Tim Leunig and Hans-Joachim Voth

Comment on Oxley's "Seat of death and terror"


You may cite this version as:
Available online: August 2006

LSE has developed LSE Research Online so that users may access research output of the School. Copyright © and Moral Rights for the papers on this site are retained by the individual authors and/or other copyright owners. Users may download and/or print one copy of any article(s) in LSE Research Online to facilitate their private study or for non-commercial research. You may not engage in further distribution of the material or use it for any profit-making activities or any commercial gain. You may freely distribute the URL ([http://eprints.lse.ac.uk](http://eprints.lse.ac.uk)) of the LSE Research Online website.

This document is the author's final manuscript version of the journal article, incorporating any revisions agreed during the peer review process. Some differences between this version and the publisher's version remain. You are advised to consult the publisher's version if you wish to cite from it.
Comment on Oxley’s “Seat of Death and Terror”*

Timothy Leunig and Hans-Joachim Voth

* We would like to thank Dr Deborah Oxley for sharing her dataset, Mike Dean and Judith Allen for excellent research assistance, Charles Feinstein for his original encouragement to us in writing about this topic, Liam Brunt for helpful advice, and three referees and the editor of this journal for their comments. Needless to say, any remaining errors are our responsibility.

# Timothy Leunig
Department of Economic History
London School of Economics
Houghton Street
London
WC2A 2AE
tel +44 20 7955 7857
fax +44 20 7955 7730
email t.leunig@lse.ac.uk

Hans-Joachim Voth
Economics Department + CREI
Universitat Pompeu Fabra
c/Ramon Trias Fargas 25-27
E-08005 Barcelona
Spain
Comment on “Seat of Death and Terror”

At its best, economic history can be a model for social science research. Open research questions lead to new data being collected. Additional empirical results offer new insights. Oxley’s recent article ‘The seat of death and terror’ sets out to advance our knowledge in this way. Taking its cue from our original article and the discussion in this Review that it caused, she brings new data to the question of how smallpox influenced heights.¹ We begin by summarising Oxley’s main conclusions, before explaining why an alternative interpretation of this new dataset is more plausible.

Oxley makes three claims: that smallpox reduced Londoners’ heights, that it did not reduce juvenile heights in London, and that it did not reduce heights outside London. This leads Oxley to conclude that “smallpox did not stunt growth”, but was instead a proxy for overcrowding.² Those living in overcrowded conditions were more likely to catch smallpox, but it was the overcrowding, and perhaps the consequent disease environment more generally, not smallpox, that made them shorter. This effect increased as they got older and suffered the penalty of overcrowding for longer.

Oxley’s data only partly support her analysis and interpretation. Many of her findings are fragile. First, Oxley excluded a large number of observations, in a way that made finding insignificant effects more likely. Second, the data collected are sub-divided to an extent that makes it highly unlikely to find strong effects from smallpox in each and every subsample. Third, her data show that smallpox mattered more for juveniles, strong evidence of the causal nature of the link between stature and the disease. Fourth, Oxley offers insufficient evidence to support her claim about the link between smallpox and overcrowding. It is contrary to what we know about the London disease environment at this time. Fifth, we show that the effect of smallpox does not differ by location – other Englishmen suffered no less than Londoners, and there is no evidence that smallpox proxied for overcrowding. In short, Oxley’s new data show that smallpox reduced heights.

I
Oxley’s analysis is based on two new data sets that she has collected, transportation records to New South Wales, and Wandsworth jail records. The NSW dataset is the more important of the two: it is more extensive, we know that heights were measured without shoes, and each person’s place of birth is given. For that reason, Oxley’s article concentrates primarily on that dataset, and for reasons of brevity we restrict ourselves to it in this comment.

Oxley acknowledges that, as is so often the case with historical data, the NSW data has its problems. The Marine Society data that we used explicitly recorded a person’s own statement about their medical history. In contrast, the NSW data state only whether someone had visible pockmarks. As Oxley notes, “some 20 to 35% of sufferers failed to exhibit pockmarks”; these smallpox sufferers will have been classified as not having suffered the disease. Of the London-tried transportees, for example, some 20% of 21 year olds are recorded as having had smallpox, but only 11% of 22 year olds appear to have had the disease – despite both surely having grown up in the same disease environment. This misclassification means that both regression co-efficients and t-statistics will be severely biased downwards, so that, as Oxley notes, “the findings uncovered here represent lower-bound estimates”.

II

Oxley collected data on 11,470 NSW transportees, but to make the NSW results comparable with those from the Wandsworth dataset, she generally restricts herself to the 2,420 comparable NSW cases, that is, to those who were born in England and tried in London. On that basis, Oxley finds that those who suffered from smallpox were half an inch shorter than those who had not.

