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ORIGINAL ARTICLE

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Extra Vitamin D Supplementation Does Not Reduce the Effects of Aging

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ABSTRACT

A recent study published in the American Journal of Clinical Nutrition claims to prove that if older people take 2000 IU of vitamin D3 supplement daily it will reduce the age-related shortening of their DNA telomeres and thereby could lead to a "three-year decrease in aging." The present author's reanalysis of the study, however, reveals that telomere shortening did not reliably differ between the supplementary vitamin D3 treatment group and the placebo control group after two years or even four years of treatment. Vitamin D supplementation is widely recommended for maintaining bone health but the claim that it can slow down aging is baseless.

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Introduction

"Vitamin D can help turn back the clock" was the sensational headline of an article in *The Australian* newspaper on May 23, 2025, reprinted from the U.K.'s *The Times* [1]. The article was referring to a four-year clinical trial of vitamin D3 supplementation versus placebo treatment reported recently in the *American Journal of Clinical Nutrition* in which the researchers, led by Haidong Zhu, PhD, claimed that adults over 50 years of age who took 2000 IU (international units) of vitamin D3 supplement daily for up to four years showed reduced age-related shortening of their telomeres – the cap on the end of each DNA chromosome that protects against the effects of disease-caused inflammation and cell deterioration due to natural aging [2]. The researchers, though cautious in their conclusions in the Abstract of their article, speculated in the article itself that vitamin D supplementation, by reducing telomere shortening, "could mean a 3-year decrease in aging" ([2], online p. 7), a claim which most readers would take to mean that they might get three years or longer to live. The authors were far less tentative elsewhere. In a *Medscape* Commentary that appeared just before the paper was published, JoAnn Manson, a professor at the Harvard Medical School and the only MD on Zhu et al.'s research team, wrote: "What we found was that vitamin D supplementation did, in fact, slow telomere shortening. In the placebo group, there was substantial shortening over the 4 years; and in the vitamin D group there was a very minimal shortening over that same period… We estimated that this difference amounted to a 3-year decrease in aging." [3]. Similar one-sidedly favorable reports on the study can be found if you search for recent vitamin D findings online.

I am a research expert, not a medico, but I routinely check the original version of popularly publicized medical studies, especially now in my later years when like many others I have become interested in any breakthrough that might give me a bit more time or, to be truthful, might make me look a bit more youthful. Alas, as I will show in this article, a close reading of Zhu et al.'s study reveals that vitamin D3 supplementation has no significant effect on telomere shortening, and therefore is unlikely to be able to help with aging.

Methodological Problems with the Study

Zhu et al.'s study was part of a U.S. nationwide double-blind clinical trial [4] called The Vitamin D and Omega A-3 Trial (VITAL), although the telomere measurement study was conducted only with people residing in the Boston area. Zhu et al.'s study [2] began with baseline telomere length measurement, and follow-up measurements were taken after two years of high-dose vitamin D treatment or placebo treatment and again after four years of treatment. The participants were males aged 50+ and females 55+, with the sample averaging 65 years of age overall, ethnically 84% Whites and 8% Blacks, with exclusions for people with a history of cancer apart from skin cancer, or with serious life-shortening medical conditions such as cardiovascular disease, kidney failure, or cirrhosis. Included were those who agreed to have their telomeres measured via blood-sample genetic testing, which many if not most people are reluctant to undergo, and which, with the albeit experimentally necessary exclusion of people with serious diseases, raises a big question about the population representativeness of the findings [5].

Another problem with the methodology was that the placebo group was not actually a placebo group but rather a "mixed low-dose vitamin D3" group, so that the study was really a comparison of low-dose vitamin D3 supplementation versus high-dose vitamin D3 supplementation [5]. This occurred because the approximately 40% of participants who were taking vitamin D supplements before the trial began were told to continue but to limit their daily intake to 800 IU, with the researchers noting that it might be unethical to ask them to stop entirely [5]. However, 800 IU exceeds the 600 IU of daily vitamin D supplementation recommended by the U.S. National Institutes of Health to maintain bone health for people under age 70 and is the same as the 800 IU recommended for bone health in those over 70 [6]. This means that possibly up to half of the n = 254 participants in the placebo group were on 800 IU of vitamin D during the study, while all of the n = 252 participants in the vitamin D3 treatment group were on 2,000 IU daily with about 40% of them on 2,800 units – hence my titling of this

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paper as being a study of *extra* vitamin D3 supplementation, with the extra being an unusually high dose.

