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Chapter Authors: Amitabh Chandra

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“Quantifying the Potential Problem of Overdiagnosis of Ductal Carcinoma in situ in Breast Cancer Screening.” *European Journal of Cancer* 39 (12): 1746–54.

## Comment Amitabh Chandra

It is always slightly terrifying to discuss a chapter by a team with so much intellectual firepower, and in this case doubly so, for Jay Bhattacharya and Alan Garber have taught me so much about health economics. My comments on their work will focus on the broader questions about assessing the productivity of medical spending, be it on cancer or other diseases.

In this chapter the authors demonstrate that the distribution of benefits from medical progress in cancer is not egalitarian. They find that spending on women with breast and colorectal cancer was not cost-effective until the mid-1990s, but started to look remarkably cost-effective after that; a finding that will excite cancer researchers and their advocates everywhere. The earlier period may even have been harmful as survival fell while expenditures increased. In contrast to the results for women, spending on prostate cancer is shown to confer immensely cost-effective benefits.

I have two comments. The first is one that Jonathan Skinner and I make in our paper “Technology Growth and Expenditure Growth in Healthcare” (Chandra and Skinner 2011). Studies of the aggregate productivity of health care spending collapse costs and benefits across technologies to measure the productivity of spending. The chapter by Bhattacharya and colleagues utilizes this framework, as does David Cutler and Murphy and Topel (2006), who estimate an increase in the value of health roughly three times accumulated health care costs during 1970 to 2000. Similarly, Lakdawalla et al. (2010) found high average cost-effectiveness for cancer treatments. A close cousin of this approach is found in the considerable work on geographic variations in spending, where health outcomes are regressed on spending. But in this research (which includes a lot of mine), the returns reflect the weighted means of survival gains and costs across different types of treatments. So it could easily be the case that one treatment is responsible for the bulk of the spending and another for the majority of the survival improvements. And while that does not change the overall conclusion about the cost-effectiveness of medical spending, it certainly changes how sanguine

Amitabh Chandra is economist and professor of public policy at the Harvard Kennedy School of Government, a research fellow at the IZA Institute in Bonn, Germany, and a research associate of the National Bureau of Economic Research.

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(or upset) one should be when confronted with presence or absence of meeting aggregate cost-effective thresholds.

To see this better, imagine that there are two treatments, A and B. Treatment A is useful and costs a dollar, but treatment B is useless and costs \$100,000. Over time we are using more of B. An aggregate productivity calculation that regresses survival on total spending may find that the extra spending “was worth it” and that “we should not cut spending.” Such conclusions leave readers and policymakers thinking that all is right with the world, and that more spending is efficient, and perhaps even necessary. But in reality we could have slashed almost 100 percent of spending without harming patients.

What is the evidence for this concern?

There are two sources of evidence that support my view that aggregate productivity calculations may miss some important facts about mechanisms. Ford et al. (2007) note that 44 percent of the reduction in coronary deaths between 1980 and 2000 was the consequence of changing risk factors related to behaviors rather than health care per se. The implication of this is that aggregate productivity studies may overstate the overall improvement in life expectancy attributable to health care expenditures given that behavioral factors accounted for nearly half of the survival improvement. In terms of what caused the gains in coronary deaths, 35 percent of the decline in mortality was the consequence of inexpensive but highly effective treatments such as aspirin, beta-blockers, blood-thinning drugs, antihypertensives, diuretics, and pharmaceuticals such as ACE inhibitors, anticholesterol drugs (statins), and thrombolytics (“clot-busters”). The marginal cost of these inputs is modest. So almost 80 percent (44 percent + 35 percent) of the survival was caused by relatively inexpensive treatments. Innovations such as angioplasty (stents), bypass surgery, cardiac rehabilitation, and cardio-pulmonary resuscitation (such as automated defibrillators) explained less than 12 percent of the mortality decline, but are responsible for an enormous portion of the costs. And so, regressing outcomes on spending might find that the extra spending was worth it, but that would lose sight of the fact that virtually all the survival gains came from “home run” technologies such as beta-blockers and aspirin.

Another example that is more relevant to the present chapter comes from my work with Mary Beth Landrum and other collaborators (Landrum et al. 2008). We looked at the care of patients with colorectal cancer, and found that high-spending regions are more likely than other regions to use recommended care but are also more likely to use discretionary and nonrecommended care, the latter of which has adverse outcomes for patients. If the time-series variation in cancer treatment mimics the geographic variation in the use of recommended and nonrecommended care, then finding that spending more is cost-effective would miss this important nuance. It might result in our spending more on colorectal cancer when the appropriate pol-

icy response would be the opposite. The same can be said if we find that spending is “cost-ineffective”—the majority of the spending may have been effective but diluted by the presence of expensive but potentially harmful treatments. Absent knowing what is being purchased with the extra money, it is difficult to use aggregate productivity calculations to ascertain the allocative efficiency of what we are spending on cancer or coronary death. For this reason, we should try to ensure that measures of aggregate productivity can be reconciled with what clinical trials have found—so when we say that spending on prostate cancer was effective, are there interventions that diffused over this time that find support for this view?

My second point is about risk-adjustment. As a result of work by Song et al. (2010) and Welch et al. (2011), we now have fairly convincing evidence that claims-based risk-adjustment may be doing the opposite of what we want it to do. If more aggressive providers screen patients more often (not only for cancer, but also for diabetes and hypertension), they will look like they have “sicker” patients. In such a world, controlling for risk will reward exactly the wrong providers—the providers with the most upcoding will get the best risk-adjusted outcomes. What are we to do about this concern? On the one hand, concerns about lead-time bias encourage us to control for more and more (and in particular, to control for stage of diagnoses). I have done this in my own work, but I fear that it was not the right way forward because of concerns about the Will Rogers effect, which is discussed by the authors. Here, higher fidelity scans result in more patients being coded as being stage 3 and 4 patients. Controlling for stage (aka, looking within stage) will make survival look better, but that is entirely a consequence of a compositional change in who is at which stage. The Will Rogers effect would caution against controlling for stage, arguing instead that we should control for tumor size, which is probably the most important predictor of survival. Interestingly, this alternative control should also help with lead-time bias.

I enjoyed reading this chapter—the wealth of information in it is impressive and it forces us to grapple with core issues in measuring the productivity of health care spending.

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