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Conflict of Interest Disclosures: None reported.

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In Reply Melis and colleagues comment on our finding that patient preference for involvement in decision making among hospitalized patients is associated with increased hospital length of stay and costs and note that this contradicts the expectation that greater patient engagement will reduce resource use.[1] They say that preference for involvement may not be associated with actual levels of involvement. However, while preferred and actual levels of involvement may diverge, patient preference for greater involvement still tends to be positively associated with the actual level of involvement.[2,3]

Melis et al also suggest that the association of greater preference for involvement with increased resource use that we observed might result from an inverse relationship between patients' preferred and actual levels of involvement. They argue that this could arise if dissatisfaction due to low prior involvement in decision making increases preference for involvement. We think this is possible but know of no evidence that dissatisfaction with past involvement increases preference for future involvement. We also note that if the observed relationship between preference for involvement and resource utilization results from low past involvement, past and present involvement must be positively correlated (ie, low past involvement led to a preference for more current involvement and low past involvement is associated with low current involvement that leads to increased resource utilization). However, if such persistent low involvement is the result of the hospital or clinician providing care, we question whether a high correlation between low past involvement and low current involvement is likely because the current admission for the patient was the only admission to our hospital for 78% of our patients and because patients with multiple admissions rarely saw the same clinicians. One could imagine that low past involvement and low current involvement might be positively correlated if low levels of involvement for certain patient groups persist across clinicians over time. However, we think that such patterns are unlikely to explain our results because we controlled for a wide range of patient demographic and socioeconomic variables. On the other hand, it seems possible that some patients might have persistent attributes we did not observe (eg, cognitive function, personality) that might affect levels of involvement in both past and present encounters. For this reason, we think that future work might reasonably investigate the cross-sectional and longitudinal relationship of patients' preferred and actual involvement in decision making and collect data on factors such as cognitive function or personality that might affect actual levels of involvement.

Melis et al also suggest that our findings derived from an inpatient setting might not be generalizable to outpatients. We agree and make the point in our article that in outpatient settings where clinicians may have incentives to increase utilization, it is likely that greater patient engagement would decrease resource utilization.

We believe that future research examining the effect of patient participation in decisions under varying physician incentives for resource utilization would be valuable in better understanding the effects of patient engagement in decision making on resource utilization.

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The Importance of Influenza Vaccination

To the Editor We represent the Infectious Diseases Society of America (IDSA) and the Society for Healthcare Epidemiology of America (SHEA) and read with concern the commentary on influenza vaccines by Doshi.[1] He contends that influenza is not a significant illness in frequency or severity and is easily prevented without vaccination. Data refute this claim, as in the last year alone, there were more than 12 000 hospitalizations and 153 pediatric deaths due to laboratory-confirmed influenza in the United States, with 90% of the pediatric deaths occurring in unvaccinated children.[2] Vaccination is associated with decreases in influenza infections and their associated health care burden with an estimated 112 900 hospitalizations, 5.8 million medical visits, and 13.6 million illnesses averted from 2005 through 2011 in the United States.[3]

Doshi[1] also argues that the influenza vaccine is not effective. Although overall protective efficacy of 55% to 70% is not ideal,[2,4] it is far better than the 0% efficacy of being unvaccinated. We agree that currently licensed vaccines are more effective in younger, healthier people than in the elderly or immunocompromised patients and that improved vaccines for these populations are desirable. It is for these reasons that targeting healthier populations is important in order to protect those vulnerable patients who are less likely to respond to the

current vaccines. In addition, his call for head-to-head comparisons of vaccination with other prevention strategies ignores the fact that efforts to prevent the spread of respiratory tract infections must be multifaceted, including the prevention of presenteeism, promoting the use of hand hygiene and respiratory etiquette, and influenza vaccination.

While Doshi[1] argues that there are insufficient data to assess the safety of influenza vaccines, we disagree. Vaccine safety is monitored closely in the United States with the Vaccine Adverse Event Reporting System and the Vaccine Safety DataLink program. Furthermore, required postmarket surveillance identified the 2 examples cited by Doshi[1] (both of which occurred outside of the United States and with formulations not in use in this country). Most studies have not found a significantly increased risk of Guillain-Barre syndrome after influenza vaccination since the 1976 campaign. Indeed, there may be higher rates of Guillain-Barre syndrome after an influenza-like illness than after influenza vaccination.[5]

Influenza is a serious individual and population health threat. Available vaccines are safe, effective, and should remain a key part of any influenza prevention program; however, more effective vaccines and additional anti-influenza prevention and treatment strategies are needed.

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In Reply My article aimed to show that many basic assumptions and claims underlying the annual campaign to vaccinate against influenza do not stand up to critical review.[1] I am grateful that the Infectious Diseases Society of America (IDSA) and the Society for Healthcare Epidemiology of America (SHEA) responded to my article, but disappointed that they did not examine the data underlying their positions, which are to my knowledge consistent with those of the Centers for Disease Control and Prevention (CDC)—and equally problematic.

The IDSA and SHEA make 4 major criticisms. First, they emphasize that influenza is a significant illness, causing more than 12 000 hospitalizations last year. I do not think 12 000 hospitalizations (approximately 0.03% of the 35 million annual US hospitalizations[2]) by itself qualifies influenza as a major public health threat. Influenza can indeed lead to severe complications including death, but my article attempted to counter the popular impression that we are all at risk of serious complications of influenza by pointing out that the majority of people do not annually contract influenza, and for most of those who do, it is self-limiting and tragedies are rare.

