Chapter Twelve

Revealing the Hidden Affliction

How Much Infertility Was Due to Venereal Disease in England and Wales on the Eve of the Great War?

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The Decline of a Nation?

By the turn of the twentieth century the British nation's declining birth-rate was increasingly the subject of anxious public and scientific debate, as the Registrar General's annual reports continued to confirm a downward national trend, which had in fact commenced from the late 1870s. The secularist Malthusian League had positively promoted birth control, and now economists and eugenicists, feminists and Fabians, as well as leading figures in the church and in the medical profession, all agreed that this was a momentous matter.[1] Previously, human fecundity—the capacity to conceive and reproduce—had not been considered a significant social variable. While the fertility of individuals or couples might be subject to some variation, with the odd exception populations and nations had dependably high fertility.[2] Since Malthus—and even more so since Darwin's generalization of Malthus's proposition to all species—it was an accepted fact that nature was fecund to a fault. Fertility was too robust, not too frail. Consequently, one of the eternal human predicaments, both for the individual and for government, was how to rein in this exuberant fertility. So the dawning perception of the nation's flagging and apparently fragile vitality—and indeed that of

several other urbanizing nations, too—was a serious shock, expressed not just in politics but also science and literature.[3]

The three decades before the outbreak of the Great War therefore saw an intensification of attention to the newly problematized issue of human fertility. Many competing theses and theories were advanced and publicly aired to account for the challenging new phenomenon. The diversity of speculation during these decades was fed by the rapidly changing state of both pure and applied knowledge within the biological and the medical sciences, concerning both evolutionary theory and germ theory.[4] Simultaneously, fraught social and political debates over sex and gender norms were intensifying into the crescendo of the militant suffragette campaign, which also raised the temperature further with the issue of infection of innocent wives by their sexually irresponsible male partners.[5] This intellectual ferment has left a considerable volume of primary source material, which includes several major official inquiries devoted to important aspects of understanding the problem of the falling birthrate in Britain: the Interdepartmental Commission on Physical Deterioration of 1903–4; the Royal Commission on the Care and Control of the Feeble-Minded, 1904–9; the 1911 census's Fertility of Marriage inquiry; and the Royal Commission on Venereal Diseases of 1913–16.[6]

These sources have been used productively by historians for a wide range of studies, including those which have provided accounts of the contested discourses of gender relations, sex, eugenics, and evolutionary theory in relation to nationalist and imperialist ideological and political themes of the period. The empirical studies of fertility and infertility conducted in this period of course all reflected these contemporary agendas. However, the value of the evidence they collected, classified, and presented to make their respective cases, or to confound their intellectual antagonists, is not necessarily exhausted by the terms of reference of those now-defunct discourses. It is also possible, with historicist care and attention both to the scientific context of the period and to the intentions and classificatory designs of the originators of the data, to revisit historical evidence, such as that collected in the early twentieth century, and subject it to a critical secondary analysis in a form that can be used to evaluate a different agenda, informed by our changed scientific and social scientific understandings today. In other words, demonstrating that demographic analysts and epidemiologists of various complexions in the past produced analyses that were ideologically constructed in various ways does not exhaust the potential value to historians of such research conducted in the past. We ignore to our impoverishment the treasure trove of evidence collected by those who demonstrably thought differently from us today. Elsewhere a combination of three such contemporary

sources from the period 1910–12 has been reanalyzed to produce comparative estimates of the absolute prevalence at that time of syphilis infection rates in the national population of England and Wales and in various sections of the populations.[7]

In this chapter we pursue those sexually transmitted infection (STI) prevalence estimates for 1910–12 a little further and link them to the theme of this volume and also to both historical and current debates about the phenomenon of secular decline in the nation's fertility that so perplexed contemporaries, and whose understanding continues to pose a challenging puzzle for historians and social scientists today. In particular we examine how these sources can offer further insights into the possible relationship between STIs and aspects of infertility during this period, which has been somewhat overlooked by demographers ever since. It has been overlooked in part because the discourse of degeneration and disease, within which these contemporary researches were conducted, has been thoroughly—and rightly—deconstructed by intellectual and cultural historians.[8] It has also been overlooked because the possibility of STIs having significant influence on infertility has come to be associated, since the 1920s, exclusively with "other" populations in Oceania, sub-Saharan Africa, and among black Africans in the United States, but not among white Europeans wherever they settled in the world.[9]

To what extent might the imperial white Anglo-Saxon nation itself, including even the metropolitan middle-class professional and administrative elite, have suffered significant infertility due to STIs? The Royal Commission on Divorce, 1909–12, certainly exposed the fact that the guilty secret of middle-class males infecting their wives was in fact a reality for some, as campaigning doctors and feminists had been alleging.[10] However, the increasing proportion of very low-fertility marriages among the middle classes in the late Victorian and Edwardian decades could just as easily have been due to the private wishes of both partners to avoid too great a domestic burden, and it has been impossible so far for historical demographers to tease out the relative importance of these two influences. This is because such private intentions remained at this time unarticulated and inadmissible by the majority. Although birth control had been a subject of public discourse on and off throughout the nineteenth century, it had never achieved respectability in polite society. Throughout the decades of Victoria's long reign—and beyond, into the first two decades of the twentieth century—open acknowledgement of a desire by a couple to restrict their marital fertility continued to be deemed religiously disrespectful by conventional educated opinion, a debilitating problem for public articulation in a culture that remained respectful of religion as an arbiter of moral values. The Malthusian League, campaigning

since the 1870s for public recognition of the rationale for birth control, was, in the eyes of the socially conservative majority, considered synonymous with morally dubious, godless secularists.[11] To want fewer children in a marriage blessed by Christian religion was at best "selfish," at worst hubristic in wishing to thwart the divine maker's plans. The various contraceptive devices required to bring about such plans were viewed by most as distasteful and unnatural and as the accoutrements of the sordid commercial sex trade and of libertines' attempts to avoid disease, not as something appropriate to the marriage bed. Yet it was evident by the 1900s that family sizes had been falling dramatically among the middle classes—and among certain other large social groups too, such as the factory workers in many textiles towns. All the possible suspected reasons for this were viewed as deeply problematic, as far as public moral discourse was concerned.

