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https://doi.org/10.7326/L15-5094-2

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Comments and Responses

Screening for Vitamin D Deficiency

TO THE EDITOR: The U.S. Preventive Services Task Force has concluded that the current evidence is insufficient to assess the balance of benefits and harms of screening for vitamin D deficiency in asymptomatic adults. As LeBlanc and colleagues' review (1) shows, this current evidence comprises studies whereby supplementation (variable doses of vitamin D2 or D3 with or without calcium) has been monitored mainly by measuring total concentrations of 25-(OH)D. Instead, future studies should focus on assessing concentrations of bioavailable vitamin D.

Concentrations of total 25-(OH)D and 1,25-dihydroxyvitamin D measured routinely in daily practice are known to differ from those of bioavailable vitamin D. Free and bioavailable vitamin D concentrations depend on the vitamin D–binding protein and ethnicity (2). In addition, we need evidence of techniques to increase concentrations of bioavailable vitamin D, such as obtaining more outdoor physical activity, which might increase biosynthesis and bioavailable concentrations of vitamin D3 and circumvent hypervitaminosis D (3). Harmful effects of hypervitaminosis D might be due solely to excess biosynthesized sequestered vitamin D as a result of inappropriate oral supplementations and of not being converted to active bioavailable vitamin D. Excess vitamin D is arteriotoxic and causes elastocalcinosis, which induces destruction of elastic fibers, which leads to arterial stiffness and causes arterial calcification through upregulation of 1,25-dihydroxyvitamin D3 receptors and increased calcium uptake in smooth-muscle cells of the arteries (4) (5).
Research resources are finite in these times of austerity; hence, they should be allocated appropriately. Robust and pertinent evidence is needed to formulate educational and interventional policies that can be implemented to prevent the global public health problem of cardiometabolic diseases and autoimmune and neoplastic conditions associated with decreased bioavailable concentrations of vitamin D.

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Disclosures: Authors have disclosed no conflicts of interest. Forms can be viewed at www.acponline.org/authors/icmje/ConflictOfInterestForms.do?msNum=L15-0124.
References:


