than the ~ 6 minutes per day noted by Damian and colleagues.[3] We note that their value was for the standard European Sun (SZA=20, ozone=305 DU)[5] with a UVI around 9, i.e., slightly lower than our higher assumed summer UVI of 11, and also that there are considerable differences in spectral shape between the artificial source and sunlight. Furthermore we have used a different boundary between UVA and UVB, which has little effect on UVA but a more marked effect on UVB. For example, in noon summer sunlight the UVB assumed by Damian and colleagues (i.e., up to 320 nm) is a factor of two greater than in our definition of UVB (i.e., to 315 nm).

The main point to note is that these immunosuppressive effects from UVA (and also from UVB) can occur in time scales of a few minutes that are comparable with the times for erythema.

Table S1. Characteristics of light sources used in UV immunosuppression studies (derived from data in[3]). Also shown are the corresponding characteristics for noon summer sunlight at Lauder, New Zealand (45°S), where values are derived from the same instrument as used to generate the data in Figure 1.

		UVB (290-320)	UVA-II (320-340)	UVA-I (340-400)	UVA (320-400)
Solar Simulator 28 seconds	*Irradiation(Wm^{-2})*	*51*	*97*	*266*	*363*
	Daily Dose (Jm^{-2})	1430	2710	7450	10160
UVA lamp 70 seconds	*Irradiation (Wm^{-2})*	*0.06*	*33*	*244*	*277*
	Daily Dose (Jm^{-2})	0	2330	17090	19420
UVB lamp 28 seconds	*Irradiation (Wm^{-2})*	*51*	*44*	*19*	*63*
	Daily Dose (Jm^{-2})	1440	1230	530	1760
Noon summer at Lauder, 45S	*Irradiation (Wm^{-2})*	*4*			*60*
	Dose for 5 minutes (Jm^{-2})	1200			18000

The slight mismatch between displayed irradiances and doses is due to rounding errors in the values reported by Damian and colleagues [3]. In this work, the older definition for the UVA and UVB boundary of 320 nm was used, rather than the currently accepted 315 nm boundary. Although that choice makes a large difference to the UVB integral (for UVB the integral to 320nm is about twice that to 315 nm, it has only a minor effect on UVA, which is the focus here.

Table S2. Immune-suppression observed (ΔEI) as a function of the cumulative UVB and UVA doses (in Jm^{-2}) from each source [3].

	Solar simulator*			UVA lamp			UVB lamp		
	UVB Dose	UVA Dose	ΔEI	UVB Dose	UVA Dose	ΔEI	UVB Dose	UVA Dose	ΔEI
Day 0	0	0	0.0	0	0	0.0	0	0	0.0
Day 1	1430	10160	13.7	0	19420	15.4	1440	1760	8.6
Day 2	2860	20320	26.6	0	38840	14.7	2880	3520	11.9
Day 3	4290	30480	19.9	0	58260	24.7	4320	5280	11.3
Day 4	5720	40640	28	0	77680	10	5760	7040	19.7
Day 5	7150	50800	-	0	97100	7.5	7200	8800	12.8

*Note that the solar simulator and the UVB source each contained significant amounts of both UVA and UVB, so it is difficult to isolate their individual contributions.

Figure S2: Effect of UVA irradiation on immunosuppression. Symbols correspond to the cumulative doses for day 1 to day 5 (plotted from data in[3]). While the UVA lamp contained no UVB, the other two lamps contained both UVB and some UVA, as shown in Table S2.

