

Commentary: Social capital, social epidemiology and disease aetiology

George Davey Smith¹ and John Lynch²

The role of social capital in the production of health has developed over recent years into a major academic concern, and is now beginning to feed through into policy discussions concerning the determinants of population health. Social capital has, of course, had greater resonance in fields such as development economics than it has so far had in health, but the confluence of these two threads is now marked. This is made clear by the work of the leading popularizer of social capital—Robert Putnam—who in his seminal 1993 book *Making Democracy Work*¹ explicitly states that health should not be considered an outcome of social capital, saying that:

we must be careful not to give governments credit (or blame) for matters beyond their control. In the language of policy analysis, we want to measure ‘outputs’ rather than ‘outcomes’—health care rather than mortality rates ... Health depends on factors like diet and lifestyle that are beyond the control of any democratic government.¹

Only 7 years later he had dramatically reversed his opinion and decided that:

Of all the domains in which I have traced the consequences of social capital, in none is the importance of social connectedness so well established as in the case of health and well-being.²

The explosion of interest in social capital has not, as yet, led to greater clarity in the conceptualization of exactly what the term social capital refers to and how the supposed connections with health are generated and maintained.^{3,4} We therefore welcome Simon Szreter and Michael Woolcock's⁵ clear and persuasive formulation and think that it will—rightly—become a touchstone for ongoing debates regarding social capital in the health field.

Other commentators in this issue of the *International Journal of Epidemiology*^{6–10} have raised a variety of important points and we will not duplicate these here. Instead we would like to elaborate some specific aspects of the epidemiological interpretation of the historical evidence. This leads to more general considerations of how to interpret social influences on population health. In the central section of their article Szreter and Woolcock discuss mortality crises in 19th century Britain and date improvements in mortality to the 1870s and 1880s, as

Simon Szreter has done previously.¹¹ They develop one specific case, that of Birmingham and the role of Joseph Chamberlain in preaching the ‘civic gospel’ of gas and water socialism over this period. In particular they consider that Chamberlain's activities were central to the development of linking and bridging social capital which protected civil society in Birmingham from the usual nepotism and corruption that sank other British cities in the central decades of the 19th century. Without Chamberlain's activities the rapid urbanization that translated itself into Simon Szreter's four Ds—disruption, deprivation disease and death¹²—would have continued to bear its consequences.

We will argue that consideration of age-specific mortality trends during the second half of 19th century shows that the social capital mechanism suggested by Szreter and Woolcock as being crucial, is an incomplete explanation. We will also address the same specific historical case raised by Szreter and Woolcock and illustrate the negative externalities—in this case international—that can result from the deployment of particular forms of social capital. Our main point is to highlight the need to consider specific, biologically plausible and well-supported aetiological mechanisms when attempting to map aspects of the social environment onto health outcomes.¹³

Mortality declines in 19th and early 20th century Britain: when did they occur?

In their account of the 19th century British mortality decline and the contribution of social capital to this, Szreter and Woolcock take little account of one of the most striking aspects of this decline—the clear cohort patterns^{14,15} (Figures 1 and 2). In the first half of the 19th century there was no obvious trend in mortality, with increases in some larger conurbations and decreases in some rural areas. However, from the mid-19th century onward, a robust pattern emerged, with initial falls in mortality in children aged 1–15, and subsequent declines in cohort-specific fashion for later age bands, such that the same cohort—born around 1845—demonstrated lower mortality than the preceding cohort at each attained age. Thus mortality at older ages only started to decline when the cohorts experiencing the initial mortality declines had entered these older age bands. This pattern was recognized by actuaries,^{16,17} medical scientists^{14,18} and government demographers,¹⁹ and as with any striking and unambiguous regularity is being periodically rediscovered²⁰ (Box 1).

The cohort approach was widely used for mortality projections, as the dotted lines in Figures 1 and 2, from a report by the actuary Derrick, illustrates.¹⁶ In an influential

¹ Department of Social Medicine, University of Bristol, Canynge Hall, Whiteladies Road, Bristol, BS8 2PR, UK. E-mail: zetkin@bristol.ac.uk

² Center for Social Epidemiology and Population Health, Department of Epidemiology, University of Michigan, 1214 South University, Ann Arbor, MI 48104-2548, USA.

