

The effect of intentional sun exposure on 25(OH)D concentration in indoor workers: evidence from a randomised controlled trial

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Abstract. Sunlight generates vitamin D, but there are limited data from randomised trials on which to base health policy advice that balances the risks of sun exposure with the benefits of vitamin D production. We aimed to assess the effect of solar ultraviolet (UV) radiation exposure on 25(OH)D concentration using a randomised controlled trial (RCT) design. The intervention tested was supervised exposure to one standard erythemal dose (SED; 100 J/m²) of solar UV radiation three days per week for three weeks with approximately 35% of the body surface area not covered by clothing. Thirty-six fair-skinned (skin type II and III) indoor workers from Brisbane, Australia were randomised into either the intervention group (n=16) or the control group (n=20); the latter did not receive any supervised sun exposure. Thirty participants (17 control, 13 intervention) completed the trial. We collected blood samples at baseline, once per week during the three week intervention period, and four weeks after the intervention finished. The cumulative UV radiation exposure over the intervention period measured using polysulphone badges was higher in the intervention group than in the control group (median 8 vs 4 SEDs, p=0.14). After three weeks, the mean serum 25(OH)D concentration increased from 60 to 65 nmol/l in the intervention group and from 55 to 57 nmol/l in the control group. After adjustment for baseline 25(OH)D, the mean change per week during the intervention phase was non-significantly higher in the intervention than in the control group (0.7 vs 0.3; p=0.35). This difference was not sustained during the follow-up period. Larger field trials are needed to inform policy about how much natural sun exposure is required to raise 25(OH)D concentrations. This study provides a basis for the design of such a trial.

Introduction

Vitamin D is essential for maintaining bone health. It is produced through exposing the skin to UV radiation, so lack of sun exposure can lead to vitamin D deficiency. Approximately 25% of Australians have a 25(OH)D concentration less than 50 nmol/L, despite abundant sun exposure. Thus Australians may need to be advised to increase sun exposure to avoid deficiency, but this needs to be balanced against the risk of skin cancer. Current sun exposure recommendations are based on studies that have used artificial UV radiation sources, which may overestimate the effect on 25(OH)D. Studies using solar UV radiation exposure will help to generate evidence-based recommendations about how much sun exposure is needed to optimise vitamin D status.

Methods

We conducted an RCT to investigate the effects of intentional exposure to solar UV radiation on change in 25(OH)D concentration. We recruited indoor office workers from Brisbane (27°S) who had Fitzpatrick skin type II or III. We randomised them to intervention and control groups. Both groups were asked to minimise sun exposure for a period of 8 weeks and supplied with sunscreen for daily use. The intervention group attended 3 solar UV radiation exposure sessions each week for 3 weeks, with their face, arms to above the elbow and legs to above the knee exposed. At each session they were exposed to one SED of radiation, measured using a PMA Solar Light UV radiation detector. Blood was collected at baseline, weekly during the intervention phase and 4 weeks after the end of the intervention phase. 25(OH)D concentration was measured using LC-MS/MS.

Results

The full results of the study have been published previously [*Khan, et al.*, 2018].

Of the 36 participants enrolled in the study 30 completed the study and provided all blood samples.

The dose of UV radiation measured by the polysulphone dosimeters is presented in Table 1. The cumulative median UV exposure over three weeks was 4.2 (range, 0.8-22) and 8.2 (range, 3-22) SEDs in the control and intervention groups, respectively (p=0.11).

Table 1. Mean UV radiation exposure received per day over the three weeks of the intervention period (based on polysulphone badge data)

	UV radiation exposure (SED) Median (Q1, Q3)		p-value
	Control (N=17)	Intervention (N=13)	
Exposure per weekday	0.13 (0.1, 0.2)	0.37 (0.3, 0.6)	0.0008
with intervention session		0.46 (0.4, 0.6)	0.0004 [#]
with NO intervention session		0.24 (0.1, 0.4)	0.11 [#]
Exposure per weekend day	0.36 (0.1, 0.9)	0.32 (0.1, 0.7)	0.88
Exposure per week	1.91 (0.7, 3.2)	2.55 (1.8, 4.3)	0.11

[#] Comparison with the exposure per weekday in the control group

At baseline, the mean serum 25(OH)D concentration was 55 nmol/L in the control group and 61 nmol/L in the intervention group. At the end of the intervention period the mean 25(OH)D concentration in the control and intervention groups was 57 and 65 nmol/L, respectively. Four weeks after the intervention ended the mean 25(OH)D concentration was 62 nmol/L in the control group and 68 nmol/L in the intervention group (Figure 1).

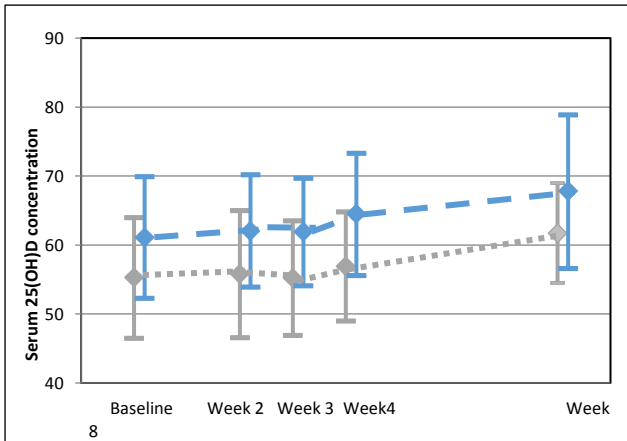


Figure 1. Mean 25(OH)D concentrations in the control (.....) and intervention group (---) during the study [♦ Mean 25(OH)D with 95% confidence interval]

The mean change in 25(OH)D concentration in the intervention group during the intervention period was 2.5 nmol/L higher than in the control group after adjustment for baseline 25(OH)D concentration, although this difference was not significant ($p=0.26$).

Discussion

We investigated the effect on 25(OH)D concentrations of delivering one SED of solar UV radiation to approximately one third of the body surface area three times per week for three weeks. The mean change in 25(OH)D concentration in the intervention group during the intervention period was more than twice as high as that of the control group (3.4 vs 1.6 nmol/l), although this was not statistically significant.

We administered nine SEDs of UV radiation, as measured by the portable electronic radiometer. However, the difference in dose between the control and intervention groups based on the polysulphone badge data was only four SEDs. This is likely due to both differences in technology and the location of the badges on the wrist rather than on a horizontal surface in direct sunlight.

The administration of nine SEDs of solar UV radiation resulted in a mean change of 25(OH)D of 2.5 nmol/L more than the background change in the control group, although this was not significant. This equates to approximately 0.3 nmol/L per SED based on the electronic dosimeter, or 0.6 nmol/L based on the badge data. This is consistent with two previous studies carried out in nursing homes and using solar UV radiation [Lovell, *et al.*, 1988; Reid, *et al.*, 1986]. Studies using artificial UV radiation sources have tended to report a greater influence on 25(OH)D concentration. For example, a study from the United Kingdom found a change of 1.3 nmol/L per SED delivered [Rhodes, *et al.*, 2010].

Although participants were wearing similar clothing as in our study, both sides of the body were exposed simultaneously; this would at least partially explain the difference.

Conclusion

The study was small, resulting in limited statistical power, and participants were exposed to a solar spectrum specific to Brisbane in spring between 10 am and approximately 12.30 pm, so the findings may not translate to other settings. However, we observed that the change in 25(OH)D concentration was approximately half of that observed in studies using artificial UV radiation, emphasising the need to conduct field studies.

References

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