Keeping Our Eye on the Ball: The American Society of Preventive Oncology in 2009

James R. Marshall

A common adage among those who play games that involve balls—baseball, for example—is that one must keep one’s eye on the ball. The baseball player attempting to catch a grounder or fly ball must watch the ball—where it has been, where it is into his or her glove; the player who diverts his or her gaze to where he or she will throw the ball after catching it chances not doing so. One must attend to the first thing first: catch—or hit—the ball. Absent that, nothing matters.

For us in the American Society of Preventive Oncology (ASPO), the first thing is cancer prevention. We must not avert our gaze from prevention, must attend to it, strategically position it within the battle against cancer. We must not lapse into complacency or comfort with the instruments on methods we employ. Our experiments must be as pure, our statistics as elegant, our biology as sophisticated as possible. But our eye must be not wander: not to satisfaction or comfort with the experimental purity, the statistical elegance, nor to the sophistication of the biology we have borrowed. No: it must train on the question: “How can we go about better preventing cancer?”

Our goal is to lessen cancer as a threat to the lives of our fellow citizens in America and throughout the world. We want first to prevent it, to keep it from occurring. Given that it has occurred, we want to help identify the best possible methods of addressing it; our participation in the search for better therapy may help us to identify better means of prevention. Increasingly, we argue that, cancer’s having occurred, we want to help those afflicted to live as positive-fulfilling and unlimited-lives as possible; we want to prevent the deleterious effects of the disease and its therapy. Increasingly, we seek to understand the costs of therapy in light of the increment to life expectancy resulting from that therapy.

In this, my final exercise of the ASPO presidential prerogative, I will begin with a very brief recapitulation of the cancer prevention history that spans the emergence and life to date of ASPO. I will touch on 3 key facets of prevention that have occurred, we want to help those afflicted to live as positive-fulfilling and unlimited-lives as possible; we want to prevent the deleterious effects of the disease and its therapy. Increasingly, we seek to understand the costs of therapy in light of the increment to life expectancy resulting from that therapy.

In this, my final exercise of the ASPO presidential prerogative, I will begin with a very brief recapitulation of the cancer prevention history that spans the emergence and life to date of ASPO. I will touch on 3 key facets of where prevention has been and juxtapose it to where it is now. I will close with some suggestions as to what ASPO needs to do in the future.

Cancer during the Emergence of ASPO

ASPO came into existence in 1976, five years after the enactment of the National Cancer Act. The ASPO mission statement emphasizes prevention. The first of the ASPO objectives is “…to stimulate development and communication on the causes of human cancer, including environmental exposures, lifestyle, and host susceptibility states”. The second is “…to encourage the development and evaluation of new methods and programs for the prevention and early detection of cancer”. For those who founded ASPO in 1975, prevention, defined strictly as causing cancer not to occur, was focal. Indeed, during most of ASPO’s history, prevention has remained at the forefront of concern.

In spite of progress, cancers still kill people. Indeed, for some cancers, we have little to show for our efforts; neither their incidences nor their mortalities have not dropped significantly. From 1975 to 77, in SEER regions, among men and women, respectively, lung cancer killed 78 and 19 per 100,000; by 2004-2006, the figures were 69 and 41 per 100,000. In 1975-77 in SEER areas, pancreatic cancer among men and women, respectively, killed 14 and 9 per 100,000; by 2004-2006, it killed 12 and 9 per 100,000. Between 1975-77 in SEER areas, ovarian cancer killed 10 per 100,000; in 2004-2006, the death toll was 9 per 100,000 (1, 2).

The resources devoted to therapy for many cancers have resulted in what can at best be described as subtle improvements. Between 1976 and 2004, the 5-year relative survivals of male and female lung cancer patients rose from 12 to 14% and from 16 to 19%, respectively. The relative survivals of male and female pancreatic cancer patients improved from 2.7 to 5.6%, and from 2.3 to 5.6%; the relative survival of women diagnosed with ovarian cancer increased from 37 to 46% (3). Prevention of these cancers, which would render the subtlety of these therapeutic advances less consequential, remains as worthy of attention today as in 1975.