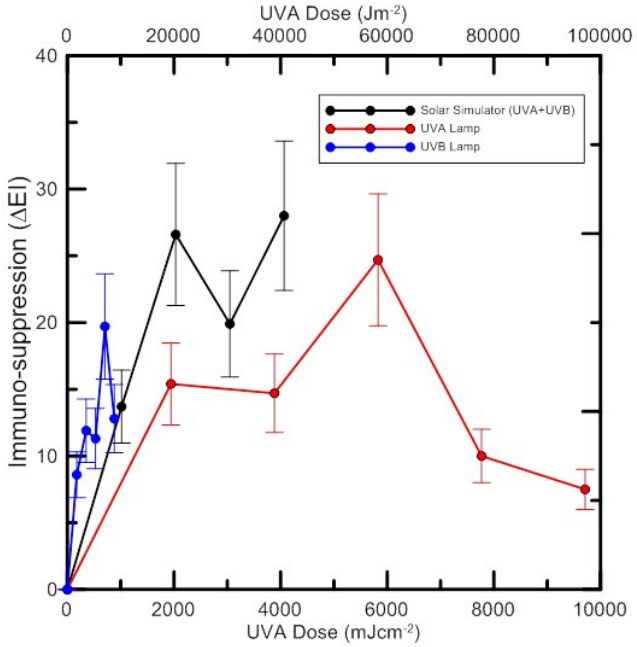


Table S3. Assessment of factors to consider in using the UV Index as a guide to time outdoors and sun exposure

BIOLOGICAL ENDPOINT	Action spectrum	Central wavelength	Threshold	Time scale	Reciprocity on short (episode) timescale	Saturation?	Reciprocity on lifetime scale (cumulative?)	Systemic or local?	Strategy to optimize	Notes
Erythema	CIE[1]	309	1 MED ~ 2 SED for skin type II	0·3-3 days	yes	yes/tanning	no	Local	< 1MED = 2 SED, e.g. limit time < 20 min if UVI > 6;	Changes with skin type. 1MED=2SED only for type II
Skin cancer	SCUP-h[1]	309	None?	3-30 years	unclear	no?	yes	Local	Avoid erythema	Is tanning protective, or is it evidence of cumulative damage?
Skin cancer	UVA-role	315-400	Same as immune suppression?	3-30 years	unclear	no?	yes	Systemic	possible with UVI<3 with long duration of exposure	Nucleotide excision repair and immune suppression may be important
Immune suppression	UVB & UVA	280-400	6 x 10^4 Jm^{-2} UVA or 22 minutes at 45 Wm^{-2}	0·3 days	yes	probably	Immune suppression may not be cumulative per se	Local and systemic	possible with UVI<3 with long duration of exposure	Not clear what happens for skin types other than II and non-allergic individuals
Vitamin D	Vitamin –D, CIE [2]	307	1·3 SED	0·3-3 day	yes	yes: reach steady state for moderate exposure	Assume no long term memory of past vitamin D exposure	Systemic	1hour (60 mins)/UVI = 1 SED < Time < 1.3 SED, three times per week	
DNA damage	Setlow[1]	304	None?	0·3-3 days	probably	no		Local		Non-human model

Action spectrum: describes the relative biological effectiveness of different wavebands of UVR for the specific outcome. The Table details the action spectrum most commonly used in relation to the biological endpoints considered here.
Central wavelength: estimated as the wavelength that divides the action in two equal parts, for SZA=30^o, Ozone=300DU

Threshold: this represents the exposure dose required for the biological endpoint to occur.
Time scale: the duration of exposure required for the biological endpoint to occur.
Reciprocity: “yes” implies that the biological endpoint depends only on the exposure dose of radiation and not on the rate at which the energy is administered (i.e. exposure to UVI = 1 for 10 minutes has the same effect as exposure to UVI=10 for 1 minute)
Saturation: is a steady-state achieved with ongoing exposure?
Strategy to optimize: refers to the UVR exposure strategy that would best achieve beneficial biological endpoints and avoid adverse biological endpoints

References

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3. Damian DL, Barnetson RS, Halliday GM. Low-dose UVA and UVB have different time courses for suppression of contact hypersensitivity to a recall antigen in humans. *J Invest Dermatol* 1999;112(6):939-44. doi: 10.1046/j.1523-1747.1999.00610.x
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5. Diffey B, Robson J. A new substrate to measure sunscreen protection factors throughout the ultraviolet spectrum. *J Soc Cosmet Chem* 1989;40:127-33.