Suzanne P Murphy

Cancer Research Center of Hawaii
Honolulu, HI (retired)

Christine L Taylor

Office of Dietary Supplements, NIH
6100 Executive Boulevard
Bethesda, MD 20892-7517
E-mail: taylorcl3@od.nih.gov

REFERENCES

1. Ng K, Scott JB, Drake BF, Chan AT, Hollis BW, Chandler PD, Bennett GG, Giovannucci EL, Gonzalez-Suarez E, Meyerhardt JA, et al. Dose response to vitamin D supplementation in African Americans: results of a 4-arm, randomized, placebo-controlled trial. Am J Clin Nutr 2014;99:587–98.
2. Institute of Medicine. Dietary Reference Intakes: applications in dietary assessment. Washington, DC: The National Academies Press, 2000.
3. Institute of Medicine. Dietary Reference Intakes: applications in dietary planning. Washington, DC: The National Academies Press, 2003.
4. Murphy SP, Barr SI. Practice paper of the American Dietetic Association: using the Dietary Reference Intakes. J Am Diet Assoc 2011;111:762–70.
5. Trumbo PR, Barr SI, Murphy SP, Yates AA. Dietary Reference Intakes: cases of appropriate and inappropriate uses. Nutr Rev 2013; 71:657–64.
6. Taylor CL, Carriquiry AL, Bailey RL, Sempos CT, Yetley EA. Appropriateness of the probability approach with a nutrient status biomarker to assess population inadequacy: a study using vitamin D. Am J Clin Nutr 2013;97:72–8.
7. Sempos CT, Druazo-Arvizu RA, Dawson-Hughes B, Yetley EA, Looker AC, Schleicher RL, Cao G, Burt V, Kramer H, Bailey RL, et al. Is there a reverse J-shaped association between 25-hydroxyvitamin D and all-cause mortality? Results from the U.S. nationally representative NHANES. J Clin Endocrinol Metab 2013;98:3001–9.
8. Institute of Medicine. Dietary Reference Intakes: calcium and vitamin D. Washington, DC: The National Academies Press, 2011.
9. Barrett-Connor E, Siris ES, Wehren LE, Miller PD, Abbott TA, Berger ML, Santora AC, Sherwood LM. Osteoporosis and fracture risk in women of different ethnic groups. J Bone Miner Res 2005;20:185–94.
10. Powe CE, Evans MK, Wenger J, Zonderman AB, Berg AH, Nalls M, Tamez H, Zhang D, Bhan I, Karumanchi A, et al. Vitamin D-binding protein and vitamin D status of black Americans and white Americans. N Engl J Med 2013;369:1991–2000.

doi: 10.3945/ajcn.114.090605.

Reply to PM Brannon et al

Dear Sir:

We thank Brannon et al for their thoughtful comments on our study (1), and greatly appreciate the opportunity to reply. In their correspondence, they incorrectly state that we defined vitamin D adequacy as 97.5% of the population achieving a 25-hydroxyvitamin D [25(OH)D] concentration of 20 ng/mL and raise concern about potential misapplication of the Recommended Dietary Allowance (RDA) for vitamin D intake in our analysis. They discuss that the RDA should not be used to assess vitamin D adequacy in populations or groups, because, by definition, the RDA-associated concentration of 20 ng/mL reflects a value that exceeds the needs of most individuals. They suggest that the correct approach is to estimate how much vitamin D is needed to ensure a low prevalence of plasma 25(OH)D below the estimated average requirement (EAR) of 16 ng/mL (2).

Although we agree with their statement on appropriate applications of the RDA, it is clear that Brannon et al misinterpreted the intent of our analysis. The EAR of 400 IU and RDA of 600 IU for adults up to age 70 y calculated by the Institute of Medicine (IOM) to be associated with plasma 25(OH)D concentrations of 16 and 20 ng/mL, respectively, are based on dose-response curves from studies in white populations. Given the lower baseline concentrations of 25(OH)D among African Americans, differences in lifestyle behaviors, and differences in germline genetic variation and vitamin D metabolism that have not yet been fully characterized, it is important to reexamine the vitamin D requirement associated with these target 25(OH)D concentrations by using dose-response curves that are actually constructed from studies in African Americans, rather than extrapolating from curves from whites. Until now, there have been very few studies evaluating a variety of vitamin D intakes in a large-enough cohort of African Americans with minimal UV-B radiation exposure to reliably construct such a dose-response curve. Our trial randomly assigned 328 community-based African Americans in Boston, MA, to receive placebo or 1000, 2000, or 4000 IU of vitamin D_3/d for 3 mo during the winter, with plasma 25(OH)D concentrations assayed at baseline and at 3 and 6 mo. By using the resulting robust dose-response curve, we found a vitamin D requirement of 1640 IU/d to be the amount that corresponded to the RDA-associated 25(OH)D concentration of 20 ng/mL in African Americans. In fact, even when we consider the lower EAR-associated 25(OH)D concentration of 16 ng/mL as our target, as recommended by Brannon et al, the vitamin D intake required to reach that value in $\geq$97.5% of African Americans is still 1200 IU/d (95% CI: 1130, 1420 IU/d) (**Figure 1**).

