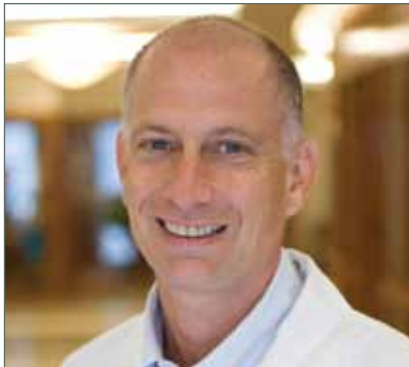


Lung Cancer FRONTIERS

The Forum for Early Diagnosis and Treatment of Lung Cancer



Gerard A. Silvestri, MD, MS, is Professor of Medicine in the Division of Pulmonary and Critical Care, Medical University of South Carolina, Charleston, SC. His interests include lung cancer screening, bronchoscopy, and health services and outcomes research. He is an Associate Editor for the journal **Thorax** and serves on the Board of Directors for The Chest Foundation of the American College of Chest Physicians.

The purpose of **Lung Cancer Frontiers** is to acquire and disseminate new knowledge about lung cancer and how it can be most quickly and effectively diagnosed and treated.

Screening for Lung Cancer

Of course it works, it can't work, does work, doesn't, does...I'm taking my ball and going home...

By Gerard A. Silvestri, MD, MS

Among the controversies in the diagnosis and management of lung cancer, the mention of screening polarizes the gentlest of academics, leads normally balanced people to the brink of madness, and provides endless debate by those on both sides who “know” the answer even while credible scientists continue to study the question. Perhaps this passion is ingrained long before we became health care providers and consumers. Most of us have witnessed the devastation of cancer in friends or relatives while being able to point to a serendipitously discovered “curable” cancer while evaluating someone with gallbladder disease. Who hasn't heard mom say “an apple a day keeps the doctor away” and “an ounce of prevention is better than a pound of cure”?

For many, the utility of screening is now hardwired into their psyche. A study by Schwartz et al.¹ assessed the general population's attitudes towards screening for cancer and found that a substantial proportion of those surveyed believe that if an 80-year-old chose not to be screened, they were irresponsible. Further, 38% had experienced one false positive screening test and 40% of those said it was the scariest experience of their life. Still, nearly all were still glad they had the test. Surprisingly, two thirds of those would be screened even if nothing could be done and nearly three-quarters would prefer total-body CT to \$1000. This study suggests that the American public has bought into screening. So, why don't we just adopt lung cancer screening? Surely it must work. A deeper look into the subject is needed before we can “know” the right answer.

Colorectal, breast and prostate cancer are actively screened for among the adult American population, while lung cancer is not. Improvements in five-year survivorship in those cancers have bolstered the argument for screening. A fact that may surprise readers is that while strong recommendations are made for screening for those cancers, credible evidence suggests that effectiveness is far from assured.^{2,3} For screening to work, the disease must be diagnosed early in asymptomatic patients, must respond better to early versus late treatment, and the

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benefits in treating a small number of patients diagnosed with the disease should outweigh the harm associated with screening a large number of healthy individuals. Does lung cancer screening fit this definition?

Studying the effects of screening for lung cancer is not a new endeavor. Three large, randomized controlled trials beginning in the 60's in the United States, and a fourth in Europe, utilizing chest radiography found no reduction in mortality from lung cancer in the screen detected group.⁴ Two of the trials had a nominal increase in mortality in the screened group, and one showed an improvement in 10-year survival but no change the overall mortality for lung cancer.⁵ Why the paradox in the results of these trials? More cases of lung cancer were detected at an earlier stage and more cases were resected. Survival of the resected patients was improved without decreasing the overall mortality of lung cancer. Researchers pointed towards differing explanations for these findings. Pro-screener believed that the design of the studies was poor, that they were underpowered to answer the question, and that the control group was highly contaminated by those who received the intervention under study (namely, the chest x-ray).^{6,7} Screening skeptics acknowledged the possibility that biases related to screening existed which may result in the appearance that it works when it does not.^{8,9} In the end, the trials were negative and no national organization recommended routine chest radiographs for screening patients for lung cancer.¹⁰⁻¹²

Fast forward to the late 90's, when several groups in Japan and the United States began to evaluate single breath-hold CT scans for screening for lung cancer.¹³⁻¹⁵ The trials had several attributes in common. They were all single-institution trials that discovered a high number of benign, non-calcified nodules (sometimes requiring invasive evaluation and management) and a small percentage of malignant ones. Importantly, the single-arm trials discovered a higher than expected proportion of early stage lung cancer, thus meeting one of the criteria for an effective screening program. Armed with this information, the National Cancer Institute launched one of the largest (50,000 persons) randomized controlled trials for screening ever undertaken (the National Lung Cancer Screening Trial (NLST)).¹⁶ The trial design has been criticized by proponents of screening. However, most feel that a trial of this magnitude can answer the question of whether screening for lung cancer would provide a reduction in mortality from this disease while minimizing harm to those without cancer who require

evaluation. The NLST trial has completed accrual, and initial results are expected sometime in 2010.