In many ways the most apocalyptic possibility—a feared general decline in biological fecundity—was most acceptable as a subject for inquiry and public debate because it implied no failings in the personal morality of the unfertile middle classes, and this may in part explain the capacity of the eugenics agenda to take the lead in public debate on the subject in the Edwardian period.[12] Thus, social Darwinist discourse of "degeneration" or "deterioration" licensed earnest and rational discussion, in the interests of science, of the feared possibility that certain worrying and still-unexplained diseases might be causes of the nation's flagging vitality, such as tuberculosis, alcoholism, feeble-mindedness, and of course syphilis (though it took the insistence of feminists to bring gonorrhea also within this sphere of discourse).[13] Eugenicists, who fondly entertained the notion that "inheritance" across the generations was the key to understanding everything, subscribed to the somewhat illogical and self-contradictory notion that any or all of these conditions were probably also heritable and might therefore help explain, with various contortions of reasoning, the decline in fertility of the race. The long-observed phenomenon of supposedly "congenital" syphilis was thought to demonstrate heritability of the disease. The new findings after 1900 that syphilis infection was capable of causing death from "general paralysis of the insane" (GPI) or other conditions into late middle age also seemed to confirm the horrifyingly long reach of the disease, apparently across generational time.[14] Meanwhile, voluntary limitation of family sizes—the other main possible cause of secular fertility decline—was only cautiously discussed in public and usually presented as an undesirable possibility, because few wanted to acknowledge that clandestine religious and moral hypocrisy could be occurring on a potentially widespread scale throughout the nation's upper and middle classes, the supposed moral paragons of the imperial civilizing

nation. However, it was increasingly being seen—in the final few years before the Great War erupted—as an issue at least meriting dispassionate research, hence the government's sanctioning of the enormous nationwide survey represented by the fertility of marriage questions included for every coresident married couple to answer at the 1911 census.

It was the second part of the extensive official report by Dr. T. H. C. Stevenson, the General Register Office's Statistical Superintendent, on the data collected at this census, whose publication was delayed by the war until 1923, that finally seemed to refute conclusively the notion that some kind of biological causes of infertility were at work in reducing the nation's fertility.[15] In his own subsequent interpretative publications, Stevenson emphasized instead the importance of volitional, social, cultural, and economic motives as the causes of declining fertility.[16] All other interwar analysts broadly accepted these conclusions, which then remained the primary and formative focus of all subsequent research, not only on Britain's secular fertility decline but on that of most other populations, too.[17]

The Possible Role of STIs in Childless Marriages in the 1911 Census

It is therefore not surprising that despite all the effort that has been devoted by social scientists and historians over so many decades to elucidating the demographic patterns and diverse possible causes of the historical secular fertility declines that occurred in so many countries during the past two centuries, there has been no critical and systematic evaluation of the possible role of STIs—most notably gonorrhea and chlamydia, which form the multidisciplinary primary focus of this volume, *The Hidden Affliction*.[18] In closing the volume, this chapter attempts to offer a preliminary evaluation of the contribution of these two hidden afflictions to the fertility decline that occurred in England and Wales as recorded in the famous "Fertility of Marriage" Census of England and Wales in 1911. Specifically, this chapter provides a quantitative estimate of the likely effect that STIs had on one component of the emerging pattern of low marital fertility: childless marriages.

It is certainly the case that untreated gonorrhea and chlamydia would also have caused some secondary sterility in marriages after the birth of a first or subsequent child. On the one hand, men and women can—and did—become infected with STIs during, as well as before, marriage. On the other hand, many contemporaries believed that a prior gonorrhea infection could also account for "a one-child sterility," as Prince A. Morrow (professor of

genitourinary diseases at New York University) explained in 1904: "A woman with gonorrhea of the cervix may readily conceive," but the process of parturition at the first pregnancy "opens the gates to the infection which may have been long installed in the external genital canal, the cervix, or uterus, and permits its ascension to the tubes, ovaries, and peritoneum."[19] However, given the intrinsic difficulties in working with the partial historical evidence that is available, the focus here is exclusively on the epidemiologically and demographically most straightforward relationship that can be empirically studied: that between rates of absolute marital sterility (a parity of zero births) of various subgroups of the population and estimates of the relevant age-specific rates of prior infection with STIs among different occupational groups with specified ages at marriage. But it should be borne in mind, that the results reported here represent a proportion, and not the sum total, of the likely impact of STIs in causing involuntary, very low fertility.

The topic of very small families in the British fertility decline before 1914 has rarely been the subject of investigation in historical demographic study. One exception is the work of Michael Anderson, who used tables from the Scottish 1911 census, where some parity distribution information was published, along with later evidence for England and Wales produced by the Royal Commission on Population of 1944–49. Anderson's is an excellent, wide-ranging study; however, it does not at any point discuss the possibility that STIs could be responsible, in part, for the patterns revealed by the occupational fertility data reviewed. This is entirely understandable, in that no robust, empirically based quantitative estimates of the extent of STIs among the British population before 1914 existed for Anderson to draw on. He was consequently constrained to conclude that such extremely low fertility as he found was mainly the product of volition, though he was clearly troubled by the paucity of cultural or literary evidence, pre-1914, which could provide explicit contemporary documentation acknowledging or valorizing this putatively novel form of volitional behavior resulting in "highly restricted" fertility in the upper and middle classes of society in the decades before 1914.[20]

This chapter presents a conservatively constructed model of the extent to which the overall level of STIs prevalent in the population of England and Wales during the decades prior to the 1911 census could have contributed to the degree of absolute childlessness enumerated at that census in marriages of completed fertility. In a previous publication contemporary primary sources from the period just prior to the outbreak of the Great War, notably including two quasi-random samples of the population administered with Wassermann tests, have been subjected to secondary analysis to yield an

estimate that at the time of the 1911 census approximately 7.8 percent of men in England and Wales had incurred an infection with syphilis by the age of 33–35 years old.[21] With the addition of various further considerations, this figure can now be used as the basis to derive an estimate of the extent to which STIs were responsible for the overall proportion of childless marriages recorded at the 1911 census of England and Wales.

