Participants in the low protein group should have stored 5.6 kg of body fat instead of the 3.7 kg of body fat observed when overfed by 3.9 MJ/d for 56 days, while energy expenditure should not have changed. An explanation is an underestimation of energy requirement for weight maintenance in the low protein group, resulting in an overestimation of the overfeeding in this group. The explanation that higher fat intake in the low protein group reduced nutrient absorption and thus brought intake and expenditure closer together would require a fecal fat loss of more than 30 g/d. If so, one would not expect participants to get fat when the fat content of the diet is increased.[3]

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Conflict of Interest Disclosures: The author has completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

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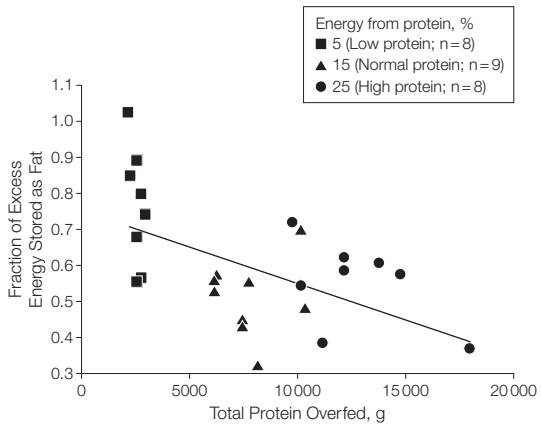
In Reply: Dr Westerterp calculates that the individuals in our study eating the low protein diet should have gained more body fat than we reported. He thinks that differences in fecal fat loss between diets is not an adequate explanation, and we agree. He goes on to offer "an underestimation of energy requirement for weight maintenance" as a potential explanation.

To evaluate this suggestion, we first reexamined the differences between our weight-stabilized estimate of energy requirements and baseline energy expenditure, and found that they did not differ significantly between diet groups (low protein diet: –83 kcal/d [95% CI, –411 to 243 kcal/d]; normal protein diet: 177 kcal/d [95% CI, –146 to 503 kcal/d]; and high protein diet: 273 kcal/d [95% CI, –165 to 711 kcal/d]; $P=.28$), but cumulatively the low protein diet group ingested less total energy than the normal or high protein diet groups (low protein diet: 46 190 kcal [95% CI, 30 742 to 51 364 kcal]; normal protein diet: 54 819 kcal [95% CI, 47 527 to 62 111 kcal]; high protein diet: 50 666 kcal [95% CI, 43 200 to 58 131 kcal]; $P=.13$).

As noted in our study, there was no difference in the amount of body fat gained by the 3 different protein diet groups during overfeeding, although the low protein diet group stored 200 g (about 2000 kcal) more. To take these small, but not statistically significant, cumulative differences into account, we evaluated fat storage as a fraction of the total energy that was overfed. The FIGURE shows that when the protein intake was low, a higher percentage of the overfed energy (calories) was stored in fat. As the dietary protein intake increased, the fraction of the excess energy that was stored as fat declined. The low protein group stored on average 75% of excess energy as fat, the normal and high protein groups about 50%. This relationship is expressed in the following equation: fat stored/overfed energy = 0.75 – 0.0000202 × (grams of protein overfed) ($P<.005$). This analysis suggests that dietary protein affects nutrient partitioning of calories.

Figure. Energy Stored in Fat as a Fraction of Total Excess Calories



The diagonal line was created by a linear regression model.

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Conflict of Interest Disclosures: The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Dr Bray reported that he has been a consultant to Abbott Laboratories and Takeda Global Research Institute; is an advisor to Medifast, Herbalife, and Global Direction in Medicine; has received royalties for the *Handbook of Obesity;* has received grants and support for travel from the US Department of Agriculture; and has received grants from the National Institutes of Health, Diabetes Prevention Program Outcomes Study, and LookAHEAD. Dr Redman and Smith reported no disclosures.

RESEARCH LETTER

2008 US Preventive Services Task Force Recommendations and Prostate Cancer Screening Rates

To the Editor: The US Preventive Services Task Force (USPSTF) recently drafted a grade D recommendation against prostate-specific antigen (PSA)–based screening for prostate cancer.[1] If this recommendation becomes final, how it will affect clinical practice remains unclear. In 2008, the USPSTF issued a grade D recommendation against PSA-based screening in men aged 75 years or older.[2] We evaluated changes in national screening rates before and after this recommendation.

Methods. According to federal regulations, the study was exempt from review by an institutional review board;

patient data were deidentified and requirement for consent to our study was waived. Demographic, socioeconomic, and functional variables were collected from the 2005 and 2010 National Health Interview Survey (NHIS), which is a cross-sectional, in-person household survey continuously performed throughout each year on approximately 87 500 individuals (90% response rate).[3] Multistage area probability sampling provides a representative sample of the US population. The Cancer Control Supplement, which contains questions regarding cancer screening, is included every 5 years.