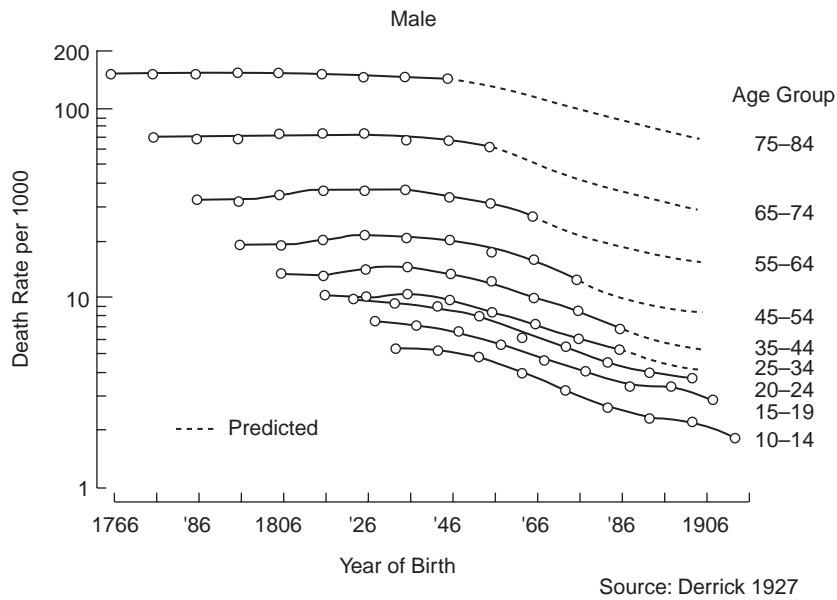


Figure 1 Generational mortality for males. Dotted lines indicate projections

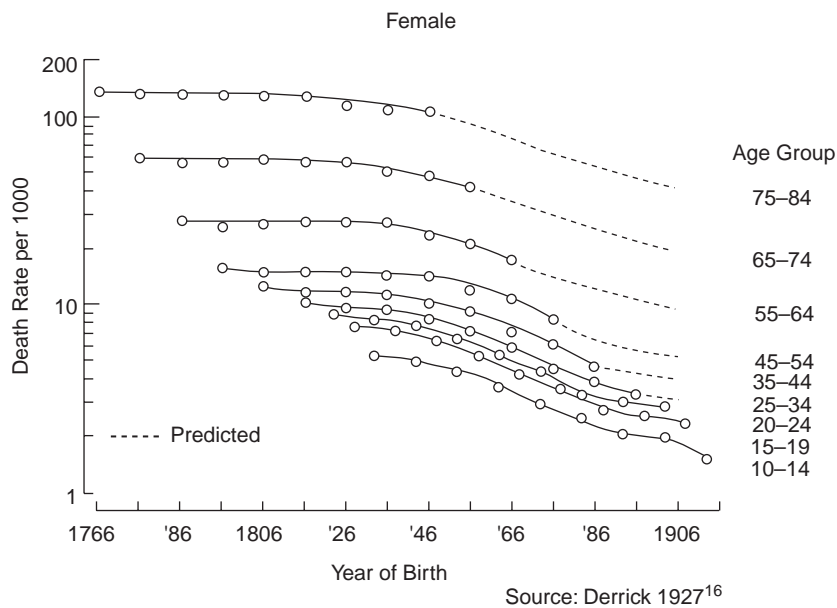


Figure 2 Generational mortality for females. Dotted lines indicate projections

paper Kermack and his colleagues concluded that the mortality data behaved as if: ‘the expectation of life was determined by the conditions which existed during the child’s early years’, and that:

the health of the child is determined by the environmental conditions existing during the years 0–15, and ... the health of the man is determined preponderantly by the physical constitution which the child has built up.¹⁴

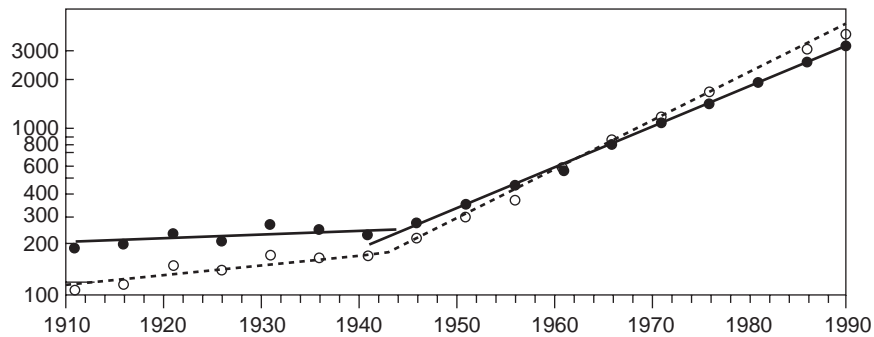
Interestingly Kermack *et al.* noted that infant mortality (under 1 year of age) showed an anomalous pattern, starting to fall only after mortality at later ages had done so. They suggested that infant mortality was dependent upon the health of the mother, and thus improvement in infant mortality followed the

generational improvement in the vitality of women of childbearing age. Thus post-natal influences seemed to underlie the cohort pattern such that infancy and childhood were the important periods. This conclusion has been supported by a recent re-analysis of the same data.²¹ Further evidence that changing environmental factors may have influenced child health and development, but not that of infants, comes from analyses of infant and child (1–4 year old) mortality by socio-economic group in Ipswich in the 1870s which shows a large social gradient in child mortality, but not in infant mortality.²²

The cohort-specific nature of the mortality declines clearly date the underlying factors to the mid-19th century, rather than to the 1870s and 1880s identified by Szreter and Woolcock. In regard to explaining these mortality declines, the data suggest that factors

Box 1**Long live the Queen's subjects**

In Britain people who reach 100 years of age receive a congratulatory telegram from the ruling monarch. In 1997 the nobel prize winning chemist, Max Perutz, reported that the number of people who received a telegram from the current King or Queen in Britain showed a dramatic increase after 1945.²⁰ Overall, the chance of living to 100 increased 12-fold from 1 in 3800 for those born in 1852 to 1 in 310 for those born in 1896. Perutz thought that something must have happened around 1845 that generated this pattern.⁸² He was unaware of the work in the 1920s and 1930s on this issue (MF Perutz, personal communication 1998) and considered that the increase in real wages in Britain that occurred from around the middle of the 19th century on was the important factor.



(from Perutz 1997)

Semilogarithmic plot of the number of centenarians taken from Thatcher (1992) and from Royal Family records (open circles), adjusted for number of births, as described in Perutz 1997⁸².

influencing health, growth and nutrition of infants and children should be the ones considered to be critical. However, the different development of mortality patterns in urban and rural areas²³ has been taken to provide evidence against such an interpretation (Simon Szreter, personal communication). Over 60 years ago Kermack and colleagues dealt with this point, through analysing the cohort effects in the town and country districts in Scotland. They concluded that such analyses provided strong evidence that 'the general level of environment during childhood, the period during which the general constitution of the individual is being built up was key'.²⁴ Their analysis suggested that the persisting higher rates of mortality in urban areas reflected environmental conditions many decades before, not the then current environmental conditions which by the early 1920s were roughly comparable between town and country.

What were the causes of the mortality decline?

Is it plausible that the improved mortality experience of people born after the middle of the 19th century was dependent on conditions experienced early in their lives? The first step is to consider which particular causes of death contributed to the overall adult mortality improvements. The second is to examine the evidence that there are early-life influences on these causes of death.

Respiratory tuberculosis

Despite obvious limitations in the reliability of 19th century and early 20th century cause of death data it is clear that a major contributor to the declines in adult mortality over this period was respiratory tuberculosis (TB).²⁵ It has long been known that TB in

adulthood often reflects reactivation of infection acquired in earlier life, and therefore improvements in early life circumstances should be reflected in reductions in TB mortality at a later age. Birth cohort influences on respiratory TB mortality were recognized by John Brownlee in 1916,²⁶ by the British Registrar General in the 1921 Decennial Supplement²⁷ through to Frost²⁸ and Springett's²⁹ classic analyses. As Mason and Smith concluded in 1985:

until the advent of effective chemotherapy, successive cohorts moved through life as though they had different probabilities of dying by tuberculosis assigned at birth.³⁰

Haemorrhagic stroke

In the 19th century a large majority of strokes would have been haemorrhagic,³¹ and these constituted around 10% of deaths of those aged over 45 in the mid 19th century.²⁵ Haemorrhagic stroke is importantly determined by early-life factors^{32,33} and trends in haemorrhagic stroke show birth cohort effects.³⁴

Bronchitis

Bronchitis, which constituted another 10% of deaths of those aged over 45 in the mid 19th century, also contributed substantially to the mortality decline²⁵ and bronchitis is again a disease for which there is good evidence of early-life influences,³⁵⁻³⁷ particularly in contexts where smoking is rare (as was the case in the 19th century) but poor environmental conditions in childhood are common.