One would have thought we could rest easy until then. Not a chance. Two recent publications in high-profile journals ratcheted up the debate. First, the International Early Lung Cancer Action Program (I-ELCAP) expanded the reach of the previously reported single-arm trial to more than 31,000 persons in a multi-institutional, multi-national setting.¹⁷ This study estimated a 10-year survival of their stage I patients at 88% and found that of the 484 cancers discovered, 85% were stage I. National media outlets pounced on this study, and proponents of lung cancer screening suggested that a randomized trial was no longer needed. However, this study has been heavily criticized.¹⁸ The lack of a control group precluded the study from investigating what would happen to a similar group of patients in the absence of screening. In addition, the lack of an unbiased outcome measure meant that the confounding features of lead time, length time, and overdiagnosis bias could not be controlled for. While the study emphasized the positive aspects of screening, it did not discuss potential harms, especially the unnecessary and perhaps harmful evaluation and management of benign disease. Still, the magnitude of this study swayed many to believe that the debate about whether or not lung cancer screening was effective had been settled, in favor of screening.

On the heels of the I-ELCAP publication, Bach et al.¹⁹ reported their analysis of the benefits of screening for lung cancer. In a longitudinal analysis of more than 3,000 current or former smokers in three screening trials, this group chose to evaluate whether a reduction in mortality could be expected from lung cancer screening. They compared the expected and actual new lung cancer resections, advanced lung cancer cases, and deaths from lung cancer, between what was documented in several single-arm screening trials and a model which predicted those events in a population of smokers in this age group who went unscreened. While 144 cases of cancer were diagnosed in the screened group, only 44 were expected. There was a 10-fold increase in the number of patients resected in the screened group, as opposed to what would be expected in this population. Unfortunately, there was no decrease in the number of advanced stage cases or deaths from lung cancer when comparing the screened group to the unscreened cohort. Since this was a model and not a randomized controlled trial, the results must be viewed cautiously. Still, this study

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suggested a significant number of cases discovered during the screening process may be overdiagnosed cancers. Further, if mortality reduction and not survivorship (which parenthetically was similar to that reported in the I-ELCAP study) was the end-point, screening may not be effective. Three-year results from a small, randomized controlled trial in Italy²⁰ seem to support the findings of Bach et al.¹⁹, although this trial is both underpowered and not mature enough to answer the question with any certainty.

The issue of overdiagnosis bias is front and center in this screening debate. What is the evidence for and against this bias? First, we recognize that overdiagnosis occurs in other cancers. Autopsy series find prostate cancer in nearly three-quarters of men over 80 years of age. The vast majority of these men died with their prostate cancer and not from their prostate cancer. Is it possible that this occurs in patients with lung cancer? After seeing so many patients with rapidly progressive, advanced lung cancer who die within a year of diagnosis, it is hard to imagine. A recent study reported by Lindell and colleagues sheds some light on the subject.²¹ This study evaluated patients over five years in a CT screening trial. They had followed 61 patients in their screening program who had at least two successive CT's such that tumor volume doubling time could be calculated prior to eventually undergoing resection. In that study, the mean tumor doubling time of those cancers was 518 days. Nine patients with bronchioloalveolar carcinoma had a mean doubling time of 780 days. Twenty-two patients with adenocarcinoma had a mean doubling time of 746 days. The majority with prolonged doubling times were women, confirming a growing consensus that the biology of lung cancer is different in women. They found a few lesions that actually got smaller on CT before getting larger again. Had these lesions not been discovered in a screening program, one wonders whether or not they would have ever gone on to cause disease in this group of patients. One must also consider the results of the chest radiograph screening trials, which some postulate must be in part due to overdiagnosis of cancer.^{22,23}

