

way of selection). Clearly, this is a larger issue, and the solution probably involves teaching these skills better throughout our medical education system from medical school to residency and into fellowships and continuing education programs. Further innovation and investigation of methods that span the entire medical educational continuum are sorely needed.

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In reply

Kaushik and Pothier raise 2 important issues. First, how should researchers assess communication skills training? We think that assessment should occur at 2 levels: (A) Does the training change physician communication behaviors? and (B) Do the changes improve patient-level outcomes? Our study was designed to address question A. Our study design using standardized patients to answer this question has strengths and weaknesses. A weakness of using standardized patients is that physicians in real life may behave differently than in a simulated situation. Clearly, an important next step would be to examine trained physicians in their own clinics, as was done by Fallowfield et al,¹ who showed that physicians given a similar type of training (small groups with trained facilitators) in fact used their new skills in real settings at statistically significant levels. An important strength of using standardized patients is that, in our study, they were scripted to provide cues so that the physicians had consistent opportunities to demonstrate their skills. Thus, acquisition of new behaviors can be measured more precisely, and we think this approach provides an economical way to rapidly test new training models.

The second issue is what level of evidence should be required for an educational intervention to be put into practice. We agree that evidence of an improved patient-level outcome for Oncotalk would be ideal, but we do not have it. Should educators wait for such patient-level evidence before using this model? We would advise them as follows. A Cochrane meta-analysis of communication skills training,² focusing on skills similar to those in our study but packaged differently, showed that 6 of 11 randomized studies demonstrated improved patient satisfaction—even though these measures have a pronounced ceiling effect. The Oncotalk model engages trainees at a developmental stage not included in these existing studies, and the outcomes indicate that the program results in behavior change in simulated situations. This is the state of the science for this particular niche, and this level of proof is substantially more robust than what happens educationally in the rest of a medical oncology fellowship. As Last points out, our baseline measurement of the communication behaviors displayed by these self-selected trainees raises a reasonable concern about current training and practice (such as, for example, oncologists who do not use the word “cancer” with their patients

with cancer). We hope our study will stimulate others to think about, design, and test innovative approaches to these important issues.

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Low Prevalence of Individuals With Optimal or Borderline Levels of Cardiovascular Risk Factors Extends to Rapidly Developing Countries

Hozawa et al¹ showed that only a small proportion of American adults aged 45 to 64 years had low cardiovascular risk, defined as optimal or borderline levels of 4 major modifiable cardiovascular risk factors. It is largely unknown whether this unfavorable epidemiological situation extends to developing countries, although there is some belief that the proportion of individuals at low risk could be larger in these countries.

We measured cardiovascular risk factors in 1255 adults aged 25 to 64 years, who were randomly selected from the population of Seychelles, a rapidly developing small island state located 1800 km east of Kenya. The majority of the population is of African descent. Methods and overall results have been described previously.² In this study, we defined “borderline” risk factors similar to Hozawa et al¹ (former smoking; systolic/diastolic blood pressure of 120-139/80-89 mm Hg and no treatment for hypertension; and total cholesterol level of 200-239 mg/dL [to convert to millimoles per liter, to multiply by 0.0259] and no treatment for dyslipidemia). “Elevated” risk factors refer to higher values (diabetes: fasting blood glucose level \geq 126 mg/dL [to convert to millimoles per liter, multiply by 0.0555] or history or treatment of diabetes).

The **Table** shows that as few as 15%/22% of men/women aged 45 to 64 years had optimal or borderline risk factors levels in Seychelles (compared with 17%/17% of male/female African Americans in the study by Hozawa et al¹). Approximately 70% of adults aged 45 to 64 years had 1 or 2 risk factors. One risk factor was found in 30%/43% of men/women in Seychelles (African Americans, 41%/38%¹) and 2 risk factors in 30%/27% of men/women (African Americans, 30%/31%¹). At age 25 to 44 years, 37%/65% of men/women had optimal or borderline levels of risk factors. Not shown in the table but a likely underlying condition for several risk factors, overweight (body mass index \geq 25 [calculated as weight in kilograms divided by height in meters squared]) was found