For some cancers, the challenge posed by cancer is radically different from what it was at the founding of ASPO in 1976. Table 1 summarizes some changes in the relative 5-year survival of patients diagnosed with more common cancers. The 5-year relative survival of breast cancer patients in 1976 was 75%; by 2004 it was pushing 90%. The 5-year survival of colon cancer patients in 1976 was 52%; by 2004 it was 65%. And the 5-year survival of prostate cancer patients in 1976 was a little less than 70%; by 2004 it was 99%. Among the other major cancers, today, the 5-year relative survival probabilities are, for leukemia, melanoma, non-Hodgkin lymphoma and bladder cancer, respectively, 51%, 92%, 65% and 81%; the majority of patients with one of these can expect to be living 5 or more years after diagnosis (3).

Part of the change involves shifts in stage at diagnosis. During the era around 1976, some 60 to 70 % of prostate cancers diagnosed were localized, while some 20 to 25% were diagnosed with distant metastases (4). Between 2004 and 2006, in the SEER areas, over 92% of prostate cancers
were local or regional in stage (5). In a study that ended in 2003, in a group of closely followed men who were closely followed and periodically screened, over 98% of cancers were organ-confined and diminutive (6). In 1976, 48% of breast cancers were localized, while 8% were diagnosed with distant metastases; by 2006, 64% of cancers were localized, while 6% were diagnosed with distant metastases (5). In 1976, 33% of colorectal cancers were localized and 22% were with distant metastases; by 2004-2006, 42% of colorectal cancers were localized, and 19% were with distant metastases (5). (These last figures are not adjusted for the numbers of colorectal cancers that did not occur because they were prevented by removal of adenomas.)

Cancer Prevention Today: Challenges for Preventionists

Cancer prevention can claim some triumphs, some partial responses, and some failures. Tobacco control, and screening and early diagnosis have received a good deal of attention. Also worth considering is the scientific method on which we have so heavily relied: epidemiology.

Tobacco. Tobacco still, today in the US, causes over 100,000 cancer deaths each year. And the residual effects of smoking will be with us for awhile: given the time required for the risk of smokers to converge toward that of nonsmokers, half of the lung cancers diagnosed this year will be among ex smokers (7-11). This industry continues to seek additional consumers of its products, expending billions each year in marketing and product development, to replace those who quit smoking or die.

Among the more important translational research contributions of the past 5 decades have been changes in tobacco control resulting from research on the smoking behavior. We know that punishing teens for purchasing, possessing, and using tobacco is ineffective, that it does little more than endorse the tobacco industry’s marketing dodge of proclaiming that smoking is only for adults (12, 13). Clean indoor air laws, (14-18) increased taxation, (19) forbidding the use of such deceptive cigarette descriptors as “light” (20) and warning labels (21-24) have had significant effects, decreasing the prevalence of smoking. We are better at identifying smoking addiction (25).

This research and its translation have had effects. Per capita cigarette consumption in 2006 was approximately 40% that of 1976 (3). Cigarette smoking prevalences in 2006 among men and women were each approximately 30% lower than they were in 1976. Legal changes make it increasingly likely that cigarette smoking will soon be a tragic and sad vestige of the 20th century.

Screening and Early Diagnosis. Advocates of cancer screening can rightly point to colon cancer as a major success story. Early evaluations of colon cancer screening indicated that the presence of very small quantities of occult blood in the stool could be an early marker of colorectal cancer, and that screening by means of a fecal occult blood test (FOBT) could decrease colon cancer and colon cancer mortality significantly (26). A definitive clinical trial validated this expectation; however, the data also indicated that approximately one colonoscopic examination would have to be performed for each two people screened, in order to realize this gain (27, 28).

An important advance has involved use of the adenomatous polyp as a very sensitive marker of colorectal cancer risk (29). It had been understood for several decades that the formation of the adenoma is a key step leading to the formation of carcinoma of the colon (30), but visual inspection of the colon by the colonoscope, then by the flexible sigmoidoscope, enabled studies to focus on the identification and ablation of adenomas.