Arguments against overdiagnosis rely on autopsy studies in which only 1-3% of patients were found to have lung cancer but died of another cause. Further, some argue that the vast majority of untreated lung cancers documented in large cancer databases have very poor five-year survival. Unfortunately, these data are confounded by the fact that many patients who do not undergo surgical resection for their lung cancer

have other competing causes of death, such as severe lung and heart disease. A subgroup of patients resected in the I-ELCAP screening study was analyzed for biomarkers using immunohistochemistry and fluorescence *in situ* hybridization. The molecular alterations found in these tumors were similar to those found in "conventionally diagnosed" lung cancers. The authors concluded that this is evidence that lung cancers detected by screening are biologically the same as clinically detected cancers and therefore can't be overdiagnosed.²⁴

Unfortunately, what is unknown is whether tumors with similar molecular characteristics behave in a biologically different way. When asked about the biology and growth characteristics of solitary pulmonary nodules, a group of lung cancer experts were unable to agree on the pattern and speed of progression.²⁵ Within the vast basic knowledge of lung cancer, the natural history of this disease remains relatively unknown. This is a critical question, because the possibility exists that small, indolent lung tumors will, at some point in the future, progress to a more aggressive form of lung cancer, which left unchecked would kill the patient. Another plausible theory is that these overdiagnosed tumors will never cause disease. Finally, changes in host immunity could, perhaps, change the trajectory of this disease. The answers to these questions are critical, because for screening to work, predictable tumor biology is needed such that small tumors grow at a certain rate and progress to larger tumors, which eventually metastasize to local, regional, or distant metastatic sites. That scenario may allow for early detection to be an effective tool at reducing mortality for this disease. Were it possible that even small tumors metastasize quickly, or that some tumors never progress past an early stage, screening may be ineffective. This complicates even the simplest of questions around screening – for example, how often should we screen? Is yearly too often, or not often enough?

While we wait for the results of this very important randomized trial (NLST), other questions continue to bubble to the surface. First, how will we deal with the evaluation and management of so many non-calcified solitary pulmonary nodules? Progress in this area has been made and one thing is clear: it appears that the length between follow-up scans for pulmonary nodules will lengthen, and perhaps the number of invasive diagnostic tests will decrease. Still, some patients without lung cancer will be exposed to unnecessary radiation, invasive, costly and perhaps dangerous testing and experience the anxiety of having to undergo that evaluation.²⁵

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While the science of screening is undertaken, significant public policy issues need to be addressed. First, it is not known whether lung cancer screening is cost-effective. While several models have evaluated the subject, the results have varied wildly.^{26,27} These published reports missed the point – lung cancer screening has not proven to be efficacious yet, so estimates of cost-efficacy are a bit premature. Even if lung cancer screening is found efficacious, public policy debate will center on whether or not to put significant resources (one estimate is for 50 billion dollars per year to screen half of the eligible population) into screening, versus funding for smoking prevention and cessation programs, which may be less costly and help to eliminate the future risk for lung cancer. Finally, if screening for lung cancer works, it would be the first time that a screening program is directed at a patient population with a specific, poor behavioral health habit, namely cigarette smoking. One study found that active smokers have poorer

access to health care, are less likely to be able to identify a medical home, have a poor understanding of the risks and benefits of screening, and are less likely to want to be screened for lung cancer than their non-smoking counterparts.²⁸

The literature is replete with studies which underscore the socioeconomic, racial, demographic, and cultural barriers to screening for other cancers. Add now this behavioral barrier and an additional hurdle will have to be overcome to implement an effective screening program.

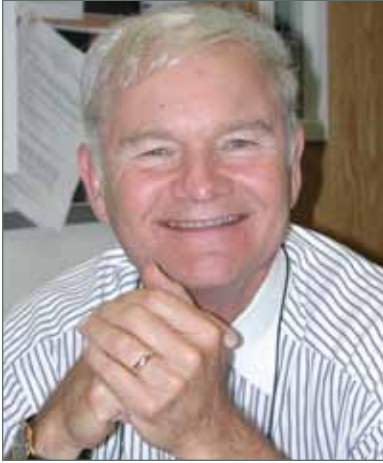
So, back to the beginning, does it work or doesn't it? The answer is far from clear. There is not a clinician or patient who doesn't hope for the sake of the millions of current and former smokers that it does work. We just need to wait. The results of the National Lung Screening Trial should end this debate.... Yeah right. I'm taking my ball and going home.

