The recent article by Skaaby and colleagues (1) reported that there were no significant associations between baseline serum 25-hydroxyvitamin D [25(OH)D] levels and incident internal cancer rates during an 11.3-year median follow-up time. One reason for the failure to find significant associations is the long follow-up time, mentioned in passing in the article. This effect has been studied with the finding that for follow-up times greater than 4 years for breast cancer and 13 years for colorectal cancer it is very unlikely to find significant inverse associations due to changes in 25(OH)D levels (2). Mean serum 25(OH)D levels for like-age populations in their study decreased from 65 nmol/L in 1993–4 to 52 nmol/L in 1999–2001 and 44 nmol/L in 2006–8 (Table 2 in ref. 1). Thus, the 25(OH)D levels of the Monica10 and Inter99 cohorts, representing 90% of incident cancers, were not constant during the follow-up period.

Another problem is that a linear relation between serum 25(OH)D level and risk of cancer incidence was assumed. As shown in meta-analyses of breast and colorectal cancer incidence rates with respect to serum 25(OH)D level, the relation is nonlinear, with rapid decrease for small increases from low 25(OH)D levels and little change for increases beyond 70 nmol/L (3).

Although prospective population studies have found mixed associations between baseline serum 25(OH)D levels and cancer incidence rates, ecologic and occupation studies have found very strong inverse associations between indices of solar ultraviolet B (UVB) doses and cancer incidence and/or mortality rates (4, 5). The advantages of such studies include the large number of cases, the fact that people have generally lived in the same location for many years so that the UVB index is applicable for a long period, and that many other cancer risk-modifying factors can generally be included in the analysis. Such studies have found significant inverse correlations between solar UVB indices and 20 types of cancer, with nine types being found in at least four different countries or regions: bladder, breast, colon, esophageal, gall bladder, gastric, pancreatic, rectal cancer, and leukemia (4). A possible drawback to such studies is that there may be effects of solar UVB irradiance in reducing risk of cancer in addition to production of vitamin D; however, no mechanisms have been proposed to explain other UVB effects for cancer risk. The mechanisms whereby vitamin D reduces the risk of cancer incidence, progression, and metastasis are well known (4).

Disclosure of Potential Conflicts of Interest

W.B. Grant reports receiving a commercial research grant from Bio-Tech Pharmacal and The Sunlight Research Forum (Veldhoven).

Received May 14, 2014; accepted May 29, 2014; published online September 2, 2014.

References

5. Grant WB. Role of solar UV irradiance and smoking in cancer as inferred from cancer incidence rates by occupation in Nordic countries. Dermatoendocrinol 2012;4:203-11.
Vitamin D and Cancer Incidence—Letter from Grant

William B. Grant


Updated version
Access the most recent version of this article at:
http://cebp.aacrjournals.org/content/23/9/1950

Cited articles
This article cites 5 articles, 1 of which you can access for free at:
http://cebp.aacrjournals.org/content/23/9/1950.full.html#ref-list-1

Citing articles
This article has been cited by 1 HighWire-hosted articles. Access the articles at:
/content/23/9/1950.full.html#related-urls

E-mail alerts
Sign up to receive free email-alerts related to this article or journal.

Reprints and Subscriptions
To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at pubs@aacr.org.

Permissions
To request permission to re-use all or part of this article, contact the AACR Publications Department at permissions@aacr.org.