For this study men aged 40 years or older without prostate cancer or other prostate-related conditions (eg, benign prostatic hyperplasia or prostatitis) who visited a physician in the prior year were included. The NHIS contains 13 questions on timing and reason for PSA testing. Men were asked "What was the main reason you had this PSA test—was it part of a routine exam, because of a problem, or some other reason?" Men who answered "because of a problem" or "other reason" were excluded from the analyses. Prostate-specific antigen screening was defined as a PSA test during a routine examination within the past year. The screening rate was estimated using sampling weights. The difference in proportion of PSA screening between 2005 and 2010 was compared using logistic regression, corrected for survey design. We conducted overall analysis and stratified by age groups. A 2-sided *P* value of less than .05 was considered statistically significant and the analysis was performed with Stata version 11.0 (StataCorp).

Results. The final cohort had 5332 men from 2005 and 4640 men from 2010 (TABLE). The PSA screening rates were unchanged in all age groups over time (FIGURE). In men aged 75 years or older, PSA screening was unchanged between 2005 (43.0%; 95% CI, 38.9%-47.0%) and 2010 (43.9%; 95% CI, 39.1%-48.7%) (*P*=.77). In 2010, PSA screening was more common in men aged 75 years or older than in men aged 40 to 49 years (12.5%; 95% CI, 10.2%-14.7%) and 50 to 59 years (33.2%; 95% CI, 30.1%-36.3%) (*P*<.001 between groups) but not in men aged 60 to 74 years (51.2%; 95% CI, 48.1%-54.2%).

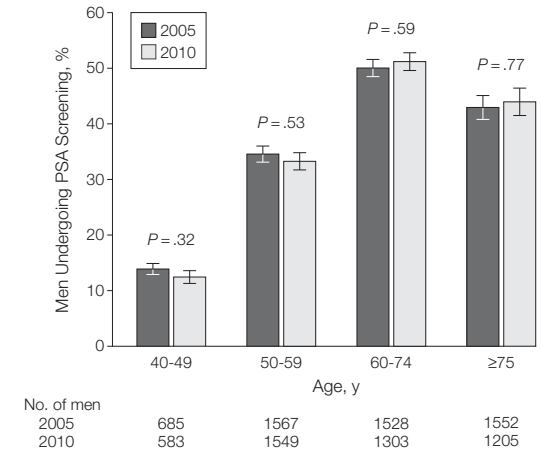
Comment. Large population-based studies have demonstrated PSA screening in men aged 75 years or older is inappropriately high given the limited likelihood of benefit.[4,5] Despite the USPSTF recommendation against prostate cancer screening in men aged 75 years or older in 2008, PSA screening rates did not change. Our findings must be interpreted within the context of the study design. Data from the NHIS are deidentified and self-reported responses cannot be verified. Because self-reported PSA screening rates in the NHIS are predominantly lower compared with medical record extraction,[6] our data are likely an underestimate. Sampling of the same individual in both years, while possible, is a rare occurrence given the sample design of the NHIS. While it is possible some men received a PSA test for reasons other than prostate cancer screening, it is likely this number is small because the NHIS questionnaire explains the test is used to detect prostate cancer and we included only men who reported a PSA as part of a routine examination. The discrepancy between the USPSTF recommendation and subsequent practice patterns may reflect lack of

Table. Demographic Characteristics of 2005 and 2010 National Health Interview Survey (NHIS) Study Cohort

	NHIS Cohort, No. (Weighted %)[a]	
	2005 (n = 5332)	2010 (n = 4640)
Race/ethnicity[b]		
Non-Hispanic white	3980 (79.98)	3074 (77.14)
Non-Hispanic black	576 (8.45)	672 (8.70)
Hispanic	582 (7.67)	580 (9.30)
Asian	119 (2.69)	252 (3.88)
Other	68 (1.21)	61 (0.97)
Education		
<High school	876 (14.08)	757 (13.55)
High school	1508 (29.18)	1230 (26.29)
Some college	1319 (25.33)	1226 (26.40)
College graduate	1594 (31.41)	1408 (33.76)
Relationship status		
Not married or living with partner	1795 (20.92)	1716 (23.95)
Married or living with partner	3527 (79.08)	2919 (76.05)
Family history		
None	2511 (48.54)	2124 (46.85)
Cancer	2196 (43.88)	1880 (45.44)
Prostate cancer	353 (7.58)	329 (7.70)
Smoking		
Never	2272 (43.81)	2088 (45.71)
Former	1968 (36.48)	1707 (37.35)
Current	1084 (19.71)	842 (16.94)
Colorectal screening		
Has never had a colonoscopy	2974 (73.67)	2251 (63.72)
Colonoscopy >10 y ago	120 (2.94)	129 (3.57)
Colonoscopy ≤ 10 y	990 (23.39)	1137 (32.71)
Body mass index[c]		
Underweight or normal (<25)	1403 (25.00)	1100 (22.05)
Overweight (25-29.9)	2397 (45.43)	2018 (44.48)
Obese (≥30)	1490 (29.58)	1505 (33.46)
Personal cancer history		
No	5032 (94.54)	4343 (93.60)
Yes	297 (5.46)	295 (6.40)
9-y Mortality risk[d]		
Low (healthiest)	252 (16.28)	258 (19.50)
Intermediate	820 (51.51)	717 (48.46)
High (sickest)	541 (32.21)	500 (32.04)
Personal health status		
Excellent or very good	2660 (52.50)	2284 (53.15)
Good	1598 (29.37)	1386 (28.86)
Fair or poor	1069 (18.13)	967 (17.99)

