

## Menopause, vitamin D, and oral health

(AUGUST 2009)

**TO THE EDITOR:** Buencamino and colleagues<sup>1</sup> reviewed the association between menopause and periodontal disease. However, they did not mention the role of vitamin D status in this setting.

Vitamin D status is usually divided into three categories based on serum 25-hydroxyvitamin D levels: “deficient” ( $\leq 15$  ng/mL), “insufficient” (15.1–29.9 ng/mL), and “sufficient” ( $\geq 30$  ng/mL). Serum 25-hydroxyvitamin D levels have been decreasing significantly for more than a decade, and as a result, a majority of the US population has a vitamin D insufficiency.

In the third National Health and Nutrition Examination Survey (NHANES III), a large US population survey, a low serum 25-hydroxyvitamin D concentration was independently associated with periodontal disease.<sup>2</sup> In particular, it was significantly associated with loss of alveolar attachment in persons older than 50 years of both sexes, independent of race or ethnicity; women in the highest 25-hydroxyvitamin D quintile had, on average, 0.26 mm (95% confidence interval 0.09–0.43 mm) less mean attachment loss than did women in the lowest quintile. Furthermore, in a randomized trial, supplementation with vitamin D (700 IU/day) plus calcium (500 mg/day) has been shown to significantly reduce tooth loss in older persons over a 3-year treatment period.<sup>3</sup>

Osteoporosis and periodontal disease share several risk factors, and it might be speculated that these pathologic conditions are biologically intertwined.<sup>4</sup> The decreased bone mineral density of osteoporosis can lead to an altered trabecular pattern and more rapid alveolar bone resorption, thus predisposing to periodontal disease. On the other hand, periodontal infections can increase the systemic release of inflammatory cytokines, which accelerate systemic bone resorption. Indeed, vitamin D deficiency has been associated with a cytokine profile that favors greater inflammation (eg, higher levels of C-reactive protein and interleukin 6, and lower levels of interleukin 10), and vitamin D supplementation decreases circulating inflammatory markers.<sup>5</sup> This might break the vicious circle of osteoporosis, periodontal disease development, and further systemic bone resorption.

Therefore, we suggest that menopausal women

should maintain an adequate vitamin D status in order to prevent and treat osteoporosis-associated periodontal disease.

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**IN REPLY:** Dr. Mascitelli and colleagues bring up an excellent point regarding the role of vitamin D. Vitamin D deficiency (and insufficiency) is such a widespread problem that it deserves attention in both dental and medical circles, and to be fair, it deserves an article of its own. Low vitamin D has been associated with bone loss and an increased risk for certain cancers and other chronic diseases.<sup>1</sup> The literature also suggests that low levels of vitamin D are associated with periodontal disease,<sup>2</sup> and that supplementation with vitamin D (and calcium) leads to better periodontal health.<sup>3,4</sup> However, since vitamin D supplementation is not a recognized way to treat periodontitis, mentioning it with therapies adjudicated as treatment modalities (such as removal of biofilm, which we stressed in our paper) risks misinterpretation by clinicians less versed in periodontal and dental conditions in general.

Nevertheless, the comment brings to light that medical, dental, and nutritional colleagues are very

interested in learning more about the pathophysiologic commonalities in the diseases we treat and in a common postmenopausal patient cohort. Our paper focused more closely on what periodontitis is, and on the more primary etiologic pathophysiology—what common resorptive pathways it shares with osteoporosis in the postmenopausal cohort, and biofilm, the primary etiology of periodontitis. But there is need for more discussion and research into bone development (during childhood and adolescence as well) and the role of nutrition during all stages of life.

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## Prostate cancer prevention

(AUGUST 2009)

**TO THE EDITOR:** Thank you for the excellent review on prostate cancer screening and prevention by Eric A. Klein, MD, in your August 2009 issue.

Dr. Klein concludes that the results of the Prostate Cancer Prevention Trial (PCPT) and the Reduction by Dutasteride of Prostate Events (REDUCE) trial were “congruent” with respect to the magnitude of prostate cancer risk prevention, beneficial effects on benign prostatic hypertrophy, and toxicity. In other words, finasteride and dutasteride produced equivalent clinical results with respect to prostate health despite the fact that dutasteride inhibits 5-alpha-reductase types 1 and 2, while finasteride inhibits only type 2.

Over the years, many patients have been prescribed dutasteride rather than finasteride because of hopes that the former might be more effective for maintaining prostate health. In August 2009, the retail price on Drugstore.com of a 90-day supply of generic finasteride is \$190, vs \$321 for dutasteride (which is available only as branded Avodart). In my experience as a practicing primary care physician, most patients would prefer to save money by switching to the less expensive generic drug if it provides equivalent prostate health outcomes compared with the more expensive branded drug.

I would like to ask Dr. Klein's opinion on allowing patients to switch from Avodart to generic finasteride in order to save money, and on the general issue of which agent to use first-line for prostate health concerns.

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**IN REPLY:** Although the two drugs were not compared head to head, the data from randomized trials suggest that they have similar effects on the amelioration of lower urinary tract symptoms due to benign prostatic hypertrophy. The full results of the REDUCE trial are not yet available and until they are published it is not possible to comment any further on whether one or the other is the better choice for prevention of prostate cancer.

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