Oxley notes that if smallpox stunted growth “a study confined to those still in their growing years should reveal greater stunting”. This is because juveniles would have had less opportunity to “catch-up” the height lost to smallpox. We made much the same point in our original article. Oxley reran the regression on London-tried transportees only, aged 21 and below, and finds a negative but insignificant result on smallpox. This regression includes only 158 smallpox sufferers. Even if smallpox actually reduced heights by 0.5 inches, the result given in Oxley’s first regression, the
small sample means that there is only a 43% chance of obtaining a statistically significant result. Therefore, finding a negative but statistically insignificant result is not good evidence against the stunting effects of smallpox.\textsuperscript{10}

There is no standard definition of a juvenile since individuals stop growing at different ages. Oxley looks at those aged 21 and under, but it is clearly possible to make the case for other ages.\textsuperscript{11} Both we and Oxley agree that if smallpox caused stunting the effect should be largest at younger ages, decreasing with age as the cumulative opportunities for catch-up increased. In contrast, if smallpox is a proxy for overcrowding, the effect should be largest at older ages, since the effects of overcrowding would be cumulative. We regressed height on smallpox for people below ages up to 24, using the complete dataset to increase the sample size and so the statistical robustness. If the focus of the analysis is on the interaction between smallpox and height, conditional on age, there is no reason to exclude observations from outside London.

Figure 1: Impact of smallpox on average height, by age

Figure 1 shows that smallpox is a statistically significant and negative predictor of height in eight of the ten age groups, and in all ten cases the co-efficient is negative. It
is most unlikely that this result would come about by chance: if you toss a coin ten
times, there is less than a 0.1% chance of getting ten tails. That the results for two are
not significant shows the limitations of this data: visible pockmarks are not the most
reliable way to separate those who had smallpox from those who did not, and the
sample sizes are not sufficient to overcome this problem. The co-efficient is largest at
younger ages, declining to around 0.25-0.4 inches from age 20 upwards. This is the
pattern that both we and Oxley agree should be seen if smallpox caused stunting – a
person of any given age will have had more time to recover from smallpox than
someone younger. That catch-up was only partial is to be expected: net nutritional
status was low overall in this period, particularly for the social classes most likely to
have been transported.\textsuperscript{12}

III

In order to strengthen the claim that smallpox in London was a proxy for
overcrowding, Oxley investigates the effect of smallpox in the rest of the United
Kingdom, and concludes that “The association between pockmarks and stunting in
London is unique.”\textsuperscript{13} Her data do not demonstrate this. Oxley admits that her
classification of NSW transportees into urban and rural counties has problems, noting,
for example, that Lancashire, defined as urban, included “large swathes of Lancashire
countryside”, as well as Liverpool and Manchester, while rural counties often
included “several large towns”.\textsuperscript{14} London is defined to include all of Kent, Essex and
Surrey.\textsuperscript{15} If the data only gave the transportees’ counties, such a classification might
be necessary, but this is not the case. The original records ask for ‘native place’, and
in general the town itself is recorded. Thus, for example, over 60% of the first ship’s
English observations give the name of the town. Oxley takes the name of the town,
finds its county, and then decides whether that county is urban or not. We believe that
it would have been more reliable to count those who stated that they lived in towns of
a certain size as living in urban areas, while classifying those who named a smaller
place, or who named only a county as living in rural areas. The original data also
contain details of the transportees’ work and many occupations are obviously rural:
carter, farm, groom, milk, plough, reap, shears, shepherd, sow and stable, for
example. These data could also have been used to help identify those transportees that
came from rural areas, but unfortunately they were not included in the data collection.
Not withstanding these issues, Oxley finds a negative co-efficient on smallpox in all four of her categories, but with only the result in London statistically significant. Two points are worth making. As table one shows, the larger the proportion of young smallpox sufferers, the stronger the stunting effect of smallpox. Where there are only a small number of young smallpox sufferers, as with the English rural population, the estimated coefficient is small, and the t-statistic low. Where the proportion is high, as in the case of London, the recorded effect is strong – as we would expect if smallpox stunted growth at the time of the disease, with some catch-up possible later on.