Contribution of these causes to the mortality decline

These three causes—respiratory TB, haemorrhagic stroke and bronchitis—accounted for about two-thirds of the total decline

in mortality for men and women aged 15–64 from the middle of the 19th century to the first decade of the 20th century.²⁵ The case would only be strengthened by inclusion of other important causes of death, such as stomach cancer and rheumatic heart disease, but reliable data for these specific causes are not available. Stomach cancer and rheumatic heart disease have demonstrable infectious influences from infancy and childhood. The proportion, already large, of the total adult mortality decline accounted for by conditions with early-life influences would be even greater if these could be added. What is clear is that diseases that have important early-life origins contributed in a very substantial way to the declining adult mortality rates across the second half of the 19th century and the first three decades of the 20th century. This is consistent with sequential improvements in childhood conditions for successive cohorts born after the middle of the 19th century.

Another source of supporting data comes from attempts to reconstruct the history of height across generations. Floud and colleagues³⁸ suggest that height may have declined among men born during the first half of the 19th century, but showed increases in subsequent cohorts born after the mid-century. Height reflects nutrition and disease environment acting in infancy and childhood, and the height data show a remarkable temporal consistency with the mortality data, with birth cohorts born after 1850 achieving increasingly greater stature and experiencing ever lower mortality.

What changed after 1850?

A search for factors that determined the mortality decline in the second half of the 19th century should, therefore, focus on influences on the health and growth of infants and children that changed around the middle of the century. The processes developing through the 1870s and 1880s, discussed by Szreter and Woolcock as examples of improving social capital, occurred too late to initiate the mortality declines. We need to consider factors that could plausibly be connected to the causes of death contributing to declines in adult mortality, through influences acting during infancy and childhood, and for which a change occurred around 1850. The following meet these criteria to a greater or lesser extent. They are clearly not independent factors—as one will in some cases influence another—but for ease of discussion we have grouped them into separate categories.

Child labour

In the 1820s and 1830s around 10% of 5–9 year olds and three-quarters of 10–14 year olds were in the labour force.³⁹ In the 1830s and 1840s, children made up one to two-thirds of textile mill workers and a third of the workforce in mines.^{39,40} The proportion of children in work declined from the middle of the 19th century,^{39,40} while those who entered the workforce did so at older ages. As Jane Humphries concludes, only the cohorts born after 1850 were able to delay their entry into work.⁴⁰

Robust evidence on the health effects of child labour in the mid 19th century is very limited, although an influence on height amongst coal mining children has been discussed.^{41,42} Current evidence from developing countries, whilst still limited, suggests children often work in hazardous conditions that are detrimental to their health⁴³ and growth.⁴⁴ Long-term consequences on health in later adulthood have also been reported.⁴⁵

Despite this paucity of direct evidence, the notion that child labour has long-term health consequences seems plausible, given the descriptions of the terrible conditions under which children laboured⁴⁶ and our understanding of the aetiological importance of those conditions in contributing to the causes of death related to mortality declines. Overcrowded conditions would contribute to infectious disease transmission, both of TB (with childhood and early adolescence being a key period for initial infection) and of respiratory tract infectious that could influence long-term lung function. Particulate and other matter in air could also have detrimental effects on the long-term respiratory health of children.³⁶ Evidence from developing countries suggests that particulates from solid-burning fuels are not only detrimental to current but also future lung function.^{47,48}

Child labour, through contributing to shorter final stature (which is in turn related to suboptimal development of lung function) is, at the very least, an indicator of childhood exposures that influence risk of later conditions, such as bronchitis and haemorrhagic stroke.

Real wages

‘The standard of living debate’ has exercised economic historians for generations.⁴⁹ In essence this asks whether standards of living improved steadily during the industrial revolution in Britain. Various attempts to compute estimates of real wages and consumption were taken to suggest that general improvements were seen across the whole of the period referred to as the industrial revolution. Over the past decade, however, a general consensus has emerged that improvements were only meaningful over the last decades of the 18th century and then from the middle of the 19th century onwards.⁵⁰ Figure 3 summarizes this, displaying an index of real wages based on estimates by Charles Feinstein.⁵¹ An illustration of such improvements from the mid 19th century also comes from data on consumption of luxury items such as tea and sugar, which show a dramatic increase from around 1850⁵² (Figure 4). Clearly, increased real wages and spending power do not automatically translate into better health outcomes.^{53,54} However, expenditure on housing quality, clothing and food, together with a reduced reliance on paid labour by mothers and children—and thus reductions of proportions of mothers and children in the labour market—could plausibly translate into better conditions and health outcomes amongst children, with consequent long-term effects on adult mortality. It is also possible that such increased real wages, allowing more time to be spent on child care and domestic hygiene, led to improvements in these domains from the mid-19th century onwards.⁵⁵

Nutrition and height

The ‘British Food Puzzle’⁵⁶—that despite apparent real increases in standard of living over the first half of the 19th century there was no evident improvement in nutrition—has ceased to be such a problem with the more recent estimates of real wages. Data on height, mentioned previously, demonstrate strikingly congruent cohort effects to mortality, with improvements occurring from the same birth cohort onward for both height and death rates.^{21,38} Height is influenced by nutrition, health (including infectious disease burden, in particular diarrhoeal disease) and also, perhaps, by drains on energy resources due to the physical

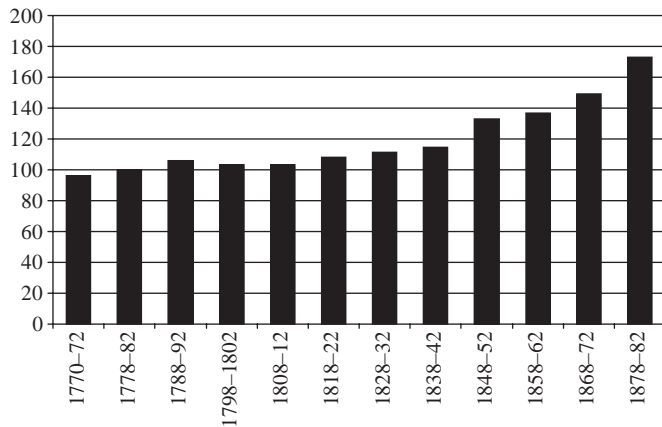


Figure 3 Real earnings, adjusted for unemployment (Great Britain) 1770–1882⁵¹

demands of child labour. Height in turn is related to the causes of adulthood deaths that appear to have declined in cohort-specific fashion to generate the improvements in adult mortality across the end of the 19th and beginning of the 20th centuries.^{33,57,58}