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Selections from the Peer-Reviewed Literature

By Richard A. Mathay, MD



Richard A. Mathay, MD, is Professor of Internal Medicine, Division of Pulmonary and Critical Care Medicine, Yale School of Medicine, New Haven, CT. His clinical interests include lung cancer, pulmonary hypertension, and the pulmonary manifestations of connective tissue disease. Dr. Mathay is a senior member of the Yale Thoracic Oncology Program (TOP). He is a founding member of the *Lung Cancer Frontiers* Editorial Board.

1. Lung Cancer Incidence, Death Rates, and Cigarette Smoking in the United States

Jemal and colleagues at the American Cancer Society (Jemal A, Siegel R, Ward E, Hao Y, Xu J, Thun MJ, *Cancer statistics, 2009. CA: Cancer J Clin* 2009; 59:225-49) estimate that in the United States among new cancer cases in 2009, in males, lung cancer (15%) will be second to prostate cancer (25%), and in females, lung cancer (14%) will be second to breast cancer (27%). However, it is estimated that in 2009 lung cancer will be the number one cancer killer for both males and females in the United States. In males, lung cancer will be responsible for 30% of all cancer deaths, followed by prostate cancer (9%) and cancer of the colon and rectum (9%). In females, lung cancer will be responsible for 26% of all cancer deaths, followed by breast cancer (15%) and cancer of the colon and rectum (9%).

For the year 2009, Jemal et al. are projecting 159,390 deaths in the United States from lung cancer: 88,900 in males and 70,490 in females. Compared to the data for the year 1990,

this represents a 2.3% decrease in lung cancer deaths among males and a 40% increase in females.

The prevalence of cigarette smoking among adults in the United States has continued to decrease (*MMWR* 2001; 50:869-873 and *MMWR* 2008; 57:1221-1226). In 2007, 19.8% of all adults smoked cigarettes, compared to 23.5% in 1999. Among males, 22.3% smoked, compared to 25.7% in 1999. In 2007, 17.4% of females smoked, compared to 21.5% in 1999.

As of the year 2004, the four states with the highest prevalence of cigarette smoking were Kentucky (27.6%), West Virginia (26.9%), Oklahoma (26.1%) and Tennessee (26.1%) (*MMWR* 2005; 54:1124-1127). The state with the lowest prevalence of cigarette smoking was Utah (10.5%). As expected, the lung cancer incidence rates in the United States from 2001 through 2005 (Jemal et al.) largely paralleled this prevalence of cigarette smoking. The four states with the highest lung cancer incidence rates in males, listed as cases per 100,000 population, were Kentucky (136.2), West Virginia (117.0), Arkansas (113.4) and Louisiana (111.3). Utah had the lowest incidence (39.6). For females, the four states with the highest rates of lung cancer were Kentucky (76.2), Nevada (69.5), West Virginia (69.4) and Delaware (66.2). Utah had the lowest lung cancer rate for females (22.4).

EDITORIAL COMMENT: Thorne et al. (*MMWR* 2008; 57:1221-1226) emphasize that for the first time smoking prevalence among adults in the United States dropped below 20% and, at 19.8%, was significantly lower than in 2006 (20.8%). These authors point out that “during the past 40 years, smoking prevalence has declined overall and among each sociodemographic subpopulation. However, large disparities in smoking prevalence continue to exist by race/ethnicity and education level.” A continuing higher prevalence exists among several populations, such as American Indians/Alaska Natives (36.4%), persons with GED diplomas (44%) and persons reporting family incomes below the federal poverty level (28.8%). Moreover, as noted, there are several states within the United States with persistently high rates of cigarette smoking and lung cancer. There is a need for more effective policy and interventions to reach and assist these populations.

Selections from the Peer-Reviewed Literature

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2. Cannabis Use and the Risk of Lung Cancer: A Case-Control Study

Aldington S, Harwood M, Cox B, Weatherall M, Beckert L, Hansell A, Pritchard A, Robinson G, Beasley R, Cannabis and Respiratory Disease Research Group, Medical Research Institute of New Zealand, Wellington, New Zealand. *Eur Respir J* 2008; 31:280-286

The aim of the present study was to determine the risk of lung cancer associated with cannabis smoking. A case-control study of lung cancer in adults ≤ 55 yrs of age was conducted in eight district health boards in New Zealand. Cases were identified from the New Zealand Cancer Registry and hospital databases. Controls were randomly selected from the electoral roll, with frequency matching to cases in 5-yr age groups and district health boards. Interviewer-administered questionnaires were used to assess possible risk factors, including cannabis use. The relative risk of lung cancer associated with cannabis smoking was estimated by logistic regression. In total, 79 cases of lung cancer and 324 controls were included in the study. The risk of lung cancer increased 8% (95% confidence interval (CI) 2-15) for each joint-yr of cannabis smoking, after adjustment for confounding variables including cigarette smoking, and 7% (95% CI 5-9) for each pack-yr of cigarette smoking, after adjustment for confounding variables including cannabis smoking. The highest tertile of cannabis use was associated with an increased risk of lung cancer (relative risk 5.7 (95% CI 1.5-21.6)), after adjustment for confounding variables including cigarette smoking. In conclusion, the results of the present study indicate that long-term cannabis use increases the risk of lung cancer in young adults.