Table 1: Effect of smallpox, by subsample, NSW-data

<table>
<thead>
<tr>
<th></th>
<th>London</th>
<th>English rural</th>
<th>English urban</th>
<th>Irish</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Oxley’s smallpox</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>coefficient</td>
<td>-0.440</td>
<td>-0.061</td>
<td>-0.193</td>
<td>-0.143</td>
</tr>
<tr>
<td>t-statistic</td>
<td>2.94</td>
<td>0.41</td>
<td>1.31</td>
<td>1.34</td>
</tr>
</tbody>
</table>

**proportion of all smallpox sufferers by age in geographical subsample**

<table>
<thead>
<tr>
<th></th>
<th>London</th>
<th>English rural</th>
<th>English urban</th>
<th>Irish</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 20: %</td>
<td>32%</td>
<td>19%</td>
<td>27%</td>
<td>21%</td>
</tr>
<tr>
<td>Under 20: N</td>
<td>93</td>
<td>56</td>
<td>88</td>
<td>132</td>
</tr>
<tr>
<td>Under 18: %</td>
<td>15%</td>
<td>6%</td>
<td>10%</td>
<td>9%</td>
</tr>
<tr>
<td>Under 18: N</td>
<td>43</td>
<td>18</td>
<td>33</td>
<td>58</td>
</tr>
</tbody>
</table>

Second, all of the smallpox coefficients in the regressions are negative. Oxley notes this, remarking that ‘it is intriguing that in each case pockmarks were associated with a negative effect’. This is more than intriguing: consistently signed but insignificant coefficients show that the effect is real, but that the sample sizes are individually too small for reliable statistical inference. If we, for example, analysed the data for each ship separately we would find that the co-efficient on juvenile age was consistently positive, but not consistently significant. It would be wrong to use the insignificance of the results to argue that people do not grow as they progress from childhood to adulthood: the consistently positive but insignificant co-efficient on age would show that subdividing the data by ship gives inappropriately small samples.

There are two ways to overcome this problem. The first is to gather more data, so that the sample size becomes sufficient for analysis. This, of course, is not possible.
because history leaves us only certain records. One statistical technique for looking at data that is divided into subsets is “meta-analysis”, used extensively in medical statistics, where data is often gathered from small, separate clinical trials. This is a method of combining the sub-samples in a more sophisticated way than simply aggregating the data. It generates both an overall result, and allows the researcher to test which, if any, of the sub-samples are statistically different from that overall result. We want to know the likelihood that the four negative coefficients reported by Oxley could have come about by chance. The results are given in table 2.

Table 2: Meta analysis results

<table>
<thead>
<tr>
<th></th>
<th>London</th>
<th>English Rural</th>
<th>English Urban</th>
<th>Irish</th>
<th>Meta analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxley’s smallpox coefficient</td>
<td>-0.440</td>
<td>-0.061</td>
<td>-0.193</td>
<td>-0.143</td>
<td>-0.196</td>
</tr>
<tr>
<td>t-statistic</td>
<td>2.94</td>
<td>0.41</td>
<td>1.31</td>
<td>1.34</td>
<td>(2.93)</td>
</tr>
</tbody>
</table>

We use the Sharp-Sterne implementation of the meta-routine. Meta analysis includes fixed effects, z-value in parentheses

The meta analysis gives an estimated overall negative coefficient of minus 0.196 inches, statistically significant at the 1% level. Furthermore, the analysis does not provide evidence that the effect varied by location – the Q-test for heterogeneity yields a statistic of 3.7, which is insignificant (p-value 0.29). There is, therefore, no reason to believe that the variation across subgroups arose by anything other than chance: smallpox reduced heights both inside and outside of London.

The estimated stunting from smallpox given in table 2 is 0.2 inches, which is less than the effect of half an inch that we found earlier. As Oxley accepts, her data provide a lower bound of the true effect because of the problems in coding for smallpox, because 20 to 35% of sufferers failed to show pockmarks. It is straightforward to correct for this using errors-in-variable estimation. We ran an OLS regression on all of the data combined, which estimates the effect of smallpox as 0.22 inches, reassuringly close to our meta-analysis estimate of 0.196 inches. However, if we assume that 20% of smallpox sufferers did not display pockmarks, the estimate rises to 0.28, and if we assume that 35% of sufferers were left unscarred, the figure rises to
0.34 inches; furthermore that estimate is not statistically different to our earlier estimate of 0.5 inches.\textsuperscript{21} It is also worth remembering that the real issue is not whether smallpox reduced heights by a quarter, a third or half an inch, but whether anthropometric history can successfully and reliably capture the conquest of one of the worst diseases to ever afflict human beings.

IV

Oxley argues instead that smallpox was a proxy for overcrowding, which caused stunting either because it was associated with lower nutritional intake, or because it implied ongoing chronic conditions. She bases this contention on her finding that only in London did smallpox reduce height significantly. We have shown that the absence of statistically strong results in other places is caused by a combination of small sample sizes, sample composition, and the use of pockmarks as a means of identifying those who had had smallpox.