It is, of course, gonorrhea that causes sterility, and not syphilis. So it is first necessary to bring forward relevant information that can permit a known rate of prevalence of syphilis to act as a guide for the prevalence of gonorrhea in the population at this time. Second, we need to establish, by consulting the results of relevant modern clinical and epidemiological studies, the quantitative capacity of gonorrhea infections to cause sterility in either women or men. Third, this information needs to be articulated with what we know of the sexual mores and practices of the majority in British society at this time in relation to courtship and marriage so that we can form plausible sex-differential estimates of the opportunity for gonorrhea infections to result in the absolute sterility of marriages. Fourth, the likely contribution to marital sterility of another major STI, chlamydia, also needs to be carefully considered and incorporated into the model.

Since the ultimate objective of this chapter is to form a quantitative evaluation of the extent to which the prevailing incidence of STIs in 1911 was responsible for a proportion of the number of childless marriages enumerated at the census, it is also necessary to adapt the previous work of historical demographers of the early modern English population to produce an estimate of the extent of childlessness that would be expected in this population, regardless of the effect of any STIs. To produce such an estimate, results from the Cambridge Group for the History of Population's parish reconstitution data are used, with a correction factor added to take into account a newly published, empirically based figure to adjust for the extent to which those parish populations were themselves likely to have been subject to a moderate level of fertility-reducing STIs.[22]

Once the available evidence for all these components of the required model has been presented, weighed, and considered, the calculations can then be combined to offer an empirically based best estimate of the extent to which STIs accounted for a proportion of the completely childless marriages enumerated at the 1911 census. The proportion turns out to be far from insignificant.

A Model of the Impact of STIs on the Infertility
of Marriages in 1911

In this section we first construct a model for male sterility due to gonor-rhea, before producing an estimate for female sterility. Previous published work has established that in 1911 men in England and Wales (married and unmarried combined) accumulated approximately a 7.8 percent chance of having been infected with syphilis by the time they reached age thirty-three to thirty-five. If we make the simplifying assumption that for most males the chance of becoming infected in this era started after the teenage years, at age twenty, and was roughly equal per year, then we can estimate that 0.52 percent of new men (one in two hundred) were infected with syphilis at each year of age between twenty and age thirty-four inclusively.[23]

While syphilis infection has no direct implications for sterility, a robust indicator of its prevalence, such as this, can be useful to derive an estimate of the extent to which men were at this time infected with gonorrhea, the primary STI causing sterility. Gonorrhea (and chlamydia) are significantly more infectious (and reinfectious) than syphilis; this is partly because the spirochete is much more fragile and must access the bloodstream for trans-mission and also because a single inoculation with syphilis usually produces immunity to subsequent infection, which is not true of either gonorrhea or chlamydia. The key issue therefore is to provide a plausible estimate of how much more prevalent was infection with gonorrhea than with syphilis at this time in Britain. There is relevant contemporary evidence for the population of Sweden, where both syphilis and gonorrhea were compulsorily notified. This Swedish data for the two most comparable years, 1918 and 1919, indi-cates that the prevalence of gonorrhea among men was approximately four times greater than syphilis.[24]

If we know that the rate of infection with gonorrhea is likely to have been four times as great, this implies that approximately 2.08 percent of men (one in fifty) were infected with gonorrhea at each individual year of age from twenty to thirty-four (assuming an approximately equal risk of exposure over this age range). That in turn means that by age twenty-four 10.4 percent had been infected with gonorrhea at some point in their lives; by age twenty-nine the figure was 20.8 percent, and by age thirty-four the figure was 31.2 per-cent. Making the deliberately conservative and simplifying assumption that the chance of further infection of either partner more or less stopped around the point of marriage, this means that the chances of ever having had an infection with gonorrhea among men marrying at age twenty to twenty-four would have been about 5.2 percent (half of 10.4 percent, assuming an even

distribution of marriage ages across this age range).[25] However, among those marrying at the more typical ages of twenty-five to twenty-nine, 15.6 percent would have had a gonorrheal infection at some point in their lives by the time they married (adding together the annual chance in all of the five years among those aged twenty to twenty-four, plus half of the five years between ages twenty-five to twenty-nine, again assuming an equal distribution of men marrying across each of those five years). Applying a similar logic produces a figure of 26.0 percent having had a prior infection among those males marrying at ages thirty to thirty-four.[26]

We now need to multiply these figures, for the rising proportion of males having had an infection at each marriage age, by an estimate of the chance of a gonorrhea infection causing male sterility. This happens through the complication of epididymitis (see figure 10.3, page 320) and, less frequently, prostatitis and secretory gland involvement.[27] About one-sixth (17 percent) of untreated cases of gonorrhea in men lead to epididymitis and, in turn, estimates vary between 23 and 41 percent of such individuals being rendered permanently sterile.[28] If we adhere to a conservative estimate of 29 percent, which is at the lower end of this range (being twice as near to the lower figure of 23 percent as to the upper figure of 41 percent), this would indicate that about 4.9 percent (just under one in twenty) of those males infected with untreated gonorrhea would be sterilized in consequence.[29]

To calculate the proportion of males marrying at ages twenty to twenty-four who were sterile at the beginning of their marriages due to gonorrhea we should multiply successively by 17 percent and by 29 percent the estimate that 5.2 percent of men aged twenty to twenty-four at marriage would have encountered an infection with gonorrhea before (or in the early stages of) marriage. This equates to 0.256 percent for men marrying at age twenty to twenty-four (5.2 percent × 0.0493, the product of 0.17 × 0.29). The comparable figure for men marrying at twenty-five to twenty-nine is 0.774 percent (equivalent to [10.4 + 5.2] × 0.0493). The comparable figure for men marrying at thirty to thirty-four is 1.282 percent (equivalent to [20.8 + 5.2] × 0.0493).