Second is the question of vaccine performance. I argued that there is no good evidence that vaccines can prevent hospitalizations and death. There are no adequately powered randomized trials of influenza vaccines in the elderly to detect any possible effect on these outcomes, and serious methodological problems have been documented in the available nonrandomized studies that report impressive benefit. But IDSA and SHEA assert that vaccinations averted 112 900 US hospitalizations during 2005 through 2011, citing a recent CDC publication.[3] This article, however, did not present any new evidence of vaccine effectiveness against hospitalization. Rather, it estimated averted hospitalizations by taking previously published estimates of vaccine efficacy against influenza cases and extrapolating this effect to influenza hospitalizations. But there is no good reason to believe a 20% reduction in cases would neatly translate into a 20% reduction in hospitalizations. Individuals with influenza vary in their risk of complications. For example, averting cases among a more healthy elderly population may do little to reduce the number of hospitalizations that occur more frequently among less healthy elderly individuals.

Third, IDSA and SHEA state that "vaccine safety is monitored closely in the United States" and the cases of narcolepsy in Sweden and Finland and febrile convulsions in Australia were identified by postmarketing surveillance. I disagree. It is at odds with the Ministerial review in Australia, which concluded the following: "This Review has revealed an adverse event reporting system that is not robust or timely and which does not conform to WHO [World Health Organization] recommendations…."[4(p6)] More importantly, even had surveillance identified these harms, it does not change the fact that serious and unanticipated harms from influenza vaccines have occurred.

Finally, of course vaccines and hand washing can be complementary. But without head-to-head trials, IDSA and SHEA's claim that vaccines are "best"[5] (ie, better than other interventions) lacks evidence.

Physicians and the public expect officials and professional societies to critically assess the evidence on which they are basing their statements and recommendations. We are ill served by authorities that do otherwise.

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Conflict of Interest Disclosures: Dr Doshi is a corecipient of a UK National Institute for Health Research grant to carry out a Cochrane review of neuraminidase inhibitors (http://www.hta.ac.uk/2352). In addition, Dr Doshi received €1500 from the European Respiratory Society in support of his travel to the society's September 2012 annual congress, where he gave an invited talk on oseltamivir.

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Red Meat and Type 2 Diabetes Mellitus

To the Editor The recent study by Pan et al[1(p1334)] contains the disclaimer "Since our study is observational in nature, causality cannot be inferred." The authors nonetheless infer that their "results add further evidence that limiting red meat consumption over time confers benefits for T2DM [type 2 diabetes mellitus] prevention."[1(p1328)]

Observational studies, however, can imply causality. Bradford Hill[2] provided guidance on when associations might support causation. Hill was clear that his 9 rules derive from common sense. I think that Pan et al[1] do not conform to most of these rules and their conclusion seems unwarranted.

The most import factor is that an association should be strong. In fact, Pan et al[1] showed very weak associations. They reported that "Compared with the reference group of no change in red meat intake, increasing red meat intake of more than 0.50 servings per day was associated with a 48% elevated risk."[1(p1328)] Many authors have pointed out the danger in confusing statistical significance with clinical or practical utility,[3] and ironically, the Harvard Health Blog (http://www.hvrd.me/10hefBI), in considering an earlier paper from this group, described how misleading relative risk can be. In fact, a simple calculation showed the incidence of diabetes in the highest group (Table 3 in the article by Pan et al[1]) at only 7.04%, while the "reference group" had an incidence of 4.26%. The absolute difference in risk is 2.8%. Given the known errors in food questionnaires, this cannot be considered a meaningful number. "No change" as a reference may also be misleading. The authors measured the effect of reducing meat consumption, which increased the frequency of diabetes in all the cohorts studied, opposite to the expectation of a consistent dose-response curve. Perhaps most telling is Hill's principle of coherence: "data should not seriously conflict with the generally known facts of the natural history and biology of the disease."[2(p10)] In fact, red meat consumption decreased[4] as T2DM increased during the past 30 years.[5]

The poor association and lack of biological plausibility suggest that the "robust" message from Pan et al[1] is that there is no reason to associate red meat with T2DM.

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In Reply We appreciate Dr Feinman's comments on our recent publication of changes in red meat consumption and subsequent risk of type 2 diabetes mellitus (T2DM).[1] Dr Feinman claimed that "Pan et al[1] do not conform to most of these rules and their conclusion seems unwarranted" for causal inference based on the criteria of Hill.[2] We would like to take this opportunity to clarify several assertions made by Dr Feinman.

Dr Feinman argued that the association was not strong enough to be considered as causal. However, in our study, the multivariate-adjusted hazard ratio comparing the "moderate to large increase" group with the "relative stable" group was 1.48 (95% CI, 1.37-1.59), which was comparable to the associations for other important risk factors for T2DM. For example, the relative risk for T2DM was 1.44 (95% CI, 1.31-1.58) comparing active smokers with nonsmokers from a meta-analysis of 25 prospective studies[3]; and it was 1.43 (95% CI, 1.20-1.72) comparing physical inactivity with moderate activity from a meta-analysis of 10 prospective studies.[4]

Dr Feinman calculated the "incidence" of diabetes in the highest (moderate to large increase) and reference groups as 7.04% and 4.26%, respectively, in Table 3 of our article.[1] It is unclear how this was calculated. In a cohort study, a better measure of disease occurrence is the incidence density. For example, the incidence density of T2DM was 4.39 per 1000 person-years for the reference group and 6.16 per 1000 person-years for the "moderate to large increase" group in the Nurses' Health Study (Table 3[1]). If we follow up 1 million people in each group for 1 year, this would translate to 1770 more new diabetes cases in the group with increased red meat consumption compared with the reference group. Considering high red meat consumption in United States and globally, the population impact is substantial. Dr Feinman mentioned that "Given the known errors in food questionnaires, this cannot