There have been no large, adequately powered randomized trials of colonoscopic screening (31). Indirect data, however, suggest that colonoscopic screening could eliminate 70 to 90 percent of colon cancer and of colon cancer mortality (32). More recent reports indicate that colonoscopy may be less effective than that, especially for the proximal lesions that tend to be most dangerous (33). It is still likely to eliminate 50 to 75% of most colorectal adenoma. It follows that colonoscopic screening could eliminate a substantial proportion of colorectal cancer. Screening by the flexible sigmoidoscope, which can be provided by primary care clinicians, only allows for examination of the distal section of the colon. Studies of flexible sigmoidoscopic screening also shown promising results (34, 35). As other techniques, such as computed tomographic colonography, also known as “virtual colonoscopy”, are developed and refined, these may take a place beside colonoscopy as a means of identifying adenomas (36).

Unfortunately, the number of common cancers for which there are clearly identified premalignant markers is distressingly small. Among the major, common cancers, colon cancer stands alone as issuing from a detectable premalignant condition. Both prostate and breast cancers are detected by indicators of cancer: not by premalignant lesions or conditions (37). There is a good deal of debate about how the benefits of prostate cancer screening by means of PSA might best be realized (38). There is some question, as well, as to the usefulness of mammographic screening of average-risk premenopausal women. The benefits of screening among postmenopausal women, on the other hand, are not widely debated (37). Thus, even though screening for prostate and breast cancer may lessen the human costs they impose, it does not really prevent those cancers. A clearly

### Table 1. Five-year relative survival (%) rates, US, 1975-1977 and 1996-2004

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<td>All Sites</td>
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<td>Breast (female)</td>
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<td>Urinary bladder</td>
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effective means of screening for lung cancer has yet to be devised. In spite of hopes raised for the use of spiral CT screening, data confirming that this new technology will decrease the human cost of lung cancer remain in short supply (39).

The limited successes of screening and early diagnosis raise new issues; they have led to the need for a new focus: survivorship. The often costly cancer treatments-interventions-at our disposal impose side effects that decrease quality of life; if they imposed no costs for quality of life, concern with survivability, with quality of life, would be needless (40-43). But, given the severity and debility that are often the sequela of cancer treatment, even for cancers that are cured, an increased concern has emerged over the balancing of the benefits against the financial and personal costs of treatment (40-45). Cancer therapy must take into account the cost of that therapy to a person’s quality of life as well as the likelihood that his or her cancer would substantially shorten his or her life, or to impose significant burdens of suffering on him or her. The emergence of survivability as a concern of preventionists within ASPO signals a significant shift in emphasis: a complication posed as a result of early diagnostic and, to a more modest extent, therapeutic success.

A challenge likely to emerge in the future concerns cancers that pose little or no immediate threat. The disease for which this is already clear is cancer of the prostate (46). As Table 1 indicates, the relative 5-year survival of prostate cancer patients in 1976 was 69%; prostate cancer patients experienced a 31% decrease in 5-year survival (1-3). Today, the relative 5-year survival of prostate cancer patients is approximately 99%; the threat imposed by early stage prostate cancer increasingly appears quite modest. The change is in the largest part due to the advent and widespread adoption of prostate-specific antigen-based screening (47). Use of that test led, between the late 1980s and early 1990s, to a substantial increase in the diagnosed incidence of prostate cancer. Although this incidence has declined since approximately 2002, it remains substantially above its late 1980s level. Many authorities agree that most of this increased incidence is an artifact, a result of the introduction into the diagnosed incidence of largely indolent prostate cancer cases that would not, prior to the introduction of the PSA test, have been identified (48, 49). The PSA test, affected by a number of factors beside prostate cancer, including benign prostatic hypertrophy and prostatitis, has led to biopsies that have uncovered cancer. There is now, however, accumulating evidence that the prevalence of cancer identified on biopsy is several times as high as the data on prostate cancer mortality would suggest (46). This suggests that only a subset of the prostate cancer identified on biopsy represents a truly malignant condition; the rest—the vast majority—is an indolent, asymptomatic condition with which many to most men will live for the rest of their lives (50). A substantial proportion of these indolent cancers may not need to be cured; but we do not today have the ability to distinguish the cancers that will remain indolent from those that will become aggressive and life-threatening (47, 51). This is important, because prostate cancer treatment often exacts severe physical and psychological costs (52, 53).