A similar approach to determine the vitamin D intake corresponding to the RDA-associated 25(OH)D concentration was undertaken by Gallagher et al (3), who randomly assigned 163 postmenopausal white women with 25(OH)D $\leq$20 ng/mL to 7 doses of vitamin D_3 compared with placebo for 12 mo. By using the dose-response curve that resulted from this study in whites, the authors were able to confirm the IOM RDA of 600–800 IU/d, supporting the appropriateness of the methodology. No concerns have been published about misuse of the RDA in that study.

Brannon et al also state that we did not account for "background" intake of dietary vitamin D. However, all subjects in our study were administered dietary and lifestyle questionnaires, and Table 1 in our article clearly shows that in this representative cohort of African Americans, the contribution of vitamin D from dietary sources was extremely low at $<$200 IU/d at baseline (1). Moreover, plasma 25(OH)D concentrations reflect contributions from all sources of vitamin D, including diet; therefore, our determination of the vitamin D_3 dose associated with specific target 25(OH)D concentrations can be interpreted as the amount of vitamin D_3 needed in addition to underlying dietary intake.

Finally, Brannon et al raise concerns about vitamin D toxicity at doses near or above the Tolerable Upper Intake Level; however, we did not see any evidence of clinically significant hypercalcemia in our African American cohort, including those treated with 4000 IU vitamin D_3/d for 3 mo (1). They also claim that high doses of vitamin D supplementation may pose special concerns for African Americans; they cite a study from the NHANES cohort that reported a reverse J-shaped relation between 25(OH)D concentrations and all-cause mortality, with an adjusted RR of 2.4 for blacks with serum 25(OH)D $\geq$48 ng/mL compared with ~30–40 ng/mL, whereas the adjusted RR was 1.6 for the same comparison in whites (4). However, Brannon et al fail to point out that blacks comprised only 10% of the population in that study, for which the abstract explicitly states, "the study was too small to evaluate the association in non-Hispanic black . . . adults." This statement is supported by a very telling, extremely wide 95% CI

