Correspondence

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In defence of our research on competition in England's National Health Service

In their Comment (published online Oct 10),¹ Allyson Pollock and colleagues misrepresent our research.²⁻⁴ Although such work might run counter to Pollock and colleagues' prior beliefs, this is not grounds to dismiss it. Nowhere in their review have Pollock and colleagues given evidence that the reforms we studied have harmed patients' outcomes. Instead, they dismiss the research as "flawed" and criticise the data in our analysis in an effort to undermine our findings, which they view as supporting the current UK Government's policies.

Researchers have little influence over how politicians use their academic work. But how researchers discuss evidence that does not support their prior beliefs is of fundamental importance. We have not unilaterally supported competition. Gavnor⁵ has pointed out that competition in health care is not always beneficial. Charlesworth and Cooper⁶ have expressed doubts over the empirical support for price competition. Likewise, the views of Cooper and Propper on this issue were reported by Timmins.7 Much of our concern over price competition is based on research by Propper and colleagues,⁸ who used techniques and data similar to those used in our three current papers²⁻⁴ to show that price competition during the National Health Service internal market increased hospital death rates. Interestingly, Pollock and colleagues have not voiced displeasure with that research.

There is nothing wrong with opposing the current Government's health reforms and nothing wrong with criticising research—this is part of the healthy competition of political and scientific ideas. However, launching inaccurate attacks on research that one does not like for ideological reasons is not only bad science, it also damages the long-run attempt to make evidence-based policy a reality rather than rhetoric. Indeed, our three papers are part of a wider academic effort to assess the effect of policies and investigate their causal effects by use of statistical analysis.⁹

Pollock and colleagues omit key details about our research. For example, they argue that a key shortcoming of the study by Cooper and colleagues³ was that it failed to account for percutaneous coronary intervention. However. Cooper and colleagues directly controlled for whether or not patients had an angioplasty. Further, mindful of the roll-out of cardiac networks, Gaynor and colleagues^₄ controlled for hospitals' share of patients who were receiving thrombolysis treatment before hospital arrival and primary angioplasty, and for those discharged on aspirin, β blockers, or statins.

Pollock and colleagues also lambast the use of acute myocardial infarction (AMI) as a quality measure, arguing that "the fact that they might be correlated with waiting times or length of stay for elective knee replacement or hip replacement does not make them a valid proxy measure of safety or quality of elective care". However, Cooper and colleagues³ also showed that AMI mortality correlates with overall mortality and hospitals' patient satisfaction. Furthermore, Gaynor and colleagues^₄ examined a battery of measures constructed by the National Clinical and Health Outcomes Knowledge Base, and found that greater competition was linked to improvements in length of stay, overall hospital mortality rates, and overall mortality rates excluding AMI care.

Pollock and colleagues do not even accurately present the other work that they cite. Contrary to their assertions, Bradford Hill¹⁰ argued in favour of the type of quasi-natural experiments we implemented, stating "occasionally, it is possible to appeal to experimental or semi-experimental evidence…here the strongest support for the causation hypothesis may be revealed". Indeed, Bradford Hill also lavished praise on Snow,¹¹ who examined the causes of cholera outbreaks in London in what is regarded as the first use of differencein-difference regression.¹² This is the same strategy we used to test the effect of competition.³⁴

No study is perfect, which is why we have peer review and open science. However, the fact that three studies by separate research teams produced consistent results strongly fortifies our collective findings. More work surely needs to be done to understand the changes competition has brought about in England. However, the way forward should be to look objectively to see what is driving our findings, rather than dismissing the results out of hand because they conflict with prior beliefs.

An extended point-by-point response to the Comment by Pollock and colleagues can be found online. We declare that we have no conflicts of interest.

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Authors' reply

In their response to our Comment, Nicholas Bloom and colleagues make four main points. The first is the claim that we misrepresented their research.1 They follow this by saying that "nowhere in the review have Pollock and colleagues given evidence that the reforms we studied have harmed patients' outcomes", but omit to acknowledge that this was not the purpose of our Comment. They then claim that our criticism was politically motivated, and that what they assume are our prior beliefs (without knowing what these are) led to a lack of objectivity. They imply, incorrectly, that because we did not comment on an equally problematic analysis of the 1990s internal market,² we would accept its results, out of political convenience. Finally, they claim that "researchers have little influence over how politicians use their academic work". Although politicians can use academics' research in a manner that they might not have expected, this has not been the fate of Bloom and colleagues' recent research. Their press releases were dramatically headlined-eq, "Hospital competition in the NHS saves lives"-and received extensive media and political attention at a crucial stage in the passage of the Health and Social Care Bill.

Bloom and colleagues fail to specify what is "inaccurate" about our criticisms of their research. Crucially, they have not engaged with our key concern, namely their error in confusing association with causation. The aim of our Comment was to assess whether Cooper and colleagues' paper met the scientific standards used to determine causality. We showed that the paper was fundamentally flawed and failed to reach these standards in several key ways.³

Turning now to the key details that Bloom and colleagues say we omitted in our critique. They say that Cooper and colleagues controlled for angioplasty in their study. The angioplasty rate in the inpatients with acute myocardial infarction (AMI) analysed by Cooper and colleagues was only about 5%. This figure compares with the findings of West and colleagues,⁴ who reported that the proportion of patients with ST-elevation myocardial infarction (STEMI) who were receiving primary percutaneous interventions (PCI) rose from 5% in 2004 to 20% in 2007. West and colleagues also noted a reduction in median "door to balloon" time from 84 min to 61 min during this period-a factor not examined by Cooper and colleagues. The 30-day all-cause mortality rate for hospitals doing primary PCI on less than 25% of STEMI patients was almost double that of hospitals doing primary PCI on more than 75% of patients. Hence, the variation in mortality between hospitals will be strongly affected by the extent to which primary PCI has become an established intervention. This association supports the assertion that it is the degree of hospital specialisation-and not competitionthat is the primary factor in determining outcomes from AMI.

Bloom and colleagues continue to defend the use of AMI as a quality measure without addressing our criticism that other research does not support the use of mortality from AMI as a measure of hospital quality. They also claim that we have misrepresented Bradford Hill by not referring to his quote that "occasionally, it is possible to appeal to experimental or semiexperimental evidence". This quote is neither a defence of their work nor evidence that we misrepresented Bradford Hill. Although Bloom and colleagues have referenced additional articles that discuss the possibility of inferring causation in the absence of an experimental design⁵⁶ in their response, they do not explain why they think these validate their work.



The fact that several research teams have applied the same statistical techniques to similar datasets and obtained compatible results is unsurprising; Bloom and colleagues have not checked to see whether their model or its components have any kind of external validity in the real world. In a review of competition, Bevan and Skellern⁷ state: "there is widespread evidence of variation in quality of care within a hospital; mortality rates of any form do not, therefore, provide a good measure of the quality of elective surgery".

Contrary to the claims made in the press release, the paper by Cooper and colleagues does not prove that competition saves lives. An organisational intervention is not the same as a clinical intervention. Introducing competition is not the same as introducing a new drug or surgical technique or cutting off access to contaminated water. Rather it requires complex technical changes to structures and resources. mediated through countless individuals. organisations. and relationships. Cooper and colleagues did not attempt to measure the intervention or the effect they claim directly. As we make clear in our Comment, there are many good reasons to doubt these findings and proper studies need to be established to test their claim; an experimental approach and primary data collection would be needed.

It is true that "More work surely needs to be done to understand the changes competition has brought about in