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The mean dietary protein intake at different stages of chronic kidney disease is higher than current guidelines

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The actual dietary protein intake of adults without and with different stages of chronic kidney disease is not known. To evaluate this we performed cross-sectional analyses of 16,872 adults (20 years of age and older) participating in the National Health and Nutrition Examination Survey 2001–2008 who completed a dietary interview by stage of kidney disease. Dietary protein intake was assessed from 24-h recall systematically collected using the Automated Multiple Pass Method. Complex survey analyses were used to derive population estimates of dietary protein intake at each stage of chronic kidney disease. Using dietary protein intake of adults without chronic kidney disease as the comparator, and after adjusting for age, the mean dietary protein intake was 1.30 g/kg ideal body weight/day (g/kgIBW/d) and was not different from stage 1 or stage 2 (1.28 and 1.25 g/kgIBW/d, respectively), but was significantly different in stage 3 and stage 4 (1.22 and 1.13 g/kgIBW/d, respectively). These mean values appear to be above the Institute of Medicine requirements for healthy adults and the NKF-KDOQI guidelines for stages 3 and 4 chronic kidney disease. Thus, the mean dietary protein intake is higher than current guidelines, even after adjusting for age.

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Dietary manipulation has been used for over a century in the treatment of kidney diseases.[1–8] This strategy is based on experimental reports that an excess of dietary protein can impair kidney function and aggravate the complications of uremia, whereas an excess of other dietary components can interfere with the protective effects of inhibitors of the renin–angiotensin–aldosterone system on the progression of chronic kidney disease (CKD).[1,9–14] There is also evidence that the application of low-protein diets in patients with advanced CKD can slow the loss of residual kidney function while correcting certain symptoms of CKD by reducing the accumulation of unexcreted waste products.[7,15–18] There is concern, however, that low-protein diets might lead to the loss of protein stores in patients with advanced CKD, in part because patients with CKD might spontaneously reduce the content of protein in their diets.[19–22] In fact, the concern prompted some to recommend an 'early' initiation of dialysis to prevent the loss of protein stores and the development of other complications.[23] Extensive investigation revealed that early initiation of dialysis did not improve the outcome of patients with CKD.[24–27] Still, if there were more information about the level of dietary protein intake (DPI) of patients with different stages of CKD, strategies could be developed to correct either an inadequate or a surfeit of DPI or abnormalities in other constituents of the diet of patients with CKD.

Assessments of dietary constituents are largely confined to population studies directed at components of the diet that could affect specific illnesses (e.g., cholesterol or different types of lipids). Information on major components of the diet is available from population surveys, such as the United States National Health and Nutrition Examination Survey (NHANES[28]). The NHANES includes health-related interviews, socioeconomic information, and a medical

examination with a battery of laboratory assessments including serum creatinine and a spot urinary creatinine with albumin. The serum creatinine has been used in estimation equations of glomerular filtration rate (GFR) to produce population estimates of CKD by applying the formula and albuminuria data to National Kidney Foundation staging for CKD.[29–31] The NHANES includes dietary intake data using a systematic approach developed by the United States Department of Agriculture for the What We Eat in America program.[32] By combining these elements (the eGFR, the National Kidney Foundation (NKF) staging for CKD, and the NHANES), a means of comparing dietary intake for non-institutionalized adults with and without evidence of CKD is available. Our purpose was to evaluate whether associations exist between the DPI of individuals with CKD and those without CKD to determine whether the stage of CKD is associated with differences in dietary protein.

RESULTS

Participants

Of the 21,456 adults $\geqslant 20$ years of age in the NHANES 2001–2008 cycles, 1,254 did not have the mobile examination visit that included the physical examination and laboratory testing. These participants were excluded from the analysis. Similarly, the 850 adults who were pregnant at the time of the examination and the 1,256 who did not have a serum creatinine measurement were excluded. Other exclusions were 506 participants who did not have multiple measures of proteinuria, as suggested by Coresh,[30] 44 participants who had received dialysis during the previous 12 months, and 718 participants who did not complete the dietary interview. Menstruating females ($n = 290$) were included in this analysis.[33] The final sample consisted of 16,828 participants.