Working mothers

As with child labour the proportion of women and mothers with young children in work appears to have declined from the middle of the 19th century. The degree to which this is a progressive or retrogressive move has been intensely debated, but the evidence suggests that on average childhood mortality was higher if the mother was in work. Thus decreasing female employment could have translated into improved childhood health, with long-term consequences for adulthood health. Again the timing fits in well with the cohort-specific declines in mortality seen across the second half of the 19th and early 20th century. As discussed above, the proportional decrease of women in full-time employment could also allow more time to be spent on child care and domestic hygiene.⁵⁵

Family size

After many decades of increase, a decline in fertility began in the 1830s and although it is very difficult to estimate precise timing, this is likely to have translated into smaller completed family size from around the middle of the 19th century. Smaller completed family size—which also is related to greater birth spacing—leads to less overcrowding, reduced infection risk from airborne and contact transmission, and improvement in the long-term consequences of these. Measles is a highly contagious infection and the results of a study of crowding and child mortality in 19th century Stockholm are informative. Burstrom and colleagues showed that cumulative incidence of measles mortality rose sharply after 12 months of age⁵⁹ and that less crowding had a specific effect on lower mortality from measles, even after control for socio-economic and other confounding factors.⁶⁰ Avoiding measles may also have been associated with improved survival at later ages. Evidence from measles vaccine trials suggests that avoiding measles is associated with far greater mortality gains in the population than can be plausibly explained by measles infection *per se*.⁶¹

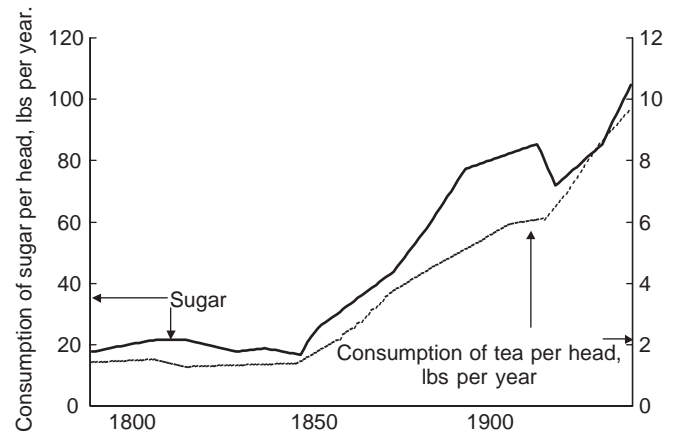


Figure 4 Tea and sugar consumption 1800–1950⁵²

While data do not exist for an earlier period, for men born in the first decades of the 20th century those coming from larger families experienced an increased risk, seven decades later, of haemorrhagic stroke and stomach cancer,³² two of the conditions that are likely to have contributed to the mid-19th century onward decline in adult mortality. Family size in contemporary developing countries is associated with childhood diarrhoea⁶² and respiratory infections. Childhood diarrhoea is related to growth, and respiratory infections may influence long-term respiratory function and thus bronchitis mortality.

Housing

Housing conditions are clearly related to health outcomes,⁶³ and in the 19th century contemporary commentators repeatedly referred to appalling housing as a potential cause of poor health, especially in urban environments. Much evidence points to the mid-19th century as a turning point in this regard, with the average number of people per house, which showed little change from 1801 to 1851, declining after then.⁶⁴ Burnett concludes that housing conditions in England were at their worst during the 1830s and 1840s, and improved subsequently.⁶⁴ This poor situation was clearly recognized at the time and specific housing legislation was introduced in the mid 1840s. Strong statistical relationships between housing density and mortality were demonstrated at the beginning of the 20th century,⁶⁵ and these are likely to have been stronger during earlier periods, when there were even worse overall housing conditions. Indeed a recent analysis by Millward and Bell concludes that improved housing conditions were central to mortality declines in the later part of the 19th century.⁶⁶ Clearly housing conditions could influence morbidity and growth in childhood and these would, in turn, have long-term consequences for adult mortality.

Our discussion here of social processes that demonstrated changes from around the mid-century is illustrative and could be extended to other domains—in particular some aspects of sanitary reform and education. The dating would be similar to that for the factors we have discussed above, when examined across the whole of Britain.

What caused the post-1850 changes?

The post-1850 changes in many of the above factors must be considered in relation to legislative and other social reforms, as well as to broader economic trends and changes in technology that reduced the need for children to work in textile mills and mines. For example, despite clear evidence of slow implementation, the various factory and mines acts—of 1833, 1842, and 1847—limited the extent of child labour and increased the age of starting work, as well as mandating that time be reserved for education of children (although this proved to be largely ineffective in practice). Hopkins concludes that enforcement of these acts was key and that this became increasingly effective from 1850 onwards,⁶⁷ precisely the time that child labour patterns improved and from which cohort improvements in mortality were seen. Many of the other factors—for example improvement in housing and in wages—reflected a mix of legislative and non-legislative social causes.

What led to such changes is a contested issue, but even in a defence of the notion of Victorian equipoise, Harling concludes that the period up to 1848:

was marked by vigorous and at times dangerous political contestation, much more vigorous and dangerous than anything that followed it in the mid-Victorian decades.⁶⁸

Chartism, the beginnings of a trade union movement and more direct—and violent—evidence of working class protest marked these decades.⁶⁸ The degree to which these were met by repression or by amelioration of social conditions through reforms, such as the factory acts, is a matter of long-standing debate.⁶⁹ But clearly some amelioration existed, as was obvious to commentators at the time. In Love and Barton's 1842 *Handbook of Manchester* sanitary reform was seen as, at least in part, being a way of quelling the revolutionary activities and propensity to riot of the working class,⁷⁰ and some decades later the soon-to-be Conservative Prime Minister AJ Balfour declared that social legislation was the 'most effective antidote' to socialism.⁷¹ Within the public health arena, Chadwick's mid 19th century reforms have, in part, been viewed as the amelioration of proletarian living conditions to the extent necessary for the maintenance of social stability.^{72,73}