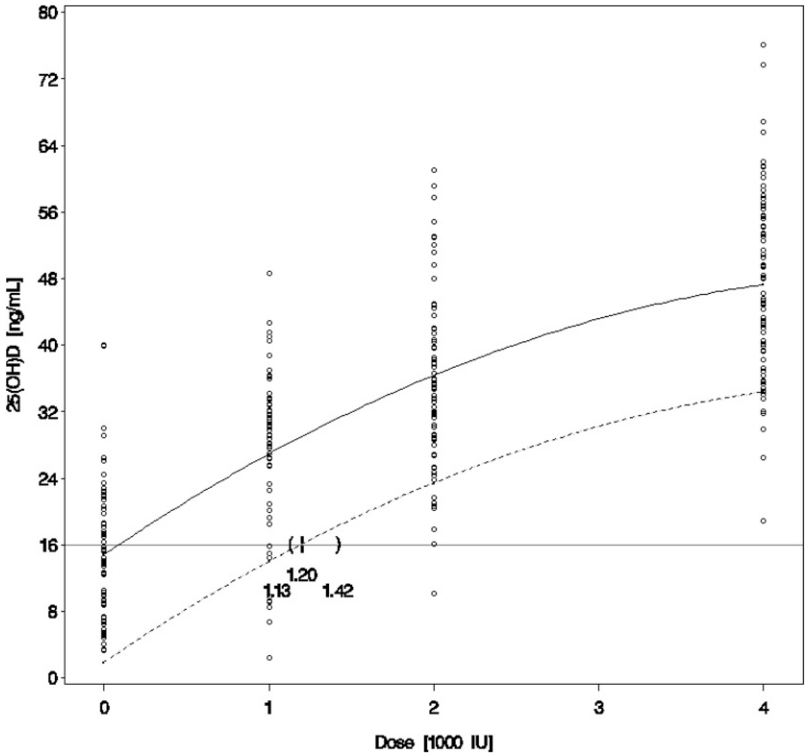

FIGURE 1. Plasma 25(OH)D concentrations at 3 mo and dose of vitamin D_3 supplementation ($n = 71$ for placebo; $n = 67$ for 1000 IU/d; $n = 76$ for 2000 IU/d; $n = 78$ for 4000 IU/d). The solid line is a quadratic fit to the observed mean plasma 25(OH)D concentration. The dashed line falls below the mean line by 1.96 SDs of the distribution of the estimated within-subject mean concentration (obtained from the random patient effect in the mixed model) and represents the empirical Bayesian prediction interval to bound 97.5% of future subjects' mean plasma 25(OH)D concentration. This prediction interval crosses the 16-ng/mL line at 1200 IU/d (95% CI: 1130, 1420 IU/d), indicating that an estimated dose of 1200 IU vitamin D_3/d is required to achieve a mean plasma 25(OH)D $\geq$16 ng/mL in $\geq$97.5% of the study population. 25(OH)D, 25-hydroxyvitamin D.

surrounding the RR of 2.4 for blacks (95% CI: 0.8, 7.0), which was pointedly omitted from the body of their letter. In addition, Brannon et al also did not mention that the RRs for death were adjusted for only age, sex, race-ethnicity, and season, and that all significant associations between serum 25(OH)D $\geq$48 ng/mL and increased mortality disappeared when the model was adjusted for additional critical confounding variables, such as BMI, physical activity, and various comorbidities and socioeconomic factors. Last, the suggestion that African Americans are adapted to low circulating 25(OH)D and may therefore be particularly susceptible to vitamin D toxicity seems unlikely in light of observations that African hunter-gatherers in Tanzania with year-round UV-B exposure have mean circulating 25(OH)D concentrations of 46 ng/mL (5).

In conclusion, the intent of our RDA analysis was not to define vitamin D adequacy in a population of African Americans but rather to provide a more accurate estimation of the vitamin D intake that is associated with the IOM-determined 25(OH)D target concentration of 20 ng/mL by using dose-response curves constructed from a rigorous, randomized clinical trial of vitamin D supplementation in African Americans. We believe these dose estimates of vitamin D from race-specific dose-response curves provide a better assessment of the relation between vitamin D supplementation and change in 25(OH)D concentrations in African Americans than does extrapolation from data obtained from white populations.

BWH has received support from DiaSorin SpA for serving as an academic consultant. No other relevant financial disclosures or conflicts of interest were reported by the authors for themselves or their spouses, partners, or children.

Kimmie Ng

Department of Medical Oncology
Dana-Farber Cancer Institute
450 Brookline Avenue
Boston, MA 02215
E-mail: kimmie_ng@dfci.harvard.edu

Bruce W Hollis

Division of Pediatrics
Medical University of South Carolina
Charleston, SC

Edward L. Giovannucci

Department of Nutrition
Harvard School of Public Health
Boston, MA

Charles S. Fuchs

Department of Medical Oncology
Dana-Farber Cancer Institute
Boston, MA

REFERENCES

1. Ng K, Scott JB, Drake BF, Chan AT, Hollis BW, Chandler PD, Bennett GG, Giovannucci EL, Gonzalez-Suarez E, Meyerhardt JA, et al. Dose response to vitamin D supplementation in African Americans: results of a 4-arm, randomized, placebo-controlled trial. Am J Clin Nutr 2014;99:587–98.
2. Institute of Medicine. Dietary Reference Intakes for Calcium and Vitamin D. Washington, DC: The National Academies Press, 2011.
3. Gallagher JC, Sai A, Templin T 2nd, Smith L. Dose response to vitamin d supplementation in postmenopausal women: a randomized trial. Ann Intern Med 2012;156:425–37.
4. Sempos CT, Durazo-Arvizu RA, Dawson-Hughes B, Yetley EA, Looker AC, Schleicher RL, Cao G, Burt V, Kramer H, Bailey RL, et al. Is there a reverse J-shaped association between 25-hydroxyvitamin D and all-cause mortality? Results from the U.S. nationally representative NHANES. J Clin Endocrinol Metab 2013;98:3001–9.
5. Luxwolda MF, Kuipers RS, Kema IP, Janneke Dijck-Brouwer DA, Muskiet FA. Traditionally living populations in East Africa have a mean serum 25-hydroxyvitamin D concentration of 115 nmol/l. Br J Nutr 2012;108(9):1557–61.

doi: 10.3945/ajcn.114.090746.