To these figures for the rising proportion of male STI sterility with later male age at marriage has to be added the chance at each female age at marriage of female sterility due to gonorrhea. Modern research has found that in a population of women generally experiencing repeated chances of conception and childbearing (i.e., a population in which it was the social norm for married women to experience more than one live birth), about 30 percent of those with gonorrhea usually progress to pelvic inflammatory disease (PID).[30] Furthermore, among those women contracting PID, if untreated for three

days (as would have been the case for all women in Britain before 1914, in that even if they did come immediately under medical care—itself unlikely—there was no effective treatment to deploy), there is a further 30 percent chance that the occurrence of PID will result in TFI (tubal factor infertility, see above, figures 7.2, page 231, and 10.4, page 231).[31] This implies that one in eleven (9.09 percent) of all women who originally became infected with gonorrhea at any point before the development of effective treatments from the late 1930s onward were likely to have become sterile due to PID.

This estimate of STI impact on female sterility has to be further adjusted to take into account the additional effect of chlamydia. It is not justifiable to discount entirely the effects of chlamydia in the pre-1914 decades simply because of the unavailability of positive evidence for a disease that was at that time unknown. As the contribution to this volume by Ian N. Clarke and Hugh R. Taylor shows, it is inconceivable that the disease was not present, and, equally, as Michael Worboys's chapter carefully documents, a range of nonspecific, non-gonorrheal conditions were recognized by clinicians during the early twentieth century, though they did not identify them as what is now called chlamydia.[32] Untreated chlamydia, like gonorrhea, has a definite capacity to cause female sterility through PID and consequent tubal blockage in a proportion of those affected, as well as leading to some ectopic pregnancies. It is also known that *C. trachomatis* causes urethritis and epididymitis in males. However, the precise mechanisms through which chlamydia infections can produce infertility consequences appear to be extremely complex and variable, and the field is currently a highly dynamic one of ongoing research.[33] Early scares of chlamydia's gross sterilizing effects were clearly exaggerated, as pointed out in Worboys's chapter. Nevertheless, there is unanimity that untreated chlamydia is a cause of a nontrivial amount of infertility in women.[34] Given all the relevant considerations discussed in detail in appendix E, the proposal adopted here is to increase any estimate of the effect due to gonorrhea on female sterility by one-third, to take into account the likely scale of the additional effect of a known but unquantifiable presence of chlamydia in the pre-1914 population. In keeping with the conservative principles of estimation adopted throughout and consistent with the present state of knowledge, no additional sterility effect on male fertility will be attributed here to chlamydia.[35]

Thus, the chance of female sterility from any single episode of infection with gonorrhea is significantly higher than the male chance (one in eleven as against a one in twenty male chance), and there is also the added risk that female sterility can be caused by chlamydia infection (which we are assuming, following the latest scientific consensus, is unlikely to cause

male infertility). However, assuming that most married women were not exposed to the chance of infection prior to marriage (except with their future spouse) or outside marriage, a major difference between the sexes is that whereas males may enter marriage already sterile from a premarriage infection with gonorrhea—and their chances of this being the case rise the later they marry—the female chances of becoming sterile due to exposure to STI infection (from her marriage partner) are invariant with respect to her age at marriage. In these circumstances, because they are a function only of her exposure just before or during the first year or so of her marriage regardless of her age, the probability of a wife being unable to conceive a first child would always relate to the chances of her male partner having acquired a transmissible infectious condition (of either gonorrhea or chlamydia) no more than six to twelve months before initiating intercourse with his spouse, or from him having acquired the infection from extramarital intercourse within six to twelve months of the marriage commencing. If acquired any later in the marriage, though its transmission to the wife might have curtailed subsequent fertility, it would have been unlikely to have resulted in her failure to conceive a first child and her complete childlessness.[36] The female chance of infection at any particular age at marriage is therefore set as being equal to approximately two years' worth of the chance of their husband having acquired either a gonorrheal or chlamydial infection in the period immediately before or during the early months of marriage.

The chance of female sterility at any age is therefore 1.04 percent (two years' worth of the male annual chance of gonorrhea infection) multiplied by 0.1212 (to reflect both the 0.909—one in eleven—chance of female sterility, inflated by a factor of one-third to take into account the additional sterilizing effect of chlamydia). This produces a value of 0.126 percent representing the chances of women at any single year of age being infected, which therefore needs to be multiplied by five to reflect the chance of sterility among a five-year age group of women, such as those marrying at twenty to twenty-four, twenty-five to twenty-nine, and thirty to thirty-four. This produces a final female sterility estimate of 0.63 percent for each age-at-marriage quinquennium grouping of married women.