Simon Szreter, in his seminal critique of Thomas McKeown's theories of mortality decline, stated that 'as so often in matters of causation, precise chronology [is] extremely important.'¹¹ We agree, and think that age and cause of death patterns are also key. In terms of the current exchange on social capital, such an approach leads to different conclusions to those drawn by Szreter and Woolcock. They consider that experiments with state-lead activity in the mid-19th century failed, since health improvements did not occur till later. We point out that this appears not to be the case. The mid-19th century is precisely the time period that needs to be considered in this regard. Instead of focusing on this period Szreter and Woolcock develop the case of Joseph Chamberlain, mayor of Birmingham in 1873–1875 and his networking through non-conformist congregations ('it took a religiously-infused moral movement to motivate the mobilisation of the collective will'), professionals, businessmen, and his middle-class social connections. The working class are here bystanders, awaiting improvements to be brought to them. The empirical problem with this argument is that Chamberlain's activities were too late to have instigated or

driven the 19th century adult mortality declines. In line with other critiques of social capital⁷⁴ we consider that this approach fails to recognise the primacy of the political (in its broadest sense)—a primacy that becomes clearer once the activities leading to the ameliorating reforms, that in turn led to the beginnings of the cohort-specific mortality declines, are considered.^{7,75}

The downside of social capital

It is now well-accepted that there are potential 'down' as well as 'up' sides to greater social capital. Building bonding, bridging and even linking ties within one community or country to the exclusion of others may not generate the desired results for either the included or excluded. After his career in public life in Birmingham, Joseph Chamberlain became the Tory Colonial Secretary in charge of Britain's far-flung empire. Chamberlain stated that:

I have felt for some time that this is a critical period in the history of the Empire. What we do now and what our colonies do will probably in the course of the earlier years of this century settle for all time the question whether a new Empire, such as has never entered into the conception of man before—an Empire bound together by invisible ties and yet of extraordinary strength—whether such an Empire shall be consolidated and maintained or whether we are to drop apart into several atoms, each caring only for our local and parochial interests ... The old idea of dominion was an authority to be used by the central State for its own advantages. The new conception of Empire is of a voluntary organisation based on community of interests and community of sacrifices, to which all should bring their contribution to the common good.⁷⁶

The 'invisible ties' of 'extraordinary strength', the 'voluntary organisation' based on 'community of interests and community of sacrifices' coming together to contribute to 'the common good' sounds like many of the descriptions of social capital in the current literature. An alternative reading of the history of the British Empire might suggest that some members of this community—i.e. the exploited colonies, created and protected by military might—contributed more, but gained less, than others. In similar vein, Robert Putnam has commented on the increased social capital in America after the disaster of 9/11 and the continuing so-called war against terrorism.⁷⁷ But surely this increase in social capital in one country—the USA—has not been beneficial to the health of infants and children in Afghanistan and Iraq.⁴

There is, unfortunately, no *automatic* link between changes thought to enhance the health of one population and either global public health outcomes or a socially progressive agenda. In the case of 19th century Britain, the decrease in the proportion of women in work from the mid-century—which, we suggest, may have contributed to improved long-term health of their offspring—could also be viewed as a key moment in the stabilization of patriarchal social relations. An understandable desire to link socially progressive activities to health improvements could underlie the translation of essentially metaphorical notions of balance, harmony, and lack of psychosocial stress into favourable health outcomes, through postulated mechanisms for which there is little empirical support.^{78,79} This tendency is reflected in Szreter and Woolcock's paper, with frequent allusions to such processes, which is understandable since reification of metaphor is central

to many of the claims of psychosocial epidemiologists working with the social capital concept. However, in the same way as Robert Putnam's claim that joining a club has the same effect on health as stopping smoking² is both misleading and dangerous, it would be wrong to suggest that such links are anything like as well established as they are for the well-documented directly material factors that influence health.

Conclusions

We have shown that mortality in Britain began to decline in cohorts born around 1850. The most important contributions to these mortality declines were from reductions in deaths due to TB, haemorrhagic stroke and bronchitis—all causes of death sensitive to early life conditions. To better understand trends in population health, two issues are key—timing and specificity. In this case, in regard to timing we need to focus on changes in social conditions around 1850 that could be linked to mortality declines. In relation to specificity we need to examine changes in those aspects of social conditions most likely to cause changes in factors causally related to TB, haemorrhagic stroke and bronchitis. We consider that reductions in the amount and age of entry into child labour, improvements in real wages, nutrition, housing and education, together with reduced family size, housing density and overcrowding, are all plausibly linked to the reductions in mortality.

We do not dispute that the enhancements to social capital in the 1870s, persuasively described by Szreter and Woolcock, were important inputs that added to the momentum for better living and working conditions. But what triggered off the perceived need, among Chamberlain's peer group, for such improvements? Absent from Szreter and Woolcock's argument is much of a role for the activities of the large majority of the population themselves—either in defensive action (the friendly societies) or, particularly, in attempts to apply more or less radical pressure for social change.

We think that particular situations require specific explanations, and neither our focus on working-class political activity or on early-life influences on adult mortality patterns will necessarily apply at other times or in other contexts when understanding mortality transformations. In Britain, indeed, a blurring of the cohort effects on mortality declines is evident after the 1930s (compare Figs 5 and 6 with Figs 1 and 2), due to the changing cause of death structure, from adult mortality trends being dominated by changes in death rates from causes such as TB and haemorrhagic stroke, to a pattern in which coronary heart disease and cancers were dominant. Overall, adult mortality trends thus became more influenced by causes of death that have smaller contributions from early life conditions.¹⁸ With the obvious truth of a pattern of combined and uneven development across newly industrializing countries the epidemiological patterns seen in such contexts may be very different from those seen in 19th and early 20th century Britain, and the contribution of early life circumstances could be much less.

Kawachi and colleagues are probably right that '... for better or for worse (in terms of population health outcomes), social capital is here to stay.'⁸ We will continue to see social epidemiological research deploying the concept of social capital despite Szreter and Woolcock correctly pointing out that social capital is likely to remain as one of the 'essentially contested concepts' like gender, race and class, which are:

simply too politically and ideologically important for those at any point on the political spectrum to concede to a definition of the term that they do not see as squaring with their own beliefs, assumptions, and principles.⁵

And yet theoretical development is needed for social capital's scientific deployment in understanding the determinants of population health. Theoretical and definitional development of concepts in social epidemiology are important to better understanding why and how class, race and gender are linked to health.⁸⁰ While the empirical evidence for links between social capital and health is limited, without theoretical

