

A response to Pollock et al. (2011)

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Introduction

Pollock et al (2011) criticize Bloom et al. (2011), Gaynor et al. (2010) and Cooper et al. (2011), which examine the effects of competition in the English NHS. In our letter to the *Lancet*, we argued that Pollock et al (2011) seem to be motivated by an ideological opposition to competition, rather than scientific facts. Here we provide here a detailed rebuttal to the issues raised by Pollock et al.

In general, the Pollock et al. review is littered with misstatements and inaccurate descriptions of our three papers. In addition, it contains broader misstatements about issues related to causality and basic econometric principles. We begin with a general discussion of issues related to identifying causation and the need for evidence-based policy. We follow this up with more detailed replies to each of the issues raised by Pollock et al.

However, an important point before we proceed. When using research to assess policies, like competition in the NHS, it is imperative to approach the scientific question at hand an open and objective mind. Research should be judged on its own merits and not whether or not it appears to support a particular political position. It is absurd, of course, to assume that we think that competition will always provide beneficial outcomes to patients. Gaynor and Town (2012) have pointed out that competition in healthcare is not always beneficial. Charlesworth and Cooper (2011) have expressed doubts over the empirical support for price competition. Likewise, the views of Cooper and Propper on this issue were reported by Timmins (2011). Here, much of our concern over price competition is based on Propper et al. (2008), who used research techniques and data similar to our three papers to show that price competition during the NHS internal market increased hospital death rates. Indeed, price competition was taken out of the NHS Health and Social Care Bill, in part, due to interventions by some of the authors of our papers.

Here, it is interesting to note that neither Pollock nor her co-authors seem to have expressed dismay over Propper et al. (2008). This is surprising, since Propper et al. (2008) use similar data and similar methods to Cooper et al., Gaynor et al. and Bloom et al. The lone difference is that Propper et al. (2008) observed that price competition in the NHS harmed patient outcomes.

General concerns about causation. On page 1, Pollock et al. assert that, ‘contrary to Cooper and colleagues’ claims, their study did not show a causal inverse relationship between patient choice and death rates’. What Pollock and her co-authors fail to understand are that the research methods and identification strategy used in our three papers are designed to test causation and build off of advancements in applied econometrics that allow researchers to rigorously assess the impact of policies (rather than rely solely on ideology in the policy-making process).

Over the last twenty-years, a large body of empirical work has emerged that is focused on assessing the impact of policy interventions. This work has taken advantage of newly available large data sets and used statistical and econometric analysis to determine causal relationships between newly introduced policies and their effect on a range of outcomes (Angrist and Pischke, 2010, Ibens and Wooldridge, 2009).

One such strategy that has received considerable attention is difference-in-difference (DD) regression. Difference-in-difference regression forms the core identification strategy used in Cooper et al. (2011) and Gaynor et al. (2010). DD regression, in its most basic form, compares a treatment group that was exposed to a policy with a control group that was not exposed to a policy, before and after the policy was introduced. Assuming that the treatment and control groups were following the same trends prior to the introduction of the policy, then the changes that the policy induces in the exposed group after the reforms were introduced can be used as evidence of causation and capture the effect of this policy. The assumptions underlying the DD strategy are tested in the Cooper et al. and Gaynor et al. papers.

Pollock et al. cite an article by Bradford Hill to illustrate the conditions necessary to imply causation. Unfortunately, Pollock et al.’s reading of Hill (1965) has been too casual. The core research techniques that Hill espouses when he praises Snow (1855) are the very techniques used in two of our papers (and indeed that Propper used with co-authors in an analysis of the internal market of the 1990s).

Bloom et al. use a different technique to try and get at the causal effect of competition on management quality. They exploit the fact that the number of hospitals in a market is partially randomly determined by the importance of local hospitals in the national political process. As hospital closure is never popular, in England’s first past the post system, a politician in a marginal constituency can win potential votes by opposing a hospital closure. But marginality is not very closely associated with resources that go to a hospital because resources are primarily given to PCTs. They test these assumptions and find them to be met (controlling for other factors that may affect hospital competition).

Thus they are able to use the number of marginal constituencies around a hospital's neighbours as an 'instrumental variable' for degree of competition that a hospital faces.