EDITORIAL COMMENT: Cannabis smoking may have a greater potential than tobacco smoking to cause lung cancer. This population-based study provides evidence to support this concept. The investigators found a major differential risk between cannabis and cigarette smoking, with one joint of cannabis being similar to about 20 cigarettes for risk of lung cancer. This is consistent with the observation of Roth and colleagues (*Am J Respir Crit Care Med* 1998; 157:928-937) that smoking “a few” cannabis joints a day causes similar histological changes in the tracheo-bronchial epithelium as smoking 20-30 tobacco cigarettes a day.

As the authors point out, cannabis smoke is qualitatively similar to tobacco smoke, although it contains up to twice the concentrations of carcinogenic polyaromatic hydrocarbons. Cannabis cigarettes are less densely packed than tobacco cigarettes, and tend to be smoked without filters to a smaller butt size, leading to higher concentrations of smoke inhaled. Smokers of cannabis inhale more deeply and hold their breath longer, facilitating the deposition of the carcinogenic products in the lower respiratory tract.

3. Chronic Obstructive Pulmonary Disease is Associated with Lung Cancer Mortality in a Prospective Study of Never Smokers

Turner MC, Chen Y, Krewski D, Calle EE, Thun MJ, McLaughlin Center for Population Health Risk Assessment, Institute of Population Health, University of Ottawa, Ottawa, Ontario, Canada. *Am J Respir Crit Care Med* 2007; 176:285-290

RATIONALE: Several studies have suggested that previous lung disease may increase the risk of lung cancer. It is important to clarify the association between previous lung disease and lung cancer risk in the general population.

OBJECTIVES: The association between self-reported physician-diagnosed chronic bronchitis and emphysema and lung cancer mortality was examined in a U.S. prospective study of 448,600 lifelong nonsmokers who were cancer-free at baseline.

METHODS: During the 20-year follow-up period from 1982 to 2002, 1,759 lung cancer deaths occurred. Cox proportional hazards models were used to obtain adjusted hazard ratios (HRs) for lung cancer mortality associated with chronic bronchitis and emphysema as well as for both of these diseases together.

MEASUREMENTS AND MAIN RESULTS: Lung cancer mortality was significantly associated with both emphysema (HR, 1.66; 95% confidence interval [CI], 1.06, 2.59) and with the combined endpoint of emphysema and chronic bronchitis (HR, 2.44; 95% CI, 1.22, 4.90) in analyses that combined men and women. No association was observed with chronic bronchitis alone (HR, 0.96; 95% CI, 0.72, 1.28) in the overall analysis, although the association was stronger in men (HR, 1.59; 95% CI, 0.95, 2.66) than women (HR, 0.82;

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95% CI, 0.58, 1.16; p for interaction, 0.04). The association between emphysema and lung cancer was stronger in analyses that excluded early years of follow-up.

CONCLUSIONS: This large prospective study strengthens the evidence that increased lung cancer risk is associated with nonmalignant pulmonary conditions, especially emphysema, even in lifelong nonsmokers.

EDITORIAL COMMENT: Although several studies have suggested that chronic bronchitis and emphysema may increase the risk of lung cancer, most lung cancer cases in published studies occurred in current or former cigarette smokers. Thus, the observed associations may be biased by residual confounding from smoking. The principal finding in this large prospective study is that increased lung cancer mortality was associated

with a history of emphysema, even among persons who had never been active smokers. The association was stronger among those who reported both emphysema and chronic bronchitis, and increased in analyses that excluded early years of follow-up, consistent with causal relationship. Although no association was seen between lung cancer and chronic bronchitis in the overall analysis, there was some suggestion of a sex difference, with chronic bronchitis possibly being more strongly associated with lung cancer in men than women.

As the authors mention, the main limitation of their study and others of this type is that they are unable to distinguish whether COPD is in the causal pathway for lung cancer or whether both COPD and lung cancer are related to an underlying exposure, or some combination of both, with or without inherited familial predisposition.

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