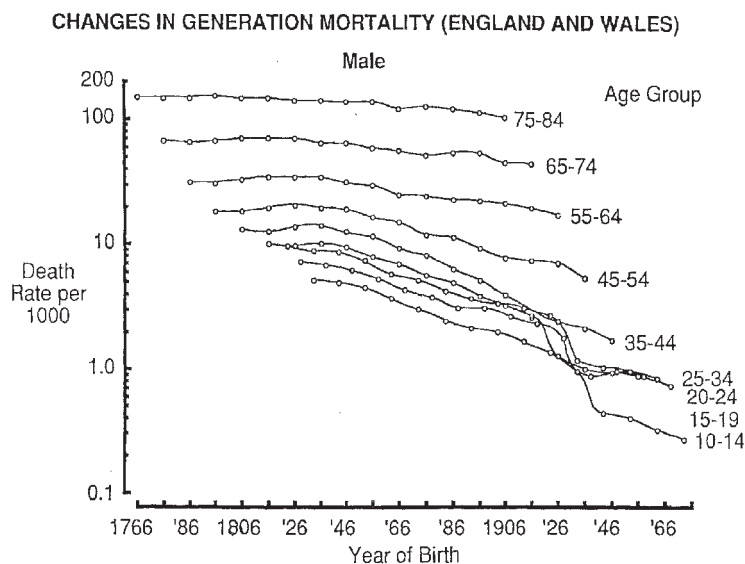


Figure 5 Changes in generation mortality (England and Wales): male

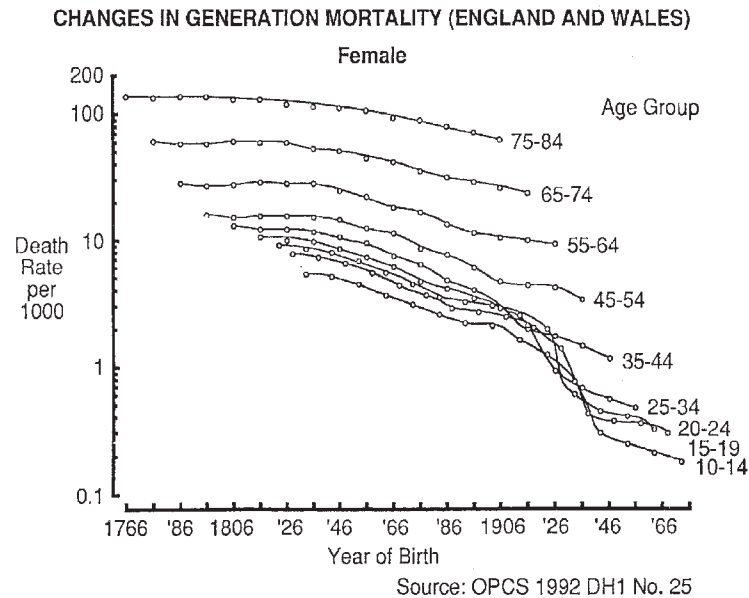


Figure 6 Changes in generation mortality (England and Wales): female

development and greater definitional clarity it is hard to understand how the concept of social capital and its different manifestations across time and place could be linked to the specific risk factors for particular population health outcomes and how these change over time.

Ironically, this very lack of definitional clarity may be of great value to the longevity of the concept of social capital. Where political, bureaucratic, business and sometimes even scientific communication is increasingly dominated by language and concepts intended to obfuscate,⁸¹ where clarity of meaning is the last thing communication is intended to convey, and where public discourse is 'sexed up' and 'dumbed down', social capital may just be the perfect term for our time.

Acknowledgement

George Davey Smith and John Lynch were supported by the Robert Wood Johnson Foundation, Investigator Awards in Health Policy Research Program.

References

- 1 Putnam R. *Making Democracy Work: Civic Traditions in Modern Italy*. Princeton NJ: Princeton University Press, 1993.
- 2 Putnam R. *Bowling Alone*. New York: Simon and Schuster 2000.
- 3 Lynch JW, Due P, Muntaner C, Davey Smith G. Social capital: Is it a good investment strategy for public health? *J Epidemiol Community Health* 2000;**54**:404–08.
- 4 Pearce N, Davey Smith G. Is social capital the key to inequalities in health? *Am J Public Health* 2003;**93**:122–29.
- 5 Szreter S, Woolcock M. Health by association? Social capital, social theory and the political economy of public health. *Int J Epidemiol* 2004;**33**:650–67.
- 6 Navarro V. Commentary: Is capital the solution or the problem? *Int J Epidemiol* 2004;**33**:672–74.
- 7 Muntaner C. Commentary: Social capital, social class, and the slow progress of psychosocial epidemiology. *Int J Epidemiol* 2004;**33**:674–80.
- 8 Kawachi I, Kim D, Coutts A, Subramanian SV. Commentary: Reconciling the three accounts of social capital. *Int J Epidemiol* 2004;**33**:682–90.
- 9 Ellaway A. Commentary: Can subtle refinements of popular concepts be put into practice? *Int J Epidemiol* 2004;**33**:681–82.
- 10 Putnam RD. Commentary: 'Health by association': some comments. *Int J Epidemiol* 2004;**33**:667–71.
- 11 Szreter S. The importance of social intervention in Britain's mortality decline c.1850–1914: a re-interpretation of the role of public health. *Social History of Medicine* 1988;**1**:1–37.
- 12 Szreter S. Economic growth, disruption, deprivation, disease and death: on the importance of the politics of public health. *Popul Dev Rev* 1997;**23**:693–728.
- 13 Lynch JW, Davey Smith G, Harper S, Hillemeier M. Is income inequality a determinant of population health? Part 2. US National and regional trends in income inequality and age- and cause-specific mortality. *Milbank Q* 2004;**82**:355–400.
- 14 Kermack WO, McKendrick AG, McKinlay PL. Death rates in Great Britain and Sweden: Some general regularities and their significance. *Lancet* 1934;**226**:698–703. Reprinted *Int J Epidemiol* 2001;**30**:678–83.
- 15 Kuh D, Davey Smith G. When is mortality risk determined? Historical insights into a current debate. *Social History of Medicine* 1993;**6**:101–23.
- 16 Derrick VPA. Observations on (1) Errors of age in the population statistics of England and Wales, and (2) the changes in mortality indicated by the national records. *Journal of the Institute Actuaries* 1927;**58**:117–59.
- 17 Davidson AR, Reid AR. On the calculation of rates of mortality. *Transactions of the Faculty of Actuaries* 1927;**11**:183–232.
- 18 Davey Smith G, Kuh D. Commentary: William Ogilvy Kermack and the childhood origins of adult health and disease. *Int J Epidemiol* 2001;**30**:696–703.
- 19 Tutt LWG. The mortality aspect of population projections. *Transactions of the Faculty of Actuaries* 1953;**21**:3–50.