There are three reasons to question Oxley’s contention that smallpox is a proxy for overcrowding. First, if it were, we should expect smallpox to be more common amongst overcrowded Londoners. This is not what the NSW data show – 13.7\% of Londoners had smallpox, compared to 14.8\% for other English urban areas, 15.6\% for Ireland, and 9.4\% for English rural areas. Whatever the problems of domestic housing in Ireland, it is not sensible to claim that Ireland was more overcrowded than London.

Second, Oxley argues that those who had not suffered from smallpox lived in better areas: “Wealth might not assist a mucous membrane in resisting an infective particle once it has been inhaled, but it could reduce the chance of encountering the variola virus in the first place by purchasing private space. In terms of the urban and disease environment, Grosvenor Square was a million miles from St Giles.”\textsuperscript{22} Since we know that virtually no transportees came from professional or middle class backgrounds it is inconceivable that more than a handful would have lived in Grosvenor Square or in similar salubrious areas.\textsuperscript{23} Oxley’s hypothesis is also contrary to what we know about the transmission of smallpox: poverty was not a good predictor of catching the disease.\textsuperscript{24} Nor does the evidence support the contention that Grosvenor Square and St Giles are a million miles apart in terms of disease. In the smallpox epidemic of 1772,
for example, St Martin’s district – which includes affluent parishes such as St James Piccadilly and St George Hanover Square – had slightly higher crisis mortality rates than poorer places such as the St Giles, Southwark, or City districts, but slightly lower levels than Holborn or Cripplegate. Smallpox was a disease that could and did spread easily and rapidly across London.

We have shown that smallpox cannot be capturing overcrowding, or severe urban disamenity. Nor, given our earlier findings on the relationship between smallpox and female headed households, is it credible to see smallpox as a proxy for poverty. Instead, we argue that the smallpox variable captures a subgroup of those that had had smallpox. That smallpox would have stunted during the period in which the person was experiencing the disease cannot be in doubt, but the duration of the effect is harder to ascertain. We know that in more recent times, and in populations whose growth rates are well in excess of nineteenth century working class Londoners, the effects of disease on heights are not short lived. Floud Wachter and Gregory report a number of studies to this effect, even for infections that were far less severe than smallpox. Full catch-up growth requires a protein intake three times the usual (modern) recommended level – something that was surely inconceivable for working class people in Britain in this era. Without such a compensating rise in protein intake, smallpox would leave the body weakened, more susceptible to future disease, and less able to make good the growth shortfall caused by smallpox itself. More work needs to be done to assess the effect of different conditions on a person’s ability to make good disease-induced height shortfalls. That would help us understand not only the role of smallpox in determining height, but also the wider question identified by Floud, Wachter and Gregory, of why well-off nineteenth century Britons were short even by the standards of less developed countries today.

Oxley brings new and important data to the question of smallpox’s effect on stature. As is so often the case in historical work, the data are not ideal – visual inspection for pockmarks is not a precise method of ascertaining whether someone had had the disease, especially at older ages. Possibly as many as a third of smallpox sufferers will be misclassified, and as a result, any estimate of the extent of stunting will
automatically and inevitably be biased downwards. Nevertheless, Oxley’s data consistently show that smallpox reduced heights. Her new data allow us to understand the effects of smallpox in a way that had not previous been possible: they show that the smallpox effect was initially large amongst juveniles, but that catch-up growth was able to overcome part, but only part, of the height shortfall.

Oxley is right that the detrimental effect of smallpox was more clearly visible in London, but is incorrect to state that it was not present elsewhere: that result came from using sample sizes too small for the question in hand. Smallpox reduced heights inside and outside London, and there is no evidence from the meta-analysis that the magnitude of this effect differed by location. We show that these effects, considered in combination, are statistically significant and historically meaningful. Oxley’s claims that smallpox was a proxy for overcrowding cannot be entertained: it is not credible to claim that overcrowding was less likely in London than in Ireland, nor that many transportees would have come from salubrious areas characterized by generous amounts of individual private space, nor that smallpox was noticeably more common in one part of London than another. Finally, as Oxley notes, if smallpox was simply a proxy for ongoing chronic overcrowding, or for consistently very low net nutritional status, we would expect to find that the apparent effect of smallpox on heights would increase rather than decrease with age. We do not find that, but rather the reverse: the effect was largest at younger ages.