Table. Prevalence of Risk Factor Categories in the General Population of Seychelles, 2004

Risk Factor Category	Age, 25-44 y		Age, 45-64 y	
	Men, %	Women, %	Men, %	Women, %
All optimal with no risk factor	6.1	25.7	0.8	6.3
No. of borderline risk factors only				
1	21.0	30.0	6.5	8.9
2	7.8	8.9	7.3	6.7
3	1.9	0.5	0.7	0.3
Total	36.8	65.1	15.3	22.2
No. of elevated risk factors, with any borderline risk factor				
1	40.0	29.9	43.8	42.7
2	19.4	3.9	29.7	26.7
3	3.8	0.8	9.9	8.1
4	0.0	0.3	1.5	0.3
Total	63.2	34.9	84.9	77.8

in 58%/79% of men/women aged 45 to 64 years and 48%/61% of men/women aged 25 to 44 years, a strong increase compared with 1989.³

Our data show that the “crucial reality that low risk is rare among Americans”^{4(p538)} also extends to a population facing rapid health transition. While not necessarily representative of all developing countries, these findings in Seychelles have important implications for prevention strategies. Premature cardiovascular disease rarely occurs among low-risk individuals, as reemphasized by Stamler,⁴ and it follows that control of classic risk factors remains the cornerstone of any prevention strategy. Furthermore, evidence that most of the total cardiovascular burden arises from the large number of individuals with only moderately elevated risk factors,⁵ and the findings that only a very small proportion of adults have optimal or suboptimal levels of risk factors across different populations, further emphasize the critical importance of public health interventions targeting the entire population.

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Are We Ignoring the Elephant in the Room?

Cook et al¹ reported no association between migraine and coronary heart disease (CHD) after a mean of 12 years of follow-up in the Physicians' Health Study (PHS). This study included information through March 2000. Kurth et al² extended the follow-up period of the PHS participants to February 2005. They concluded that “migraine was associated with increased risk of major CVD [cardiovascular disease]”^{2(p795)} and that “[t]he associations between migraine and major CVD, ischemic stroke, and MI [myocardial infarction] were not significantly modified by smoking and hypertension status or by randomized aspirin assignment.”^{2(p797)} The authors “had no information about the use of migraine-specific drugs (ie, triptans and ergot alkaloids).”^{2(p800)} However, in a study by the same authors regarding the Women Health Study “women were asked on the 48-month questionnaire to provide information regarding medication use during the previous 2 weeks. The frequency of migraine-specific drug use among women who reported active migraine at baseline was 5.3%.”^{3(p290)} So what did these patients use to treat their migraine?

Nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly used to treat migraine. The Food and Drug Administration (FDA) approved celecoxib, rofecoxib, and valdecoxib in 1998, 1999, and 2001 respectively. Of the patient visits “in which either a COX-2 [cyclooxygenase-2] inhibitor or NSAID was prescribed, the frequency of COX-2 inhibitor use increased from 35% (1999) to 55% (2000) to 61% (2001 and 2002).”^{4(p171)} Rofecoxib and valdecoxib were taken off the market September 2004 and April 2005, respectively, secondary to associated increased risk of cardiovascular disease. Also, the FDA required all NSAIDs manufacturers to add a warning regarding cardiovascular risk. “Current and new users of all classes of nonaspirin NSAIDs had elevated relative risk estimates for MI.”^{5(p978)} Possibly, patients with migraine headache are more susceptible to the negative cardiovascular adverse effects of NSAIDs, especially selective COX-2 inhibitors, than other patients such as those with nonmigraine headache. Finally, the use of NSAIDs and COX-2 inhibitors could interfere with the cardioprotective effect of aspirin. Kurth et al² mentioned nothing about NSAIDs or COX-2 inhibitors in their article. Although the use of NSAIDs was one of the exclusion criteria to enter the PHS, their use should have been addressed during the trial and in the posttrial follow-up, especially in this subgroup of participants with migraine headache.

The authors should have provided any data regarding the use of these medications. If the data were not avail-