Assuming that in the generality of the population of England and Wales, males and females were more or less of the same age as each other at marriage (males typically in fact were a couple of years older at marriage, which simply has the effect of again making the following estimated figures conservative or minimal estimates of the role of STIs), the overall chance of marital sterility having been caused by the prevailing rates of STIs in 1911, among both husband and wife combined, are as follows for different female ages at marriage:

20–24: 0.886 percent
25–29: 1.399 percent
30–34: 1.912 percent[37]

The reason these figures rise with age at marriage is primarily a function of the accumulating chance of male sterility. Although most doctors in Britain before the Great War were apparently quite unaware of the substantial male role in marital sterility, as Christina Benninghaus documents in chapter 10, this would have been no surprise at all to the medical community—or even to the wider, educated public—in Germany. There, several influential clinical studies incorporating sperm testing had shown about one-third or more of sterile marriages to be due to male infertility.[38]

These figures can now be compared with the actual proportions of marriages sterile (i.e., childless) at these ages in the population of England and Wales reported at the 1911 census (table 12.1, row 3), being, respectively, as follows: 6.0 percent; 11.8 percent, and 22.6 percent. However, when comparing the two sets of figures, it must be borne in mind that a very significant proportion of the latter was due to other natural causes not attributable either to STIs or to birth control, and this has to be subtracted before the truly additive effect of the prevailing rates of STIs in 1911 can be quantified.

The rates of childlessness in 1911 due solely to natural variability in fecundity and other natural sources of infertility can be estimated by comparison with a historical reference standard provided by the Family Reconstitution Files of the parishes collated by the Cambridge Group, as analyzed by James Trussel and Christopher Wilson (see appendix A on the choice of Trussel and Wilson's methodology for the analysis of sterility used in this chapter).[39] However, the rates of sterility found by Trussel and Wilson are themselves not entirely free from the effects of STIs, since it is unlikely to have been the case that these English rural and small-town parish populations were entirely immune from STIs in the period circa 1600–1800. Allowance can be made for this because there now exists an empirically based quantitative estimate, published in 2017, of the likely prevalence of syphilis in rural England and Wales in the 1770s. This estimate has been constructed in such a way as to be demographically comparable with the published figures available for the population of England and Wales in the period 1910–12. This study found that in the mid-1770s in rural Cheshire and North Wales 0.93 percent of both sexes had sought treatment for the pox by age thirty-five (as against about 8 percent of the city residents of Chester).[40] Most of the sixteen parish populations in the pre-1850 set of family reconstitutions analyzed by Trussel and Wilson related to small rural settlements of the kind found in the agricultural

region of west Cheshire and northeast Wales surrounding Chester. The two exceptions are the market towns of Banbury and Gainsborough, though both of these were very much smaller than Chester in this period.[41] That would suggest that the parity patterns found in most of the pre-1850 parish populations analyzed by Trussel and Wilson most probably reflected a very moderate prevalence of STIs, similar to the low rate of 0.93 percent rate for the pox empirically established for rural Cheshire and North Wales.

There needs to be some further adjustment to take into account the higher incidence of STIs likely to have been found in the townships of Banbury and Gainsborough. Allowance can be made for this, first, by deriving an empirical estimate from the raw data of the Cambridge Group's Family Reconstitution Files (FRF) of how much greater was the age-adjusted prevalence of sterile marriages in Banbury and Gainsborough than in the aggregate of more rural parishes, given that the primary cause of such excess sterility (once age at marriage has been controlled for) is most likely to have been the differential incidence of STIs (there is no evidence for volitional birth control in this period).[42] Second, the two different subpopulations can be weighted by an estimate of the relative size of Banbury and Gainsborough against the other fourteen parishes in the Trussel and Wilson group of sixteen parishes.

This reanalysis of the FRF data using all marriages of completed fertility (i.e., corresponding to the Trussel and Wilson methodology) found that across all marriages where wives were aged fifteen to thirty-four at marriage (and weighted for the different numbers of couples in each quinquennium of marriage age), those in Banbury and Gainsborough were 1.387 times more likely to be sterile than those in the other fourteen smaller parishes, combined.[43] That tendency to a higher rate of sterility then needs to be weighted by the relative population size of these two parishes. The earliest reliable estimate of the relative sizes of all sixteen parish populations shows Banbury and Gainsborough with a combined size of 8,922, while the other fourteen parishes totaled 20,427.[44] Therefore, to allow fully both for the higher STI prevalence in the more urban pair of parishes and their proportionate size among the sixteen parishes, the figure of 0.93 percent pox prevalence for entirely rural parishes (derived from Cheshire) needs to be adjusted upward by a factor of 0.424.[45] This results in an estimate that the comparable pox-prevalence rate (by age thirty-five) in all sixteen parishes, combined, would have been 1.324 percent (0.93 + [0.93 × 0.424]). This double-corrected estimate takes into account the fact that the more urban parishes of Banbury and Gainsborough had higher rates of STI infection than the other rural parishes, and their relative population weighting within the total of all sixteen parishes in the Trussel and Wilson data.

Following the method detailed in appendix B for adjusting the parish populations to take account of STIs in affecting a proportion of marriages recording no live births, table 12.1 provides a summary of the key steps in this process. Row 1 reproduces the Trussel and Wilson figures for sterility (proportion of marriages with parity zero) at the three main female ages at marriage in their original unadjusted form. These are then adjusted for STIs in row 2, by modifying the estimated prevalence of STIs as indicated by the 0.93 percent rates for rural Cheshire and North Wales, further modified to a rate of 1.324 percent to reflect the most probable evidence-based estimate for these sixteen parishes. Thus, in effect, row 2 equates to a pre-1800 English population entirely free from STIs.

Rows 3–5 of table 12.1 then provide the proportion of sterile marriages derived from the I-CeM database version of the 1911 census for England and Wales, together with the underlying total number of couples these figures are based on (for details on I-CeM see the next section of this chapter and note 48). Following these, rows 6 and 7 indicate the percentage by which the proportion of sterile marriages observed for 1911 are in excess of, first, the Trussel and Wilson unadjusted rate for pre-1850 England (row 6), and, second, the Trussel and Wilson rate adjusted for STIs (row 7). Subtracting the Trussel and Wilson STI-adjusted rate (row 2) from the rates of sterility observed in 1911 at the census of England and Wales (row 3) provides the set of "excess" sterility rate figures given in row 8.