Analysis Problems with the Study

As has become typical in academic research, Zhu et al. used an elaborate statistical analysis, in their case a repeated-measures (telomere length at baseline, end of year 2, end of year 4) factorial analysis of variance with a between-groups (vitamin D3, placebo) comparison of means. I will focus on their model 1 results because, as in the real world, this model makes no adjustments for demographics or so-called lifestyle factors including obesity or smoking or other medications taken – adjustments that in any case made no substantial difference if you look at their adjusted results in model 2 and model 3 (see their Table 2 on the fourth page of their article [2]). Also, I will focus on the statistical confidence intervals that were reported in their table rather than on the p-values. As Cumming [7] has shown, p-values are notoriously unstable, and a much better test is to examine whether the 95% confidence intervals around the means overlap, in which case the correct conclusion is that the means are not significantly different.

My Table 1 shows the mean telomere length findings (in bold) together with the 95% confidence limits around them, with overlaps indicated by the right brackets]. Looking first at the vitamin D3 treatment group over the first two years, it can be seen that the upper limit (UL) of the end-year 2 telomere length overlaps with the lower limit (LL) of the baseline telomere length, meaning that there was no statistically reliable reduction in telomere length after two years of high-dose vitamin D3 supplementation, which at first seems to support the researchers' contention of prevention of telomere shortening. However, this is obviated by the fact that the same result was observed in the placebo group over the two years. Nor did the extension of high-dose vitamin D3 treatment to four years reduce shortening significantly. And if no effect was shown in four years, no one could be expected to continue high dosing in the hope that a reduction effect might show up later. The inference to be drawn from Zhu et al.'s study is that even with a favorably selective sample, high-dose vitamin D3 supplementation does not reduce telomere length and therefore cannot have any effect on aging.

Table 1: Mean telomere length measured in kilobase units (in bold) and upper and lower 95% confidence limits in the vitamin D3 group and the placebo group at baseline and at year 2 end and year 4 end.

Time point	Vit D3 group		Placebo group	
Baseline	UL	8.90	UL	8.96
	Mean	**8.78**	Mean	**8.73**
	LL	8.65	LL	8.61
Year 2 end	UL	8.86	UL	8.74
	Mean	**8.73**	Mean	**8.61**
	LL	8.59	LL	8.49
Year 4 end	UL	8.89	UL	8.71
	Mean	**8.75**	Mean	**8.57**
	LL	8.62	LL	8.43

Note: Right brackets (]) show the overlapping 95% confidence limits.

Suppose for a moment that by chance Zhu et al.'s mean telomere length findings, as most people have assumed, are numerically accurate. There are still important unanswered questions. Apparently, according to several researchers cited in Wikipedia [8], it is not telomere shortening itself that matters but rather the rate of shortening. My Table 2 shows the means and the percentage change (in bold) in average telomere length for the two groups over two years' time and over four years' time, which are essentially the average two-year change rate and the average four-year change rate, respectively. The problematic results in this table are that the shortening in the high-dose vitamin D group appeared to reverse so that this group's telomeres appeared strangely to lengthen slightly after two years of treatment, and that in the placebo group the natural age-related rate of shortening appeared strangely to slow in the second two-year period. A likely explanation is that the telomere length measures were not accurate and that the whole study suffered from measurement error.

Table 2: Mean telomere length and percent change (in bold) in the vitamin D3 group and the placebo group from baseline to year 2 end and year 4 end.

	Vit D3 group		Placebo group	
Time point	**Mean**	**Change**	**Mean**	**Change**
Baseline	8.78		8.73	
		-0.57%		**-1.37%**
Year 2 end	8.73		8.61	
		+0.23%		**-0.46%**
Year 4 end	8.75		8.57	

Conclusion

Medical researchers, as I have previously alleged, are not well trained in research methodology or statistics and accordingly most cannot be expected to understand the findings from clinical trials [9-11]. This certainly is the case with the researchers' misinterpretation of the clinical trial of high-dose supplementary vitamin D3. The danger is that the reported results are likely to be accepted by medical practitioners and by older readers of popular accounts in the media, and will be latched on to by the pharmaceutical companies that manufacture vitamin D supplements and the retail pharmacies and supermarkets that sell them. Of course, there are good reasons for vitamin D supplementation [5] but a reduction in aging is not one of them, and it is important that this recent propaganda about vitamin D and aging is immediately dispelled.

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