Did patient flows change after the reforms? On Page 1, Pollock et al. assert that Cooper et al. 'do not ascertain whether choice significantly effected the destination of patients'. First, it is important to point out that choice and competition can have an effect without patient flows actually changing. That is because hospitals may respond to the *threat* of potential competition and prospective loss of market share. Second, Pollock et al. casually dismiss work by Gaynor et al. who demonstrate that patient flows did indeed change after the reforms. Gaynor et al. show that, after the introduction of Choose and Book, care-seeking patterns changed. Hospitals which had lower waiting times and lower death rates drew more patients from more neighborhoods and from further away.

Pollock et al also take Cooper et al to task for not mentioning a report by Dixon and colleagues, which found that 30% of patients post reform, went to a non-local provider and 49% of patients were aware of choice (Dixon et al., 2010). We did not view this report as contradicting the results observed in our papers and we note that the Dixon paper was specifically mentioned in Gaynor et al. We view the fact that almost half of NHS patients know that they can choose an alternative provider as evidence that these reforms should have an effect. For example, consistent with the view that patient choice is influencing hospital behavior, the Strategic Plan for the Frimley Park NHS Hospital reveals that improving quality in order to expand market share is explicitly part of their overall business strategy.¹

Use of AMI as a quality indicator: Pollock et al. were critical of the use of AMI mortality as a quality indicator in our papers. Pollock et al. also note that Cooper et al. show that AMI mortality is correlated with waiting times and Length of Stay (LOS). However, they fail to note that Cooper et al. demonstrated empirically that AMI mortality is correlated with overall mortality and patient satisfaction. This is a glaring omission by Pollock et al.. In addition, they dismiss the fact that Gaynor et al. (2010) examine a battery of measures constructed by NCHOD and show that greater competition was associated with improvements in length of stay, overall hospital mortality rates, and overall mortality rates excluding AMI care.

We are all aware that there is no one single measure of quality of care and of the case for and against the use of mortality rates as a quality marker. However, within the international health policy and health economics literature, 30-day AMI mortality is a widely used and accepted measure of general hospital quality. A number of peer reviewed studies looking at the impact of policy reforms (including competition) on hospital quality use 30-day AMI mortality as a quality indicator.² In addition, there are a

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http://www.frimleypark.nhs.uk/images/stories/about_us/publications/FRIMLEY_1112_APR_Strategic_plan.pdf

² Propper et al. (2004) published in the *Journal of Public Economics*; Propper et al. (2008) published in the *Economics Journal*; Kessler and McClellan (2000) published in

number of public and private hospital rankings, which use AMI mortality as a measure of general hospital quality, including the UK Government (see <http://2008ratings.cqc.org.uk/findcareservices/informationabouthealthcareservices.cfm>).³

As Cooper et al. state (page 237), they chose to measure AMI mortality for four reasons. The most dominant reason is that it attenuates bias from endogeneity between hospital quality and measures of market structure (Cooper et al. outline this mathematically in Appendix A on page 251). Cooper et al.'s use of AMI is predicated on the belief that it is correlated with overall hospital performance, but it will not drive the structure of the market for elective care. As a result, as they write in their paper, they hypothesize that hospital competition is leading to overall improvements in hospital management, which lead to reductions in death rates for patients with an AMI. Gaynor et al (2010) provide a model with a more formal explanation of this hypothesis.

Use of In Hospital Mortality: Pollock and her co-authors also criticize Cooper et al.'s use of 30-day in hospital mortality. To bolster their case, they cite Goldacre et al. (2004) for support that in hospital mortality does not capture enough in hospital deaths to be meaningful. Unfortunately, it appears that Pollock and her colleagues did not read the conclusion of Goldacre et al. (2004), who suggest that for AMI, death rates in hospital are not meaningfully different for death rates out of hospital and are a suitable measure of quality. Here, Goldacre concludes, "Within these limitations, we conclude, first, that in-hospital CFRs identify most deaths within 30 days of MI or stroke for patients who survive long enough to reach hospital care. We conclude, secondly, that, for most people who die within 30 days of admission for MI or stroke, these diseases are certified as the underlying cause of death on their death certificates" (pp 341).

Later, Pollock et al. again criticize the use of 30-day AMI mortality use in Cooper et al. (2011) for not excluding those over 74 years of age. But Pollock et al fail to note that Gaynor et al. (2010) *do* test whether excluding those patients alters their findings and find that it does not. In addition, Gaynor et al. (2010) test whether competition reduces AMI mortality *including* deaths in the community as well as overall hospital mortality and overall hospital mortality excluding AMI deaths. Across all three measures of mortality, higher competition is linked to reductions in death rates.

the *Quarterly Journal of Economics*; Kessler and Geppert (2005) published in the *Journal of Economics and Management Strategy*; Shen (2003) published in the *Journal of Health Economics*; Gowriskaran and Town (2003), published in *Health Services Research* (Also includes mortality for patients with pneumonia); Volpp et al. (2003) published in *Health Services Research*.