The figures in row 8 indicate the amount of childlessness at each of these three different female ages at marriage in the population of England and Wales at the 1911 census that is in excess of that which can be explained by the amounts of such sterility found in the FRF rural parish populations, adjusted to be free from STI effects (row 2). These, then, are the quantities of childlessness in 1911 that remain "unexplained" by any base rate of "natural" infertility and that are therefore due either to the prevailing STI rates or to voluntary restraint of fertility within marriage. We have no direct, quantifiable evidence on the latter, but we do have for the former, and so the approximate contributions of each can then be gauged.

This is done by bringing together these estimates of the amount of "excess" sterility in different female age-at-marriage groups shown in row 8, with the estimates, calculated previously (see top of page 384), for the proportions rendered sterile by the prevailing STI rates in the population of England and Wales in 1911, which are shown in row 9. Making, again, the conservative, minimizing assumption that males and females were more or less of the same age as each other at marriage, the bottom row (10) of table 12.1 shows the percentage of sterile marriages in 1911 most probably due to the effects of

Table 12.1 Marriages of completed fertility in England and Wales at 1911 census: estimates of proportion of childless marriages due to STIs. Female marriage ages: 20–24 (married 25–29 years); 25–29 (married 20–24 years); 30–34 (married 15–19 years).

Row		Age at marriage		
		20–24	25–29	30–34
1	Trussel and Wilson's pre-1850 unadjusted % zero parity	4.6	9.1	16.6
2	Trussel and Wilson's pre-1850 STI-adjusted % zero parity	4.3	8.4	15.8
3	England and Wales, 1911, % zero parity	6.0	11.8	22.6
4	England and Wales, 1911, total number of couples	236,815	149,338	63,154
5	England and Wales, 1911, total number of couples with zero parity	14,273	17,650	14,260
6	England and Wales, 1911, proportionate excess over Trussel and Wilson's unadjusted % zero parity ([row 3 minus row 1] divided by row 1)	30.4%	29.7%	36.1%
7	England and Wales, 1911, proportionate excess over Trussel and Wilson's STI-adjusted % zero parity ([row 3 – minus row 2] divided by row 2)	40.0%	39.8%	43.1%
8	England and Wales, 1911, % "excess" marital sterility (zero parity): row 3 minus row 2	1.7	3.4	6.8
9	England and Wales, 1911, sterility (% zero parity) attributable to STIs in 1911 population	0.886	1.399	1.912
10	England and Wales, 1911, proportion of "unexplained" childless marriages due to STIs (row 9 divided by row 8)	52.1%	41.1%	28.1%

Note: Rows 1, 2, 3, 8, and 9 report arithmetically comparable percentage figures; rows 6, 7, and 10 present derivative figures for analytic purposes discussed in the text. For use of Trussel and Wilson sterility estimates, see appendix A, and for the derivation of row 2 from row 1, see appendix B. Source for row 3: I-CeM. The Integrated Census Microdata Project.

STIs. These are as high as 52.1 percent at the younger-than-average female age at marriage of twenty to twenty-four, and 41.1 percent when wives were aged twenty-five to twenty-nine at marriage (the average female age at marriage in 1911 was twenty-six years[46]), falling to 28.1 percent among wives aged thirty to thirty-four at marriage.

Overall, taking into account the different numbers of couples marrying at these different ages (row 4), the conclusions that follow from this exercise are that STIs were most probably responsible for about 45 percent of all excess childless marriages at this time, after allowing for those attributable to natural variability in human fertility.[47] On the one hand, therefore, these calculations show that it is probable STIs did play a substantial role in accounting for childless marriages at the 1911 census. On the other hand, it can be argued that this exercise nevertheless confirms that voluntary restraint was apparently responsible for somewhat over half the overall excess in childless marriages and for slightly over two-thirds among that minority of society delaying marriage the most, where the wife was aged thirty or above at marriage.

Childlessness and STIs among the Professional Middle Classes in 1911

To pursue this issue a little further, it is possible to examine in somewhat more detail a selection of marriages recorded at the 1911 census drawn from the professional upper and middle classes. This is the section of society that tended to delay marriage the most. It is also predominantly from this section of society that the variety of contemporary views on the nation's infertility, mentioned at the beginning of this chapter, emanated. These views included the concern that venereal diseases could be significantly implicated in the falling fertility of the nation and, according to some feminists and medical observers, that this was the case even in relation to the low fertility of their own privileged section of the nation. Can we now begin to quantify how much truth there may have been in this?

The official published reports on fertility emanating from the 1911 census tabulated an interesting range of information, which has for instance made possible the analysis of national average fertility rates of male occupations. However, the individual-level records that underlie the published tables have only since 2011 been digitized and made available for research in the form of the I-CeM database.[48] As a result, detailed parity-specific fertility information from the 1911 census can now for the first time be calculated for individual male occupations. Moreover, this occupation-specific fertility information

derived from the I-CeM data can also be analyzed alongside a near-contemporary set of statistics on the prevalence of syphilis-related causes of death: namely part 4 of the decennial supplement to the seventy-fifth *Annual Report of the Registrar General* (*ARRG*). This official publication tabulated for a range of selected occupations the extent (age-standardized) to which men aged twenty-five to sixty-five died during the years 1910–12 from various causes of death, including the three most closely associated with a previous infection with syphilis: general paralysis of the insane (GPI), locomotor ataxy, and aneurysm.[49] Combining the two contemporaneous official sources, the 1911 census and this information from the death registers, enables the propensity to die from these three "parasyphilitic" causes of death to be directly related to patterns of childlessness for certain occupational groups.

