



Mid Atlantic Regional Chapter of the American College of Sports Medicine

Annual Scientific Meeting, November 2nd - 3rd, 2018
Conference Proceedings
International Journal of Exercise Science, Issue 9, Volume 7



Skin Erythema and Blood Flow Responses to Acute Ultraviolet Radiation Exposure

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Skin exposure to ultraviolet (UV)-B light elicits an inflammatory response, resulting in increased skin blood flow and erythema (reddening) over the next 24 h, but the initial time course is unclear. It is likewise unknown whether these responses differ after exposure to broad spectrum UV (UV-AB) light and how they are impacted by sunscreen on the skin. **PURPOSE:** To examine 1) the time course of skin blood flow and erythema after acute UV-B and UV-AB exposure, 2) the relation between peak blood flow and erythema, and 3) the impact of sunscreen application on these responses. We hypothesized that 1) the blood flow and erythema responses would be greater after UV-AB compared to UV-B exposure, 2) the peak responses would be related, and 3) sunscreen would attenuate responses to both UV-AB and UV-B. **METHODS:** The ventral aspect of both forearms of 7 healthy adults (23±3yrs; 5M/2F) were exposed to either UV-B (75 sec, 6 mW/cm² UVB) or UV-AB (75 sec, 6 mW/cm² UVB + 8 mW/cm² UVA). One 2-cm² site on each arm was pre-treated with topical SPF-50 sunscreen. Red cell flux (laser-Doppler flowmetry) and erythema index (EI; reflectance spectrometry) were measured at each site before, immediately after, and 2, 4, 6, and 8 hours post-exposure. Cutaneous vascular conductance was calculated (CVC=flux/MAP) for each time point, and both CVC and EI were expressed as change from baseline. **RESULTS:** EI increased from baseline after exposure to UV-B (4 h, $p=0.04$; 6 h, $p<0.01$ 8 h, $p<0.01$) and UV-AB (6 h, $p=0.02$; 8 h, $p<0.01$), but increases in CVC (UV-B, 6 h: $p=0.02$; 8 h: $p<0.01$; UV-AB: 8 h, $p=0.07$) were delayed by ~2 h. EI and CVC responses were not different between UV-B and UV-AB ($p>0.05$) and there was no relation between peak EI and CVC following exposure to either UV-B ($R^2=0.08$; $p=0.55$) or UV-AB ($R^2=0.03$; $p=0.70$). Sunscreen blunted the CVC responses to UV-B (6 h, $p=0.04$; 8 h, $p=0.003$) and UV-AB (8 h, $p=0.02$), and reduced the EI response such that it was not different from baseline after UV-B or UV-AB ($p>0.05$). **CONCLUSION:** Exposure to UV-B or UV-AB induced a linear increase in EI and a delayed increase in CVC, with no direct relation between these responses. Sunscreen blunted the EI and CVC responses. These data suggest that an erythema-independent inflammatory response to UV occurs in the cutaneous microvasculature, and that sunscreen may protect against this response.