Testing Satter's Division of Responsibility in Feeding in the context of restrictive snack-management practices

Dear Sir:

In a recent article, Rollins et al (1) stated that they set out to test their interpretation of Satter's Division of Responsibility in Feeding (sDOR; according to the authors, "parent provides, child decides") relative to snack management and correctly cite Satter (2) as advocating shared control in feeding between parent and child. This research showed that when feeding is based on restriction and avoidance, there is no good way to manage children's snacks in general and high-sugar, high-fat snack foods in particular. Moreover, it showed that sDOR cannot be successfully applied in the context of food restriction and avoidance. On the other hand, the study showed that children have lower BMIs and a lower tendency to eat in the absence of hunger (EAH; a protocol for observing a child's likelihood of eating high-calorie snacks soon after a meal) when mothers avoid intruding on children's prerogatives of how much they eat.

Rollins et al (1) examined the "parent provides" (what and when but not where) part of the equation by testing mothers on their use of restrictive feeding practices identified in previous research relative to 7 snack foods: popcorn, pretzels, chips, chocolate chip cookies, chocolate, fruit-flavored chewy candies (eg, Skittles; Wrigley), and ice cream. Feeding practices were as follows: limit buying, limit when, limit how much, limit second helpings, and (purchase but put food) out of reach. Four patterns of controlling feeding practices emerged:

1) Unlimited access: girls were allowed to choose their own snacks and eat them when they wanted to in self-determined amounts.
2) Sets (time) limits + does not restrict snacks (controlled when and how much and did not keep snack foods out of reach).
3) Sets (time) limits; restricts high-fat, high-sugar snacks (controlled when and how much and kept 50% of snack foods out of reach).
4) Sets (time) limits; restricts all snacks (controlled when and how much and kept all snack foods out of reach).

Based on their relatively moderate scores in BMI and EAH, the group 1 "unlimited access" girls were the most successful. Based on increasing levels of BMI and/or EAH, girls in the other 3 groups were less successful. Group 2 girls were lowest in BMI but relatively high in EAH, indicating that the girls are likely to be at risk for excessive weight gain as they get older and are able to gain access to food on their own. Group 3 girls had the highest BMIs and lowest EAH, indicating that the girls may have become acclimated by gaining access to these foods on their own. Group 4 girls had the second-highest BMIs and a strong tendency to EAH. Measurements of approach and inhibitory control showed little variation among the four groups.

Correctly following sDOR requires parents to take leadership with feeding by being reliable about providing regularly scheduled meals and snacks, taking responsibility for food selection through purchasing and meal and snack planning, and exercising their parental authority in not allowing children to have food handouts between times. Within the context of their leadership with feeding, parents give children autonomy with eating by letting them eat as much or as little as they want at those regularly scheduled eating times (3). sDOR only "works" when all of the components are in place: parents manage the what, when, and where of feeding and allow children to determine the how much and whether of eating (2). Moreover, managing structure within the context of sDOR is providing, not restricting or depriving, and the intent is to support children in eating as much as they need, not limiting their food intake (4). Within the context of sDOR, parents do not attempt to control how much the child eats in any way, not by portion control, not by running out of food, not by exhorting the child to use self-restraint, not by giving the child the "look."

As indicated in **Table 1**, none of the study patterns replicated sDOR. Instead, the patterns represented a deconstruction of sDOR by including some but not all of the components. Group 1 mothers, who had the most successful daughters, did not take leadership with feeding but did give autonomy with eating. In the other 3 groups,

TABLE 1
Patterns of restrictive feeding practices in relation to Satter's Division of Responsibility in Feeding[1]

	Parent takes leadership with what, when, and where of feeding			Parent gives child autonomy with how much and whether of eating	
	What	When	Where	How much	Whether
Group 1: unlimited access to snacks	~	0	0	+	+
Group 2: sets (time) limits and does not restrict snacks	~	+	0	+	+
Group 3: sets (time) limits and restricts high-fat, high-sugar snacks	+	+	0	0	+
Group 4: sets (time) limits and restricts all snacks	+	+	0	0	+

[1] 0, not present; +, present; ~, unclear.