In his presentation to the Royal Commission on Venereal Diseases (1913–16), drawing on the occupation-specific mortality figures later published in the seventy-fifth *ARRG*, T. H. C. Stevenson showed that the mostly professional males who composed Social Class I of the new official social classification scheme (which he devised for application to the 1911 fertility census[50]) died as a result of these syphilitic causes of death at a rate higher than the national average. Indeed, only the unskilled laboring class (Social Class V), at the other end of the social spectrum, recorded a significantly higher rate.[51]

A small set of five professional middle-class occupations identifiable in both the 1911 I-CeM census data and the tables of the seventy-fifth *ARRG* have been selected for further analysis here: clergy, barristers and solicitors (often combined as "lawyers" in the ensuing analysis because of the relatively small numbers of barristers), medical practitioners, and bankers.[52] These were chosen for their typicality as professions and for the variability they display in their recorded parasyphilitic causes of death (for further details on the provenance and construction of the professional groups, see appendix C).

Combining the available data from the seventy-fifth *ARRG*, Stevenson's evidence to the Royal Commission on Venereal Diseases 1913–16 (which also included deaths from "Syphilis" itself) and the estimates presented earlier in this chapter, table 12.2 shows the imputed chances of contracting gonorrhea by age thirty-five among the males of the four listed professional categories. The principal "translation" device here is that the absolute figure of 8.3 percent at the head of the second column, expressing the chance of contracting syphilis by age thirty-five (which is the previously published estimate by Szreter for Social Class I) and the ratio relationship between each of the figures for each individual occupation and the figure for Social Class I at the head of the first column, permits each of the occupations to be assigned a corresponding absolute value in the second column, bearing the same ratio

relationship to the figure of 8.3 percent at the head of the second column as its corresponding figure in the first column bears to the figure of 26 at its head.[53] Table 12.2 demonstrates that Registrar General's Social Class I was far from being a homogeneous group in terms of the prevalence of STIs. While lawyers and bankers recorded levels substantially above the average for the class as a whole, the rate for physicians was around half of their rates and the rate for clergy, as might be expected, was significantly lower than the other selected occupations.

Table 12.2. Occupation-specific estimates of contracting gonorrhea

	Age-standardized mortality rate from parasyphilitic causes of death	Accumulative chance of contracting syphilis by age 35 (%)	Accumulative chance of contracting gonorrhea by age 35 (%)
Social Class I	26	8.3	33.2
Lawyers	31	9.9	39.5
Clergy	6	1.9	7.7
Physicians	16	5.1	20.4
Bankers	33	10.5	42.1

Note: The occupational specific figures cited in the first column are taken from column 11 of the main table of the supplement to the seventy-fifth *ARRG*: Registrar General, 1901–12, England and Wales Supplement to Registrar-General's Seventy-Fifth Annual Report: Part IV; Mortality of Men in Certain Occupations in the Three Years, 1910, 1911 and 1912, *Online Historical Population Reports*, 1901–12, accessed February 22, 2019, www.histpop.org, pp. 2–96. They are age-standardized rates of mortality that accurately express the relative extent to which different male occupational categories of the population died from the three parasyphilitic causes of death in the age range of 25–65 years old during the years 1910–12. The figures in themselves represent the number of deaths from these three causes combined that would have occurred if the occupational death rates recorded in 1910–12 had been operating on a sample of the general male population enumerated at the 1901 census, which had a particular age structure. The reason for standardizing on the 1901 age structure and not that of 1911 was that Stevenson was using this exercise to also compare occupational rates between 1900–1902 and 1910–12. It does not affect the validity of the comparative measures cited here for 1910–12, so long as they have all been standardized against the same population age structure. Using lawyers as an example, the figures for individual occupations in the second column of this table are calculated with reference to the figure of 8.3% at the top of the second column (for social class I as a whole) as follows: $8.3 \times (31 / 26)$, producing the figure of 9.9% in this case. This is then multiplied by 4, reflecting the greater infectiousness of gonorrhea, to produce the figure of 33.2% in the third column. In contrast to the figures given in the first column, which are relative ratio figures, those in the two other columns are absolute measures.

Having established the estimated rates of contracting gonorrhea for each of the selected middle-class occupations shown in table 12.2, we can now turn to the proportion of marriages for each occupation producing zero live births to evaluate the evidence for a relationship between the two. However, before proceeding to that aspect of the analysis, we should first consider whether, or to what extent, there is evidence for an association between the chances by age thirty-five of each occupational category accumulating STI infections and the proportions of men remaining unmarried in their twenties and thirties in each occupation. Such a relationship would imply that the higher STI rates disproportionately afflicted the unmarried within each occupation and could not therefore be adduced as a necessary influence on marital sterility. That consideration is somewhat protracted and therefore is undertaken in appendix D, which finds no consistent evidence to support the hypothesis that STI infections before age thirty-five were confined disproportionately to the never-married sections of each of the professional occupations studied here.

Table 12.3 therefore shows for each of the selected professional occupations the proportion of marriages with no live births, by age at marriage of the wife (same marriage durations as table 12.1), relative to the comparable national figures for England and Wales in 1911; and the excess over the Trussel and Wilson adjusted (STI-free) estimates of sterility for English FRF parish populations as presented in table 12.1. Among the four categories of selected professional occupations, table 12.3 shows that the excess childlessness was over 50 percent greater than the national average of 39.8 percent among the professionals' modal female age-at-marriage group (wives marrying aged twenty-five to twenty-nine) and nearly twice as high specifically among the lawyers. The effect was even more pronounced where marriages had been contracted with relatively younger brides (aged twenty to twenty-four), and again this was marked particularly among lawyers. The excess was more muted—and little different from the national average—for marriages where the bride was relatively older (aged thirty to thirty-four). Overall, these findings confirm that the much greater proportions of childless marriages found in professional marriages indicate a more pronounced role for volitional causes than was the case in the general population. However, by contrast in the case of bankers marrying wives aged 30–34 very few marriages were childless because of birth control. Row 7A shows that almost three-quarters of the excess sterility among this older-marrying group was due to STIs.