The evidence shows that smallpox did reduce height. The estimated effect is greater when the data are of higher quality, when the sample size is larger, and when there are more young people in the dataset. The last is important. The effect of smallpox was most pronounced in the young, with older people having partly recovered from the initial height shortfall. Oxley’s new data thus lends further support to our initial conclusion that suffering one of the worst diseases in human history reduced stature.

Bibliography


Nicholas, S. (ed.), *Convict workers: reinterpreting Australia’s past* (Cambridge, 1988)


Appendix

To demonstrate the problems caused by small sample sizes, we conduct the following experiment. Assume that smallpox actually reduced heights by 0.5 inches, to 64.5 inches on average, and that sufferers and non-sufferers are distributed with the standard deviations from Oxley’s full sample. We used a random number generator to obtain two distributions to match Oxley’s full sample – 9,965 and 1,539 respectively. We drew 1000 random subsamples, each equal to Oxley’s sub-sample of 1,086 people without smallpox, and 158 with the disease. On average, there was a 0.501 inches height difference, but in 57% of cases the result was not significant at the 5% level. In other words, using only the English-born London-tried sub-sample means that an insignificant result is more likely than a significant one even if the true difference between the heights of smallpox sufferers and those unaffected is 0.5 inches.²⁹

Figure 2: Distribution of t-test (ratio) statistics from Monte Carlo simulations with 1,000 replications for true differences of 0.5 inches
1 We thank the Australian Research Council for funding the data collection.


3 Ibid., p. 633.

4 Ibid., p. 645.

5 Ibid., p. 645.

6 Ibid., p. 644, table 2, column 3.

7 Ibid., p. 645.

8 Voth and Leunig, ‘Did smallpox reduce height?’, p. 553.


10 See appendix for details.

11 Oxley finds that those aged 22-25 were taller than those aged 21, suggesting that 21 and under is too restrictive, while Nicholas and Shergold make the case that transported convicts had all but stopped growing at 19, but that 24 should be taken as the absolute limit at terminal heights. We therefore include all ages up to 24; readers who believe that an earlier cut off would be more appropriate should simply ignore the results for older ages. Ibid., p. 644, Nicholas (ed.) Convict Workers, p. 80.

12 We know that transported convicts were broadly representative of the English working class, for whom net nutritional status would have been relatively low. Catch up growth requires treble the standard recommended protein allowance, something that was most unlikely to have been possible. Ibid., pp. 70, 74-5, Feinstein ‘Pessimism Perpetuated’, p. 635, Floud, Wachter and Gregory, Height, health and History, p. 26.

13 Oxley, ‘Seat of death and terror’, p. 651.

14 Ibid., p. 648.

15 Floud, Wachter and Gregory, Height, health and history, p. 201 This leads, for example, to two men being classified as Londoners, even though they lived in Tenterden, a small Kentish village 60 miles – a day’s journey – from London (Ship 8, indent 54 and 55).

16 Oxley, ‘Seat of death and terror’, p. 651.

17 A comparison of the original records for ship one and the Oxley dataset suggests that collecting the data on English and Irish transportees who died at sea, but whose details are on the original records, would increase the sample size by 4%.

18 Egger and Smith, ‘Meta-Analysis. Potentials and Promise’.

19 Hedges and Olkin, Statistical methods for meta-analysis.

20 This result is robust. When, for example, we exclude London altogether and restrict ourselves to non-London England and Ireland we still find that smallpox statistically reduced height, and that the size of the effect does not vary by place. The same is true if we restrict the analysis to those aged under 22.

21 Using a signal-to-noise ratio of 0.8 and 0.65 respectively via the STATA routine eivreg. The t-statistic is highly significant at 3.2 in all three cases. It is worth noting that the underlying assumption is that being pockmarked was uncorrelated with the severity of the disease. To the extent to which pockmarked transportees suffered from a worse forms of smallpox, errors-in-variables estimation may overstate the true impact.


23 Only 0.3% and 3.1% of male transportees came from the professional and middle classes, numbers that were low in both absolute terms and relative to British society at the time. Nicholas, Convict Workers, p. 71.

24 In our earlier work we find that being a member of a female headed household was not a strong predictor of having suffered smallpox. Voth and Leunig, ‘Did smallpox reduce height?’, p. 550.

25 Landers, Death and the metropolis, p. 307.

26 They note, for example, that, in Lagos, checks to growth from illness are temporary for the rich, but not for the poor. Floud, Wachter and Gregory, Height, health and history, p. 249.

27 Ibid., p. 261.

28 Ibid., p. 249.

29 The Stata do-file that performs the Monte Carlo simulation is available at http://www.econ.upf.edu/~voth/oxleymc.html.