- 20 Perutz MF. Long live the Queen's subjects. *Phil Trans R Soc Lond B* 1997;**352**:1919–20.
- 21 Harris B. Commentary: 'The child is father of the man.' The relationship between child health and adult mortality in the 19th and 20th centuries. *Int J Epidemiol* 2001;**30**:688–96.
- 22 Razzell P, Spence C. Poverty or disease environment? The history of mortality in Britain 1500–1950. In: Breschi M, Pozzi L (eds). *The Determinants of Infant and Child Mortality in Past European Populations*. Udine, Italy: Forum, 2004.
- 23 Szreter S, Mooney G. Urbanisation, mortality and the standard of living debate: new estimates of the expectation of life at birth in nineteenth-century British cities. *Economic History Review* 1998;**50**: 84–112.
- 24 Barclay RS, Kermack WO, McKendrick AG. Comparison of the specific mortality rates in town and country districts of Scotland since 1871. *J Hyg* 1940;**40**:423–33.
- 25 Charlton J, Murphy M. Trends in causes of mortality: 1841–1994—an overview. In: Charlton J, Murphy M (eds). *The Health of Adult Britain: 1841–1994, Vol. 1*. London: The Stationery Office, 1997, pp. 30–57.
- 26 Brownlee J. Certain considerations regarding the epidemiology of *Phthisis pulmonalis*. *Public Health* 1916;**29**:130–45.
- 27 Her Majesty's Stationery Office. *The Registrar-General's Decennial Supplement England and Wales 1921. Part III: Estimates of Population Statistics of Marriages Births and Deaths 1911–1920*. London: HMSO, 1933.
- 28 Frost WH. The age-selection of tuberculosis mortality in successive decades. *Am J Hyg* 1939;**30**:91–96.
- 29 Springett VH. An interpretation of statistical trends in tuberculosis. *Lancet* 1952;**March 15**:521–25.
- 30 Mason WM, Smith HL. Age-period-cohort analysis and the study of deaths from pulmonary tuberculosis. In: Mason WM, Fienberg SE (eds). *Cohort Analysis in Social Research: Beyond the Identification Problem*. New York: Springer-Verlag 1985, pp. 151–227.
- 31 Lawlor D, Davey Smith G, Leon D, Sterne J, Ebrahim S. Secular trends in mortality by stroke subtype in the 20th century: a retrospective analysis. *Lancet* 2002;**360**:1818–23.
- 32 Hart CL, Davey Smith G. Relation between number of siblings and adult mortality and stroke risk: 25 year follow up of men in the Collaborative study. *J Epidemiol Community Health* 2003;**57**: 385–91.
- 33 Song YM, Davey Smith G, Sung J. Adult height and cause-specific mortality: a large prospective study of Korean men. *Am J Epidemiol* 2003;**158**:479–85.
- 34 Olalla MT, Medrano MJ, Sierra MJ, Almazán J. Time trends, cohort effect and spatial distribution of cerebrovascular disease mortality in Spain. *Eur J Epidemiol* 1999;**15**:331–39.
- 35 Davey Smith G, Hart C, Blane D, Hole D. Adverse socioeconomic conditions in childhood and cause specific adult mortality: prospective observational study. *BMJ* 1998;**316**:1631–35.
- 36 Strachan DP, Sheikh A. A lifecourse approach to respiratory and allergic diseases. In: Kuh D, Ben-Shlomo Y. *A Life Course Approach to Chronic Disease Epidemiology*. 2nd Edn. Oxford: Oxford University Press, 2004.
- 37 Elo IT, Preston, SH. Effects of early-life conditions on adult mortality: a review. *Popul Index* 1992;**58**:186–212.
- 38 Floud R, Wachter K, Gregory A. *Height, Health and History*. Cambridge: Cambridge University Press, 1990.
- 39 Horrell S, Humphries J. 'The exploitation of little children': Child labor and the family economy in the industrial revolution. *Explorations in Economic History* 1995;**32**:485–516.
- 40 Humphries J. Child labor: lessons from the historical experience of today's industrial economies. *The World Bank Economic Review* 2003;**17**:175–96.
- 41 Kirby P. Causes of short stature among coal mining children, 1823–1850. *Economic History Review* 1995;**48**:687–99.
- 42 Humphries J. Short stature among coal mining children: A comment. *Economic History Review* 1997;**50**:531–37.
- 43 Scanlon TJ, Prior V, Lamarao MLN, Lynch MA, Scanlon F. Child labor. *BMJ* 2002;**325**:401–03.
- 44 Hawamdeh H, Spencer N. The effects of work on the growth of Jordanian boys. *Child Care Health Dev* 2003;**29**:167–72.
- 45 Kassouf AL, McKee M, Mossialos E. Early entrance to the job market and its effect on adult health: evidence from Brazil. *Health Policy Plann* 2001;**16**:21–28.
- 46 Engels F. (1987, first published 1845) *The Condition of the Working Class in England*. Harmondsworth: Penguin.
- 47 Smith KR, Mehta S. The burden of disease from indoor air pollution in developing countries: comparison of estimates. *Int J Hyg Environ Health* 2003;**206**:279–89.
- 48 Sunyer J. Urban air pollution and chronic obstructive pulmonary disease: a review. *Eur Respir J* 2001;**17**:1024–33.
- 49 Mokyr J. The new economic history and the Industrial Revolution. In: Moyker J. (ed.). *The British Industrial Revolution: An Economic Perspective*. 2nd Edn. Colorado: Westview Press, 1999.
- 50 More C. *Understanding the Industrial Revolution*. London: Routledge, 2000.
- 51 Feinstein CH. Pessimism perpetuated: real wages and the standard of living in Britain during and after the industrial revolution. *Journal of Economic History* 1998;**58**:625–58.
- 52 Hobsbawm E. *Industry and Empire*. 2nd Edn. Harmondsworth: Penguin Books, 1999.
- 53 Davey Smith G. *A Lifecourse Approach to Health Inequalities*. Bristol: Policy Press, 2003.
- 54 Johansson SR. Death and the doctors: elite mortality in Britain from 1500 to 1800. Cambridge Group for the History of Population and Social Structure. Working Paper Series: No. 7, 1999.
- 55 Mokyr J. Why 'more work for mother?' Knowledge and household behavior, 1870–1945. *Journal of Economic History* 2000;**60**:1–41.
- 56 Clark G, Huberman M, Lindert PH. A British food puzzle, 1770–1850. *Economic History Review* 1995;**48**:215–37.
- 57 McCarron P, Hart CL, Hole D, Davey Smith G. The relation between adult height and haemorrhagic and ischaemic stroke in the Renfrew/Paisley study. *J Epidemiol Community Health* 2001;**55**:404–5.
- 58 Davey Smith G, Hart C, Upton M, Hole D, Gillis C, Watt G, Hawthorne V. Height and risk of death among men and women: aetiological implications of associations with cardiorespiratory disease and cancer mortality. *J Epidemiol Community Health* 2000;**54**:97–103.
- 59 Burstrom B, Aaby P, Mutie DM. Validity of measles mortality data using hospital registers and community surveys. *Int J Epidemiol* 1995;**24**:625–29.
- 60 Burstrom B, Diderichsen F, Smedman L. Child mortality in Stockholm during 1885–1910: the impact of household size and number of children in the family on the risk of death from measles. *Am J Epidemiol* 1999;**149**:1134–41.
- 61 Aaby P, Bhuiya A, Nahar L, Knudsen K, de Francisco A, Strong M. The survival benefit of measles immunization may not be explained entirely by the prevention of measles disease: a community study from rural Bangladesh. *Int J Epidemiol* 2003;**32**:106–16.
- 62 Gorter AC, Sandiford P, Davey Smith G, Pauw JP. Water supply, sanitation and diarrhoeal disease in Nicaragua: results from a case-control study. *Int J Epidemiol* 1991;**20**:527–33.
- 63 Shaw M. Housing and public health. *Annu Rev Public Health* 2004;**25**:397–418.
- 64 Burnett J. Housing and the decline in mortality. In: Schofield R, Reher D, Bideau A (eds). *The Decline of Mortality in Europe*. Oxford: Clarendon Press, 1991.