³ Healthgrades.com – US hospital Rankings; Michigan Hospital Association (aggregated with congestive heart failure and angina); UK Department of Health; California Hospital Outcomes Project; The Greater New York Hospital Association; The University Hospital Consortium.

Depiction of Bloom et al. Research. Pollock et al. refer to work by Bloom et al. as an ‘association with management quality is based on interviews with 161 senior staff and did not take account of relevant causal factors’. This mischaracterizes the work. The management survey was a detailed telephone interview that takes over an hour to complete and has been widely validated as a measure of management quality in a range of settings across the world inside and outside of health care. Using this survey, Bloom et al. then go on to show that management quality and clinical outcomes (such as AMI mortality) is causally related to competition using the instrumental variable strategy described above. This approach controls for a wide range of confounding factors including patient case mix and population density.

Controls for Percutaneous Coronary Angioplasty: Pollock et al. also argue that changes in AMI were due to the uptake of percutaneous coronary angioplasty and unrelated to competition. Pollock et al. fail to note that the Cooper et al. (2011) study directly controlled for whether or not patients had an angioplasty (Appendix C) and also included regional strategic health authority*year interactions. These should capture differential regional trends in AMI care in different regional care, such as the uptake of PCI.

Further, mindful of the roll-out of cardiac networks, Gaynor et al. introduced additional tests to determine whether or not uptake of PCI were driving their results including controlling for hospitals’ annual performance against MINAP collected data on shares of patients receiving thrombolysis treatment before and at hospital arrival, receiving primary angioplasty, and discharged on aspirin, beta blockers and statins. Contrary to Pollock’s claims, it turns out that controlling for these factors actually increases the effect of competition. Again, this was conveniently ignored by Pollock et al.

Measuring Market Structure: Pollock et al. criticize one of the competition measures used in Cooper et al. (2011), although they do not elaborate on their particular criticism. However, they fail to note that Cooper et al. (2011) spend a great deal of time analyzing different measures of competition. They measure competition in 13 separate ways and illustrate that their results across each measure of market structure are consistent. Pollock et al. argue that Cooper et al. exclusively use straight-line distance to measure market structure. Again, they fail to note that Cooper et al. also measure market structure using the time it would take to travel thirty-minutes on the primary road network. Likewise, Pollock et al. fail to note that Gaynor et al. also use two separate measures of market structure and their results remain consistent across both their measures.

Correlation with Urban Density: Pollock et al. argue that criticize Cooper et al.’s for not testing whether urban and rural differences, arguing, “urban and rural provision was mentioned and explored in a subsidiary analysis that used the concentration of secondary schools as a measure of rurality, but urban and rural differences were not integrated into the main analysis”. This is a hugely misleading statement. Pollock and her co-authors fail to note that Cooper et al. directly test whether outcomes in London improved more rapidly than those in the rest of the country and whether outcomes in urban areas improved more rapidly than in areas across the rest of the country in an effort to look at

time variant city effects. Here, outcomes in urban areas did not improve faster than those rural areas and outcomes in London did not improve faster than those in the rest of England. Instead, our results were a direct function of hospital market structure.

Data on Private Providers: Pollock et al. also argue that Cooper et al.'s analysis failed to include data on private sector providers or independent sector treatment centres. However, Pollock et al. again fail to note that Gaynor et al. tested whether the presence of ISTCs was associated with differences in outcomes. They found that it was not.

Conclusion

Given the numerous errors in Pollack et al., the most charitable interpretation is that these authors have failed to properly read or understand the three papers that they heavily lambast. We have rebutted all of their criticisms and none of their points stand up to scrutiny.

A less charitable interpretation of their comment is that they have attacked our research and cherry-picked their discussion in order to further a political aim to stop the Health Bill's progression. There is nothing wrong with opposing the Bill and nothing wrong with criticizing research – this is part of the healthy competition of political and scientific ideas. But to make a lazy and inaccurate attack on research you do not like for ideological reasons is not only bad science but also damages the long-run attempt to make evidence based policy a reality rather than empty rhetoric.

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