

SUN BEDS, SUN AND COD LIVER OIL AS VITAMIN D SOURCES

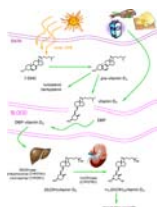
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BACKGROUND



Vitamin D synthesis and metabolism

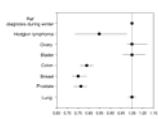
Vitamin D is a pro-hormone that have complex biological action beyond the known effect on calcium phosphorus homeostasis. Research of the recent years showed that most human tissues have receptors for vitamin D and are able to process it to active metabolites (25(OH)D and 1,25(OH)₂D). Vitamin D controls over 200 genes responsible for cell differentiation, apoptosis, angiogenesis, immunomodulation, insuline production, myocardial contractility, etc.

Humans have two sources of vitamin D: exposure to ultraviolet B radiation (UVB, 280-320 nm) and intake. Sun is believed to contribute 90 % of the circulating levels of vitamin D while very few foods contain naturally vitamin D. In the mean time it is known that sun exposure is a risk factor for skin cancer.

Vitamin D status is monitored by quantification of the serum concentrations of 25(OH)D. Optimal levels are considered to be above 80 nmol/L.

This creates a **controversy**: How much should people expose to sun in order to maximize the vitamin D production while not increasing the risk for skin cancer.

We have shown that prognosis in a cohort of over 180 000 cancer patients from Norway depends strongly on the season of diagnosis. Patients diagnosed during summer have 20 – 50 % lower risk of death compared with patients diagnosed during winter. We have hypothesised that this may be explained by the difference in vitamin D status at the time of diagnosis and therapy start.



Seasonal variation of cancer prognosis

AIMS OF THE STUDY

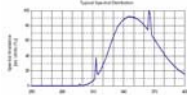
- to determine the contribution of moderate sun bed exposure to serum 25(OH)D levels;
- to estimate the decay time of a high 25(OH)D level obtained by sun bed exposure;
- to evaluate if the recommended ingestion of vitamin D is sufficient to maintain the 25(OH)D concentration obtained by sun bed exposure;
- to evaluate the contribution of recreational sun exposure (mid-summer, at the Black Sea) to increase serum levels of 25(OH)D.

MAT & MET

Volunteers in the sun bed – vitamin D intake study

Ten volunteers (20 – 35 y.o.), skin type I and II, living in Oslo, Norway were whole body exposed twice a week to the radiation of a sun bed (Life Sun S 100 W, Wolff System), starting with 0.5 MED (minimal erythema dose) and escalating to up to 1 MED per exposure for 4 weeks. After that, half of the volunteers were given a daily supplement of 200 IU vitamin D in the form of cod liver oil capsules, while the other half of the persons received no supplements.

The study took place in the months October to February when sun is too weak to induce any vitamin D.



Spectral characteristics of the sun bed lamps used in this study.



Cod liver oil capsules, Peter Møller, Norway

2 capsules = 200 IU = recommended dose

Volunteers in the Black Sea study

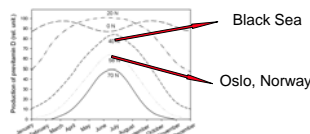
Six volunteers (21 – 35 y.o., skin type II) were whole body exposed every day for variable duration (total exposure 7-108 hours), in the period July – August.

Blood samples

Blood was sampled at baseline and weekly in the sun bed study and at baseline and at the end of exposure in the Black Sea study. Serum was separated by centrifugation and stored at -20 C. Samples were analysed in one batch using an HPLC assay.

RESULTS

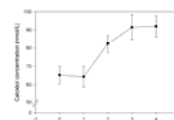
Seasonal variation of UV radiation weighted with the action spectrum for previtamin D synthesis at different latitudes.



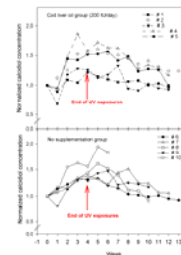
Comparision of UV intensities in the sunbed with the misummer sun in Oslo.

	UVA (mW/cm ²)	UVB (mW/cm ²)
Sun bed	16	0.25
Sun in Oslo (16.07.2007)	4.3	0.22

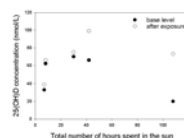
Increase in the serum 25(OH)D levels after biweekly sun bed exposures



1. No erythema was observed at any time during the study.
2. The base level was in the range 40 – 100 nmol/L. (late autumn-winter).
3. On the average the level was raised from 65 nmol/L to 92 nmol/L, i.e. by 40 %.
4. After the end of the sun bed exposure, serum levels decayed within eight weeks to levels similar to the initial state. This was true for both vitamin D-treated and control groups.



Increase in the serum 25(OH)D levels after sun exposure at the Black Sea



1. The base level was in the range 20 – 70 nmol/L. (midsummer). One volunteer had severe vitamin D deficiency (20 nmol/L).
2. In five out of six volunteers serum 25(OH)D increased, the level of increase being in the range 7 – 265 %.
3. The volunteer that was deficient in vitamin D (20 nmol/L) had the highest increase in serum 25 (OH)D.

CONCLUSIONS

1. Moderate exposures to a commercial sun bed give large improvements of the vitamin D status.
2. Ten sun bed exposures during winter brought serum 25(OH)D at typical summer levels.
3. Recommended vitamin D intake (200 IU) failed to prevent 25(OH)D decay after termination of sun bed exposures.
4. Recreational exposure at the Black Sea induced an increase in the circulating 25(OH) but the study was too small to draw conclusions on dose-response patterns.
5. Our cancer epidemiological data seem to indicate that maintaining summer vitamin D levels throughout the year may increase significant the survival.

FUTURE WORK

1. Comparison of the efficiency of two exposure patterns in elevating 25(OH)D levels -FINISHED-
2. Comparison of the efficiency of sun bed exposure to high vitamin D intake (1500 IU) - 2007 - 2008.

