Sunshine and multiple sclerosis

Bruce V Taylor

In 1960, Donald Acheson made the seminal observation that the global distribution of multiple sclerosis (MS) may be related to available hours of sunshine; this observation was met with disbelief at the time. However, over the subsequent more than 50 years, it has become clear that he may well be right. The effect of sunlight exposure and subsequent vitamin D production (generated by photolysis of 7-dehydrocholesterol in the skin by ultraviolet radiation (UVR)) on the onset and progression of MS has become a significant research focus particularly over the last 10 years, with large-scale clinical trials of vitamin D intervention now underway in many countries.

A lot of the focus has rightly been on the role of vitamin D due to its pleuripotent effects on the immune system; however, recent work has suggested that sunlight itself may be an independent modulator of MS clinical course and MS onset acting on inflammation and/or neuroprotective pathways independent of the actions of vitamin D. Zivadinov et al describe a cross-sectional study of the effects of recalled sun exposure on MRI-measured brain volumes, particularly whole-brain volume and grey matter volume. They found that those within the highest quartile of summer sun exposure had the highest brain volume after adjustment for Expanded Disability Status Scale. Interestingly, adding measured vitamin D levels to the model did not significantly alter the association, indicating that the effect of sunlight exposure was independent of the measured vitamin D levels.

A similar finding has been reported by Lucas et al in an Australian multicentre study of clinically isolated syndrome (CIS) where both vitamin D levels and UVR exposure were independent risk factors for the development of CIS. Within the current study there are some limitations, namely that it was a cross-sectional study and relied on recalled rather than measured sun exposure and therefore the question of reverse causality cannot be excluded, also discussed by the authors. However, the results are intriguing as they open up another avenue of research into potential mechanisms for MS causation and progression and perhaps intriguingly a potential for neuroprotection via non-pharmacological means.

The potential role of sunlight as a protective agent in MS is also now an important co-factor when designing studies of vitamin D and other therapeutic agents and needs to be measured in such studies as it may well confound the results. Well-designed studies accruing data longitudinally and measuring both vitamin D and sunlight exposure may well be able to tease out the contributions of both factors on MS clinical course.

Over the last 30 years, there has been a push to minimise exposure of fair-skinned people to sunlight to deal quite correctly with what in some countries was an epidemic of UVR-induced skin cancers. The question now is have we gone too far or is there a role for judicious non-erythema-inducing UVR exposure to promote a healthy immune system via both direct and indirect vitamin D-mediated effects? Clearly, this is a debate that needs to be had before current sun exposure policies are changed and more research is needed before we have the answer.

Competing interests None.

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