For some cancers, and especially for cancer of the prostate, distinguishing the largely indolent cancers from those with a truly malignant and aggressive phenotype has become a major challenge (54-57). Nonetheless, few disciplines are better poised than the one that dominates ASPO-epidemiology, with all its particular flavors-to lead investigations into the distinction of the truly malignant from the more indolent cancer phenotype. This of course requires a shift in the focus of investigation from the simple fact of cancer’s having been diagnosed to the particular form of cancer, especially with respect to its aggressiveness.

**Epidemiology.** Epidemiology-etiologic research within populations at risk—remains the basic science of cancer prevention. We in cancer prevention research have access to a number of methods. Although the ecological method is with some important dissents (58-60) widely dismissed, patterns in ecological aggregates, and changes over time within those ecological aggregates have provided critical insights (WCRF, 2007). The changing risks people experience when they change societies or social environments, have provided important leads.

Experiments can be short term, with changes in biologic markers of cancer risk, as in the growth or regrowth of adenomatous polyps (61-66). Experiments can test intervention effects on biomarkers of carcinogenesis following lifestyle interventions (67) or chemopreventive intervention (68). They can reflect long-term changes, with cancer as the clear outcome, as in the finasteride, (6) dutasteride, (69) tamoxifen (70) and raloxifene (71) trials.

We have for the past 30 years relied heavily on what might be called the omniexposure survey, with cancer as a binary endpoint, and large numbers of subjects queried about as many as possible of their exposures in the recent to distant past. Diet, physical activity, occupation, physical environment, medication, alcohol, tobacco, sunlight, illness history, fertility, sexual practices: all of these have been studied. The association of each of these exposures with the cancer endpoint is first considered. Those that appear most strongly associated with the endpoint are culled for additional analysis, and considered together; in general, the correlation of each of these with the endpoint, with an adjustment intended to eliminate confounding effects due to the associations among the study exposure, the other exposures and the endpoint, represents the culmination of this form of endeavor. It is generally assured that this sifting-sorting process systematically purifies our estimates, thus enabling us to observe the “true” impact of the exposure on risk. Whether it does can be debated (71a). The discussion that typically follows invokes basic science or other epidemiology interpreted as providing assurance to the reader that this association is plausible and that its linkage with the outcome reflects its causal role.

Many of us have used this method, either in case-control or in cohort studies. We are used to it, understand that, under the appropriate circumstances variously outlined some 30 to 50 years ago (72, 73), it will direct us to the right answers.

What omnisurveys of lifestyle share is heavy reliance on self-reports: these are distinct in that they are not just subjective, but nonverifiable. It has been claimed that these self-reports can be validated by replicability, or by comparison to other forms of self-report, such as brief period recall, or the collection of some kind of diary (74).
The survey method has, to be sure, added significantly to what we know about some cancer etiologies; smoking in the respiratory cancers is an example. The survey studies documenting associations of smoking and the respiratory cancers have provided relatively consistent answers, and the data have to an impressive degree coincided with other types of evidence; laboratory animals exposed to tobacco smoke develop cancer, and exposure to several tobacco constituents and metabolites are tumorigenic (75). Smokers who quit experience decreased risk; and the sooner they quit, the less is the impact of their smoking on their cancer and other risks (76, 77). And, clearly, decreasing tobacco use among men in the US has preceded, by about 10 years, substantially decreased lung cancer risk.

Unfortunately, these successes are observed with relatively small numbers of exposure-cancer associations. The performance of epidemiologic methods which contributed so powerfully to the literature on tobacco and smoking do not appear to be generating consistent findings for other exposures or other findings.

In no arena have omnisurveys been used more heavily that with diet. Their performance in that arena is not to date encouraging. The literature is strewn with inconsistent results, and with failure of findings to be supported by findings based on other methods. For example, epidemiologic studies identifying beta carotene as protective against lung cancer have not been confirmed (78, 79); epidemiologic studies of antioxidants against many cancers have not been confirmed (61, 80); studies suggesting that fruits and vegetables and fiber protect against colon cancer have not been confirmed (64, 65, 81, 82). Epidemiologic studies have suggested, although with an embarrassing degree of inconsistency, that a diet high in animal products and low in plant products increases the risk of breast cancer; human experimental studies have provided little to no support for the suggestion.