- ⁶⁵ Bowley AL. Death-rates, density, population, and housing. *J R Statist Soc* 1923;**86**:516–46.
- ⁶⁶ Millward R, Bell FN. Economic factors in the decline of mortality in late nineteenth century Britain. *European Review of Economic History* 1998;**2**:263–88.
- ⁶⁷ Hopkins E. *A Social History of the English Working Classes, 1815–1945*. London: Edward Arnold, 1979.
- ⁶⁸ Harling P. Equipose regained? Recent trends in British political history, 1790–1867. *Journal of Modern History* 2003;**75**:890–918.
- ⁶⁹ Taylor M. Rethinking the Chartists: searching for synthesis in the historiography of chartism. *The Historical Journal* 1996;**39**:479–95.
- ⁷⁰ Douglas I, Hodson R, Lawson N. Industry, environment and health through 200 years in Manchester. *Ecological Economics* 2002;**41**: 235–55.
- ⁷¹ Murray P. *Poverty and Welfare 1830–1914*. London: Hodder and Stoughton, 1999.
- ⁷² Hamlin C, Sheard S. Revolutions in Public health: 1848 and 1998? *Br Med J* 1998;**317**:587–91.
- ⁷³ Hamlin C. *Public health and social justice in the age of Chadwick*. Cambridge: Cambridge University Press, 1998.
- ⁷⁴ Harriss J. *Depoliticizing Development: the World Bank and Social Capital*. New Delhi: Leftword, 2001.
- ⁷⁵ Navarro V, Shi LY. The political context of social inequalities and health. *Int J Health Serv* 2001;**31**:1–21.
- ⁷⁶ Garvin JL. *Life of Joseph Chamberlain. Vol. 5 Joseph Chamberlain and the Tariff Reform Campaign*. London: Macmillan, 1969.
- ⁷⁷ Putnam R. A better society in a time of war. *New York Times*. 19 October 2001.
- ⁷⁸ Davey Smith G, Ben-Shlomo Y, Lynch J. Life course approaches to inequalities in coronary heart disease risk. In: Stansfield SA, Marmot MG (eds). *Stress and the Heart*. London: BMJ Books, 2002.
- ⁷⁹ Davey Smith G. The biopsychosocial approach: a note of caution. In: White PD (ed.). *Understanding Illness: the Biopsychosocial Approach*. Oxford: Oxford University Press, (in press).
- ⁸⁰ Krieger N. Theories for social epidemiology in the 21st century: an ecosocial perspective. *Int J Epidemiol* 2001;**30**:668–77.
- ⁸¹ Watson D. *Death Sentence. The Decay of Public Language*. Sydney: Knopf, 2003.
- ⁸² Centenarians. And they all lived happily ever after. *Economist* 7 February 1998.

IJE vol.33 no.4 © International Epidemiological Association 2004; all rights reserved.
Advance Access publication 28 July 2004

International Journal of Epidemiology 2004;**33**:700–704
doi:10.1093/ije/dyh263

Rejoinder: Crafting rigorous and relevant social theory for public health policy

Simon Szreter and Michael Woolcock

We wish to express our sincere thanks to the editors of the *International Journal of Epidemiology* for hosting such an interesting exchange on the idea of social capital and its application to public health. We are flattered that scholars and practitioners of such repute have responded so vigorously to our paper, and thank them also for their efforts.

Obviously we cannot hope to respond to every point raised by the discussants: some comments are more constructive than others and, given the relatively short space available to us, some provide a more obvious entry point for a reply than others. Let us begin, however, with several initial clarifying remarks, before proceeding to a more detailed response. It is emphatically *not* our view, and nowhere in the paper do we claim: (1) that social capital (however conceived) is the sole (or always primary) variable that explains (or should be used to try to explain) public health outcomes (as claimed or implied by three reviewers); (2) that our *definition* of social capital includes ‘the state’;² (3) that empirical indicators of ‘linking social capital’ are limited to positive outcomes alone, like ‘good governance’;³ (4) that by adopting the social capital terminology we are arguing (implicitly or explicitly) that ‘the state’ must necessarily retreat;⁴ and/or (5) that enhancing ‘competitiveness’ and the

‘imperialist’ reach of ‘capitalism’ is the overriding (wittingly or unwittingly) objective when using the language of ‘capital’.^{4*} In the passages below, we endeavour to restate and clarify our position by putting it in a broader context of what social theory in general seeks to accomplish, what our particular articulation of social capital theory seeks to accomplish, and what the methodological and practical implications are of pursuing our line of (evidence-based) reasoning in the field of public health.

Social theory: what should it seek to accomplish?

The broad dialectical challenge in social theory is (or should be) addressing the structure-agency problem (also known as the micro-macro problem)⁶—that is, unpacking the interactions and interconnections between individual choices and larger institutional forces.⁷ Unfortunately, much of the impetus in social theory in recent decades—with self-contained camps emerging

* The Soviet Union had capital (in many forms), Marx wrote a book on capital, and indeed, according to an excellent intellectual history, was the first person to coin the phrase social capital.⁵