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The Role of Sunlight Exposure in Reduction of Cardiovascular Diseases

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Cholesterol is a precursor to vitamin D. Since sunlight is required to turn cholesterol into vitamin D, avoiding the sun will likewise undermine our ability to synthesize vitamin D.

Vitamin D deficiency has been associated with the prevalence of cardiovascular disease (CVD), suggesting that vitamin D does not participate solely in "classical" calcium metabolism in bone, the intestines, and the kidney. Both the consumption of vitamin D from foods containing and/or fortified with vitamin D_3 (cholecalciferol) and sun light exposure increases vitamin D in the body [1,2]. Synthesis of vitamin D starts in the skin as a non-enzymatic process, which begins during sunlight exposure, when the absorption of ultraviolet B radiation results in transformation of 7-dehydrocholesterol, a metabolite of cholesterol that is stored in the skin, to precholecalciferol (previtamin-D_3) that is immediately converted into cholecalciferol (vitamin D_3). Then cholecalciferol is transported to the liver where it undergoes hydroxylation, which results in formation of calcidiol or 25-hydroxyvitamin D ($25(OH)D_3$). The second metabolic process takes place in the kidney, where calcidiol undergoes hydroxylation at the C-1 position to the hormonal, resulting in the most active metabolite known as 1,25-dihydroxyvitamin D (calcitriol) [3]. Serum $25(OH)D_3$ (calcidiol) is the major storage form of vitamin D that its serum level is a clinical indicator of overall vitamin D_3 status. [4]. Previous studies in Caucasians have shown that serum levels of $25(OH)D_3$ were inversely related to hypertension, diabetes, carotid atherosclerosis, myocardial infarction, congestive heart failure, stroke, microalbuminurea, and kidney dysfunction. However, neither the role of vitamin D deficiency in the development of CVD nor the practical recommendation for its supplementation to prevent CVD has been established [5]. Many previous studies have found that vitamin D deficiency is an independent risk factor for cardiovascular disease [5-7] and type 2 diabetes [8]. Although, Rajasree et al revealed arteriotoxicity evidence of vitamin D overload in South Indian patients, and there was an elevated level of serum 25-hydroxyvitamin D_3 in patients with ischemic heart disease [9]. It has been confirmed that cholesterol level increased during winter months. Furthermore, there was evidence that cholesterol level diminished by outdoor activity [10]. Investigators in South Africa performed a study on black and white teachers. They have found that blacks demonstrated a substantially higher prevalence of CVD compared to whites [11].

Conclusion

Sunlight exposure with consumption of cholesterol for vitamin D synthesis might be the protective factor from CVD. Therefore, vitamin D supplement is questionable in regard to CVD prevention. This could also explain the higher rate of CVD among people with dark skin who live in cold climate countries where the effect of reduced sunlight exposure is more prominent. Therefore, the prevalence of cardiovascular diseases according to in-door and out-door occupation might be different. The average duration of sunlight exposure could have an effect on cholesterol level.

References

1. Lee JH, O'Keefe JH, Bell D, Hensrud DD, Holick MF (2008) Vitamin D deficiency an important, common, and easily treatable cardiovascular risk factor? J Am Coll Cardiol 52: 1949-1956.
2. Abuannadi M, O'Keefe JH (2011) Vitamin D and cardiovascular health. Prim Care Cardiovasc J 4: 59-62.
3. Wranicz J, Szostak-Węgierek D (2014) Health outcomes of vitamin D. Part I. characteristics and classic role. Rocz Panstw Zakl Hig 65: 179-184.

4. Holick MF (2007) Vitamin D deficiency. N Engl J Med 357: 266-281.
5. Park S, Lee BK (2012) Vitamin D deficiency is an independent risk factor for cardiovascular disease in Koreans aged ≥ 50 years: results from the Korean National Health and Nutrition Examination Survey. Nutr Res Pract 6: 162-168.
6. Kendrick J, Targher G, Smits G, Chonchol M (2009) 25-Hydroxyvitamin D deficiency is independently associated with cardiovascular disease in the Third National Health and Nutrition Examination Survey. Atherosclerosis 205: 255-260.
7. Ginde AA, Scragg R, Schwartz RS, Camargo CA Jr (2009) Prospective study of serum 25-hydroxyvitamin D level, cardiovascular disease mortality, and all-cause mortality in older U.S. adults. J Am Geriatr Soc 57: 1595-1603.
8. Renzaho AM, Nowson C, Kaur A, Halliday JA, Fong D, et al. (2011) Prevalence of vitamin D insufficiency and risk factors for type 2 diabetes and cardiovascular disease among African migrant and refugee adults in Melbourne: a pilot study. Asia Pac J Clin Nutr 20: 397-403.
9. Rajasree S, Rajpal K, Kartha CC, Sarma PS, Kutty VR, et al. (2001) Serum 25-hydroxyvitamin D3 levels are elevated in South Indian patients with ischemic heart disease. Eur J Epidemiol 17: 567-571.
10. Grimes DS, Hindle E, Dyer T (1996) Sunlight, cholesterol and coronary heart disease. QJM 89: 579-589.
11. Hamer M, von Känel R, Reimann M, Malan NT, Schutte AE, et al. (2015) Progression of cardiovascular risk factors in black Africans: 3 year follow up of the SABPA cohort study. Atherosclerosis 238: 52-54.