In a large diet-intervention trial conducted among 48,000 average-risk postmenopausal women, randomization to a dietary intervention had no overall impact on breast cancer risk during a follow-up period of 8 years. A subset analysis indicated that, among women in the highest baseline fat intake quartile, the intervention decreased risk by some 20% (83). One randomized trial indicated that a low-fat diet decreased the risk of breast cancer recurrence; (84) a more comprehensive trial in which the intervention encouraged decreased animal product and fat intake but also substantially increased plant intake had no effect (82).

Myriad reasons have been advanced to explain the lack of convergence among epidemiologic studies; one is that diet and lifestyle have little to do with cancer risk. Another is that the data are simply too messy to provide useful guidance; substantial percentages of the variance observed in food intake and in nutrient intake dietary reports are noise, or error, and the errors are not well enough behaved to allow us to assume enough about them to enable us to appropriately adjust for their presence (85-88). A third is that case control studies do little more than muddle the evidence; they are so methodologically complex and sensitive to statistical aberration that they can not be trusted to provide useful information: cases are sick and likely to recall past exposures differently than cases do, appropriate controls are difficult to identify, and those identified are reluctant to participate in research. A fourth is that the numbers of associations are so large, and so large relative to the number of observations in many of our studies, that our "findings" are little more than perturbations due to the play of random chance. A fifth is that we cannot extrude enough information from strictly observational data to enable appropriate control of confounding (71a).

Whether the problem is that the data are not good enough, or that we are simply overwhelming the pages of our journals with chance findings, these omniexposure lifestyle studies are not, absent imaginative analysis and interpretation, converging. And few to none of these studies have led to interventions that have had any impact on cancer risk.

**ASPO's Charge for the Future**

What does ASPO need to promote? What does prevention need to do?

As prevention scientists, we want better, more elegant, more parsimonious answers, want to impress our fellows. Having settled on certain methods, we tend to view the problems we encounter as best addressed by the use of those methods.

Our principal goal as prevention scientists is not to address questions of biologic mechanics; it is less to regale our fellows with our scientific sophistication than to translate a broad array of knowledge to, in advance, block the course of cancer. We must of course adhere to scientific canon, and our biology must be sound. Forays into biologic mechanisms may be needed, but these, in the use of which we are for the most part amateurs, must be means to obstruct the onset and progress of cancer. Many of the more significant advances in prevention sciences—those of Snow, Semmelweis, Wynder—did not involve breathtaking scientific elegance. Our job as prevention researchers is to prevent the occurrence of or sequelae of cancer; everything else is secondary.

**Tobacco.** We probably don't need a great deal more epidemiologic research on tobacco and cancer. Given its importance as a pathogen, we nearly always need to consider and adjust for it. This requires that we measure it well: the importance of measurement error increases with the strength of an exposure as a risk factor or confounder (85).

Given the ubiquity of tobacco's effects, it would be amazing if there were more than a handful of pathologies not exacerbated by tobacco use. But much more important than additional epidemiology on the impact of tobacco is our need for better means of preventing people from becoming addicted, and of helping the addicted to quit smoking. We clearly need better ways of using social policy to blunt the efforts of the tobacco industry to addict more people. We must develop more effective means to educate the public, to support cessation among cigarette addicts who seek to quit smoking, and to campaign for social policy that will discourage smoking.

For each of these-education, tobacco addiction chemoprevention and policy changes—to succeed in the future will require additional research. Well-intended efforts are not enough. And we cannot blithely assume that the
education and policies of the past century will succeed in the future.

Screening. There is enough evidence on the value of screening that we should redouble our efforts to do it better: to find better markers of risk, especially of premalignant lesions. The cancers for which we have ready markers of very early cancer or premalignant lesions is distressingly small. For cancers of the bladder, stomach, pancreas, ovary, uterine corpus, lung, breast, prostate, the leukemias, lymphoma and multiple myeloma, we have little in the way of premalignant conditions to identify.

This dearth of identified premalignant condition leads should be seen as a continuing challenge. We prevention scientists in ASPO cannot address the need for new screening and early diagnostic approaches alone, on our own. But we need to respond to this need for markers, for means of screening. The world of cancer research today is awash with research and findings on the etiology of the many cancers for which we today have no premalignant markers; we must invigorate our engagement with basic and clinical scientists over etiology, listen to them, work with them, point the way toward effective identification of screening and of early diagnosis markers. We need to link our expertise in population science with that of our colleagues in basic science: they need our guidance and input, as we need theirs.

