Burden of Disease associated with low Vitamin D status in New Zealand

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Abstract. Low vitamin D status is associated increased risk of all-cause mortality. The New Zealand population has low vitamin D levels, with marked ethnic variations; and paradoxically high rates of skin cancer. We carried out a comparative risk analysis, to estimate the avoidable disease burden for vitamin D, by comparing the current population distribution of 25-hydroxyvitamin D [25(OH)D] with two counterfactual distributions. The numbers of all deaths avoided by increasing the population mean 25(OH)D level by 10 nmol/L (males 500, females 510), and from increasing it up to 100 nmol/L (males 2430, females 2660), were much higher those from all skin cancers combined (males 260, females 140). The higher mortality burden from low vitamin D status, than from sun-induced skin cancer, has implications for the current policy on sun exposure and vitamin D intake.

Background

Evidence that vitamin D may protect against a wide range of diseases is rapidly increasing. Cohort studies have shown that low blood levels of 25-hydroxyvitamin D [25(OH)D], the metabolite that best reflects vitamin D status, predicts increased risk of colorectal cancer (2008) and cardiovascular disease (Pittas, et al.). In addition, a recent meta-analysis of randomised clinical trials has shown that vitamin D supplementation reduces all cause mortality (Autier and Gandini, 2007).

Levels of 25(OH)D in the range of 90-100 nmol/L are considered optimal for health (Bischoff-Ferrari, et al., 2006). However, mean 25(OH)D levels in New Zealand are much lower than this, being: Pacific Island people 37 nmol/L, Maori 42 nmol/L, and New Zealand European/Others (NZEO) 52 nmol/L (Rockell, et al., 2006). These low levels are a paradox, since > 90% of vitamin D is synthesized from sun exposure (Holick, 2004), yet the New Zealand population has one of the highest melanoma rates (Sneyd and Cox, 2006).

Because of the low vitamin D levels in New Zealand, we decided to estimate the number of deaths that might be avoided by increasing 25(OH)D levels in the general population. We used the World Health Organisation Burden of Disease method (Murray, 1996), and compared these with the number of deaths from skin cancer caused by sun exposure.

Methods

Full details of the methods and results have been described (Grey, et al., 2010). Briefly, we searched the medical literature to identify five cohort studies which compared blood 25(OH)D levels measured at baseline with subsequent risk of all-cause mortality. We then estimated linear regression coefficients from mortality hazard ratios for 25(OH)D categories published in these cohort studies, and calculated a pooled estimate of the relative risk of all-cause mortality per 1 nmol/L increase in blood 25(OH)D (= 0.0042; se = 0.00102).

This regression coefficient was then applied to mortality data for the New Zealand population aged ≥30 years (average of 2002 and 2003), in two counterfactual scenarios using the Burden of Disease comparative risk assessment method (Murray, 1996).

This involved comparing the number of deaths under the current distribution of 25(OH)D blood levels in New Zealand, from the 1997 national nutrition survey (Rockell, et al., 2006) with the scenario of: (1) increasing mean 25(OH)D levels by 10 nmol/L, which is feasible in the short term through public health strategies (eg. increased sun exposure, nutritional supplements or food fortification); and (2) increasing mean 25(OH)D levels up to 100 nmol/L, to provide an estimate of the potential full benefit at 25(OH)D levels associated with optimal health outcomes (Bischoff-Ferrari, et al., 2006).

The combined number of deaths from all skin cancers due to excessive sun exposure was estimated by applying standard population attributable fractions previously used for melanoma (90%) and for squamous cell carcinoma and basal cell carcinoma (each 70%) (Lucas, et al., 2006).

Results

The proportions of all deaths avoidable from increasing the population mean 25(OH)D by 10 nmol/L (4%), and from increasing it up to 100 nmol/L (males 18%, females 19%), were much lower than the proportions of skin cancer deaths attributed to sun exposure (Figure 1).

![Figure 1](image-url)
causes) avoidable from increasing the population mean 25(OH)D by 10 nmol/L (males 500, females 510), and from increasing it up to 100 nmol/L (males 2430, females 2660), were much higher those from all three skin cancers combined (males 260, females 140) (Figure 2).

The ratio of deaths from comparing all deaths avoidable by increasing population mean 25(OH)D levels by an achievable 10 nmol/L, with skin cancers deaths attributable to sun exposure, is shown in Figure 3, by ethnic group. In all groups, the ratio is well above 1, indicating that public health strategies to increase vitamin D levels have the potential to prevent more deaths than are currently caused by excessive sun exposure. The ratio is much higher among Maori and Pacific Island people, due to a combination of their high all-cause mortality rates and low skin cancer rates, compared to NZEO.

Figure 2. Number of all deaths avoided by increasing 25(OH)D, and number of skin cancer deaths attributed to sun exposure (blue = males, red = females).

Figure 3. Ratio of deaths avoided by increasing 25(OH)D by 10 nmol/L, to deaths attributable to sun exposure – by ethnicity (blue = males, red = females).

Discussion

Our results suggest that a greater burden of disease could be avoided by increasing the vitamin D status of the general population than that attributable to excessive sun exposure. The potential benefits from increasing vitamin D status are greatest for Pacific Island and Maori peoples; and current policies around sun avoidance may not be appropriate for them, as they have both low vitamin D levels and low rates of skin cancer.

The major limitation of our analyses is that we have assumed a causal association between low vitamin D levels and increased risk of all-cause mortality. Large scale clinical trials are needed to confirm the recent meta-analysis of previous studies, primarily carried out to see if vitamin D (with calcium) prevents fractures, which showed a reduction in all-cause mortality (Autier and Gandini, 2007). If vitamin D is beneficial in clinical trials, a range of public health strategies could be used to increase population vitamin D levels, including safe sun exposure (without burning), increased availability of vitamin D supplementation and food-fortification.

References