[a] The totals within each category vary minimally due to missing variables.
[b] The categories on the questionnaire were provided by the NHIS but selection of the category was performed by the survey participant.
[c] Calculated as weight in kilograms divided by height in meters squared.
[d] Data were available for men aged 65 years or older and were derived from the study by Schonberg MA, Davis RB, McCarthy EP, Marcantonio ER. External validation of an index to predict up to 9-y mortality of community-dwelling adults aged 65 and older. *J Am Geriatr Soc*. 2011;59(8):1444-1451.

Figure. Rates of Prostate-Specific Antigen (PSA) Screening by Year and Age Group Between 2005 and 2010



Error bars indicate standard deviations.

guideline awareness, financial incentives, or patient or physician confidence in PSA screening. Clinical practice patterns following the 2011 USPSTF recommendations should be monitored.

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Author Contributions: Dr Eggener had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.
Study concept and design: Prasad, Drazer, Huo, Eggener.
Acquisition of data: Prasad, Drazer, Huo.
Analysis and interpretation of data: Prasad, Drazer, Huo, Hu, Eggener.
Drafting of the manuscript: Prasad.
Critical revision of the manuscript for important intellectual content: Prasad, Drazer, Huo, Hu, Eggener.
Statistical analysis: Prasad, Drazer, Huo.
Obtained funding: Eggener.
Administrative, technical, or material support: Hu, Eggener.
Study supervision: Eggener.
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CORRECTIONS

Unreported Financial Disclosures: In the Review entitled "Association of LDL Cholesterol, Non–HDL Cholesterol, and Apolipoprotein B Levels With Risk of Cardiovascular Events Among Patients Treated With Statins: A Meta-analysis," published in the March 28, 2012, issue of *JAMA* (2012;307[12]:1302-1309), the following disclosures should have been reported: "Dr Mora reports receipt of travel accommodations/meeting expenses from Pfizer; Dr Durrington reports provision of consulting services to Hoffman-La Roche, delivering lectures or serving on the speakers bureau for Pfizer, and receipt of royalties from Hodder Arnold Health Press; Dr Hitman reports receipt of lecture fees and travel expenses from Pfizer, provision of consulting services on advisory panels to GlaxoSmithKline, Merck Sharp & Dohme, Eli Lilly, and Novo Nordisk, receipt of a grant from Eli Lilly, and delivering lectures or serving on the speakers bureau for GlaxoSmithKline, Takeda, and Merck Sharp & Dohme; Dr Welch reports receipt of a grant, consulting fees, travel support, payment for writing or manuscript review, and provision of writing assistance, medicines, equipment, or administrative support from Pfizer, and provision of consultancy services to Edwards, MAP, and NuPathe; Dr Demicco reports having stock/stock options with Pfizer; Dr Clearfield reports provision of consulting services on advisory committees to Merck Sharp & Dohme and AstraZeneca; Dr Tonkin reports provision of consulting services to Pfizer, delivering lectures or serving on the speakers bureau for Novartis and Roche, and having stock/stock options with CSL and Sonic Health Care; and Dr Ridker reports board membership with Merck Sharp & Dohme and receipt of a grant or pending grant to his institution from Amgen. Drs Simes, Zwinderman, and Downs reported no conflicts of interest." This article was corrected online.

Errors in Table. In the Original Contribution entitled "Treadmill Exercise and Resistance Training in Patients With Peripheral Arterial Disease With and Without Intermittent Claudication: A Randomized Trial," published in the January 14, 2009, issue of *JAMA* (2009;301[2]:165-174), data in the "Intermittent claudication" row of Table 1 were incorrect. The number (%) for the treadmill walking exercise group should have read "18 (35.3)"; for the lower extremity resistance training group, "18 (34.6)"; for the control group, "20 (37.7)"; and for the *P* value, ".94."

Error in Financial Disclosure. In the Original Contribution entitled "Cognitive Behavior Therapy Augmentation of Pharmacotherapy in Pediatric Obsessive-Compulsive Disorder: The Pediatric OCD Treatment Study II (POTS II) Randomized Controlled Trial," published in the September 21, 2011, issue of *JAMA* (2011; 306[11]:1224-1232), the financial disclosure for Dr March was incorrect. It should have read: "Dr March reported that he is a scientific advisor for Pfizer, Lilly, Bristol-Myers-Squib, and Shire Pharmaceuticals; receives research support from Pfizer, is a member of the data safety and monitoring boards for Otsuka and Lilly, is an equity holder in MedAvante; and receives royalties from MultiSystems for the Multidimensional Anxiety Scale for Children and for books from Guilford and from Oxford University Press."