No screening test will be useful unless those at risk are screened (27). The very challenging task for the behavioral scientists in the ASPO of the future will be to devise sustainable and effective means of encouraging, of arranging, for people to be screened. We need mechanisms that will enable and encourage those at risk to be appropriately screened. We need to more effectively educate our audiences about the need to be screened.

Epidemiology. It may be time for us to face the discomforting truth that epidemiology, with cancer at a specific site, linked to a broad and unfocused mélange of exposures, has given us about all it can; we need now to increase our reliance on other methods. Our papers have filled thousands of pages and pounds of journals; yet few of them have led to meaningful tests of intervention.

Some have suggested that the answer to the inconsistency and lack of convergence of omniposure epidemiology lies in the study of gene-environment interactions. Thus, many in cancer epidemiology, recast as “molecular epidemiology,” now search for genetic variability, as in single nucleotide polymorphisms or haplotypes comprised of sets of these polymorphisms that might define vast differences in the epidemiologic associations that have been the staple of more conventional or what has been described as “traditional” epidemiology. This strategy will generate mountains of new data and findings. Nonetheless, to the degree poor measurement of highly collinear variables with poorly behaved measurement error and very large ratios of variables to observations are problems of “traditional” epidemiology, they are greater problems yet for epidemiology in which study groups are dredged and dissected then dredged again, in the search for subsamples in which associations are noteworthy or statistically significant.

We need to continue to search for risk factors in populations at risk of cancer. However, we may need to narrow our focus, attend to a greater degree to those who are, by virtue of genetics or the formation of premalignant lesions, at elevated risk; we can potentially add significantly as others have by attending to the development, recurrence and the progression of premalignant lesions (89). We may need to focus to a greater extent on the development of the lethal cancer phenotype, as we seek to distinguish those cancers that are readily cured from those that are not. To take the already noted example of prostate cancer, the number of average risk American men who develop prostate cancer is huge relative to the number of patients who develop the highly aggressive forms that kill. We may less need an epidemiology of prostate cancer than epidemiologies of the lethal phenotypes.

As we search for cancer risk predictors, we need to re-examine ourselves of the importance of measurement error; if we cannot measure an exposure well, we cannot assess its importance well; we will misestimate its importance, its role as a confounder and its importance as an effect modifier. There is no getting round it; to ignore the importance of measurement error is to take our eye off the ball. As we seek to measure more adequately, we may need to rely to a greater extent on biomarkers, and on multiple measurement of exposures. To insist on accurate measurement may require that we consider smaller samples and numbers of exposures, rather than unbounded assortments of them; it would be far better to have based our epidemiology on the accurate assessment of exposure in small samples to a small number of factors than on the very imprecise assessment of exposure in huge studies to a huge number of factors.

As we seek to identify the determinants of cancer risk and outcome, we may need to rely to a greater extent on the use of experimentation. As Kuller (90) so wisely pointed out, the finding that an exposure is associated with altered risk needs to be followed, not by an endless cavalcade of replications, but by experimentation. The goal of much of our observational etiologic research is to enable alteration of risk. As we have examined so many facets of lifestyle—smoking, alcohol consumption, dietary practice, physical activity, sexual practices, ponderosity—our goal has been to provide guidelines as to how people should live: how they should change their lifestyles. The finding that those with elevated levels of any exposure are at elevated risk of any cancer is often taken to mean that those with elevated levels can decrease their risk by decreasing their exposure; this is an assumption that almost always needs to be validated by experiment. A commentator recently bemoaned the fact that, in spite of enthusiasm for colonoscopic surveillance, it has not been shown experimentally to be effective in lessening cancer risk (31).

The challenges of cancer prevention have changed in the past 30 years, although we are well placed to seek the advances our society asks of us. Facing these challenges, we will need to keep our eye on the ball. As the tobacco epidemic recedes, the respiratory cancers may decrease in importance. We have excellent reason to pin more hope on expansion of our portfolio of screening regimens; we have yet to realize the full potential of screening. And we need to look carefully and critically.
at the instruments and methods we are using to identify new preventive strategies.

Disclosure of Potential Conflicts of Interest
No potential conflicts of interest were disclosed.

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