Demographic and clinical characteristics

For the period of 2001–2008, results of the NHANES analysis indicate that 85.1% of the US adult population (171 million) had no evidence of CKD. However, 2.3% (4.7 million) had stage 1 CKD, 3.8% (7.6 million) had stage 2 CKD, 8.3% (16.6 million) had stage 3 CKD, and 0.4% (800,000) had stage 4 CKD or more severe CKD (i.e., stage 4 + CKD) but had not begun dialysis treatment. Characteristics of the adults participating in the NHANES varied (Table 1): the mean ± s.e. (s.d.) age was 46.5 ± 0.3 (16.5) years; the body mass index was 28.3 ± 0.1 (6.4) kg/m^2; 49.2% were male; 72.7% were non-Hispanic (NH) white, 10.3% were NH black, and 12% were Mexican American or Latino. Fewer than 2% of these participants reported having been told they had 'weak or failing kidneys' as designated in the NHANES interview questionnaire.

DPI and participant characteristics

The estimated DPI differed between men and women, $F(1,61) = 64.32$, $P < 0.0001$, $r^2 = 0.007$. The DPI of women was -0.11 g/kgIBW/d below the value in men (Figure 1a). DPI also differed according to race/ethnicities, $F(3,61) = 17.2$, $P < 0.0001$, $r^2 = 0.006$. Specifically, NH blacks consumed 0.06 g/kgIBW/d less protein than NH whites, whereas the DPI of participants with Mexican American or Latino origin was 0.13 g/kgIBW/d more than NH whites (Figure 1b). Age affected DPI in an inverse manner; adults in the youngest age groups reported the highest DPI level, whereas the older participants reported lower values of DPI (Figure 1c). Those in the oldest age group (75 + years) had a DPI 0.32 g/kgIBW/d below the value of the 20–34-year-olds (reference group). In addition, participants in the 45–54-year-old group had a DPI of 0.08 g/kgIBW/d below that arising from the reference group. Overall, the few participants who knew that they had kidney disease had a significantly lower DPI (-0.14 g/kgIBW/d) compared with those with CKD who did not know of their kidney disease, $F(1,61) = 7.37$, $P < 0.009$, $r^2 = 0.0009$. The mean percentage of high biological value (HBV) DPI was 58.4% (1.1%).

DPI according to stage of CKD

We evaluated DPI according to the stage of CKD and found that there was a significantly lower value of DPI in those with subnormal eGFR compared with participants without evidence of CKD (Table 2). Additional analysis demonstrated that either the total grams of DPI or the DPI normalized for actual body weight were significantly higher in those without evidence of CKD compared with the different stages of CKD: $F(4,61) = 33.97$, $P < 0.0001$, $r^2 = 0.016$; $F(4,61) = 34.87$, $P < 0.0001$, $r^2 = 0.014$; and $F(4,61) = 22.35$, $P < 0.0001$, $r^2 = 0.009$ for total grams of DPI, the DPI normalized to actual body weight, and DPI normalized to ideal body weight, respectively. When each stage of CKD was compared with NoCKD, DPI normalized to ideal body weight demonstrated that the intake of participants with stage 1 CKD was not different from those without CKD. At stages beyond stage 1, the DPI was significantly lower ($P < 0.0001$). In comparison with those without CKD, the average DPI was lower across different stages of CKD by 9–10 g/d (0.02 to 0.13 g/kgIBW/d). There was a significant difference in the mean percentage of HBV DPI across the stages of kidney disease, with individuals in stages 3 and 4 CKD consuming a statistically higher percentage of HBV DPI than those in the NoCKD group.

Influence of participant characteristics on DPI according to the stage of CKD

When sex was combined with CKD (Figure 2a), the DPI continued to show a difference ($F(9,61) = 18.53$, $P < 0.0001$, $r^2 = 0.015$). However, the difference was not related to sex, $F(1,61) = 1.30$, $P = 0.26$. The main effect of CKD stage remained significantly related to DPI ($F(4,61) = 20.82$, $P < 0.0001$). In addition, there was no interaction between the stages of CKD and sex, $F(4,61) = 0.82$, $P = 0.52$, indicating that eating less protein in the advanced stages of CKD was independent of whether participants were men or women.