Indeed, we know from table 12.2 that some of these professional occupations, notably lawyers and bankers, exhibited an incidence of parasyphilitic mortality about 25 percent higher than the national average, while physicians

Table 12.3. Sterility estimates, percentage excess over Trussel and Wilson's figures (in pre-1850 English parish populations) and proportion due to STIs in selected professional occupations by age of wife at marriage (completed marriages), 1911.

Row		Age at marriage		
		20–24	**25–29**	**30–34**
1	Clergy (% childless marriages)	9.1	13.6	22.2
2	Physicians (% childless marriages)	9.3	13.7	22.9
3	Lawyers (% childless marriages)	10.7	15.6	23.9
4	Bankers (% childless marriages)	7.8	13.5	18.2
	England and Wales 1911 % excess over Trussel and Wilson's STI-adjusted % zero parity (table 12.1, row 2)	40.0	39.8	43.1
5	Clergy: % excess over T&W STI-adjusted % zero	112.3	61.1	40.6
5A	Proportion of excess due to STIs	3.9%	3.6%	4.9%
6	Physicians: % excess over T&W STI-adjusted % zero	116.9	62.3	45.0
6A	Proportion of excess due to STIs	9.9%	9.4%	11.7%
7	Lawyers: % excess over T&W STI-adjusted % zero	149.6	84.8	51.4
7A	Proportion of excess due to STIs	14.9%	13.4%	19.9%
8	Bankers: % excess over T&W STI-adjusted % zero	81.9	60.0	15.3
8A	Proportion of excess due to STIs	29.1%	20.2%	71.6%

Source: The Integrated Census Microdata Project (I-CeM), http://doi.org/10.5255/UKDA-SN-7481-1

Note: England and Wales figures (between rows 4 and 5) taken from table 12.1, row 7. Figures in rows 5, 6, 7, 8 express rows 1–4 as excess values over those of table 12.1, row 2.

were roughly a third below this average, and clergy were way below the national average—by more than three-quarters. This points to very different sources of marital infertility among these professional occupations. Rows 5A, 6A, 7A, and 8A of table 12.3 show the proportion of sterile marriages in each five-year female age at marriage group attributable to each occupation's STI rates (shown in the third column of table 12.2). These estimates were calculated utilizing exactly the same method described above in detail on pages 380–87, which ultimately produced the figures in row 10 of table 12.3, but, of course, now using the relevant, occupation-specific STI figures. On the one hand, it seems that almost all of the high level of "excess sterility" among religious ministers and their wives was due only to deliberate fertility-restricting behavior (even though the clergy didn't marry quite so late as the other three professions). Among bankers in particular, although

they tended to exhibit somewhat fewer sterile marriages than the other three professional categories, a larger proportion of the amount of childlessness that they did experience was due to the relatively high susceptibility of their occupation to STIs. Table 12.2 indicates that lawyers also contracted STIs at almost the same, relatively high, rates as bankers. But table 12.3 shows that, in addition to this, lawyers must also have practiced a much higher degree of volitional birth control than bankers, resulting in their significantly lower figures in row 7A, compared with row 8A.

Thus, the implications of the analysis in this chapter are thought-provoking in terms of wider theories concerning the springs and motivations of family limitation in this period. Table 12.3 indicates that the country's leaders of religious faith, widely regarded as the most stalwart objectors to atheistic contraptions of birth control, may have been among the most thoroughgoing family planners in the entire populace at this time. Putting these two characteristics together suggests a strong role for abstinence, especially in this section of society. On the other hand, very low fertility in the banking profession, supposed bastion of secular prudence, was due less to deliberate birth control and more to the risky behavior that resulted in STIs, particularly among those supposedly the most "prudential," who postponed marriage for longest.

The Hidden Affliction and the Quiet Revolution of Secular Fertility Decline

This exercise in the historical reconstruction of the scale of the hidden affliction of infertility due to venereal diseases in pre–Great War Britain has demonstrated the need for the impact of STIs to be taken into account when attempting to understand the scale and the incidence of extremely restricted fertility among married couples in the late nineteenth and early twentieth century. Table 12.1, row 10, indicates that among the vast majority of the population who married at one of the two most common female ages at marriage of twenty to twenty-four and twenty-five to twenty-nine years of age, STIs were in fact potentially responsible for over 45 percent of the excess number of childless marriages (relative to a population with no STIs). If, however, we focus on that section of the general population delaying marriage to a most unusual extent, where wives were aged thirty to thirty-four at marriage, STIs were responsible for a lower proportion of just over a quarter of the excess childless marriages, despite the fact that in absolute terms the chances of husbands entering marriage sterile, due to a previous infection

with gonorrhea, were higher at this age than when they married younger. This study has confirmed therefore that the major role in the fertility decline was played by increasing volitional birth-controlling behavior. However, it is of importance to observe that, apart from the notable exception of bankers, such volitional control seems to have been generally positively correlated with delayed marriage as a closely associated form of behavior, which, as has been previously argued, indicates the likely importance of a regime of attempted abstinence from sexual intercourse within marriage, rather than the employment of contraceptive devices.[54]

Another conclusion from this exercise is that students of Britain's modern historical fertility decline need to pay far greater attention to involuntary sterility as a potentially contributory factor. The secular decline in national fertility rates has been referred to before as the silent or quiet revolution, in part signifying the acknowledged difficulty all students of the phenomenon have encountered when trying to offer convincing accounts for its causation that fully engage with all the evidence of social and geographic diversity.[55] It seems that one part of our collective difficulties may be due to the substantial but diverse role played by another dimension of historical silence—the "hidden affliction" of this book's title. Furthermore, the focus here, for simplicity's sake, has been only on the proportion of childlessness—zero parity marriages as reported at the 1911 census—that can be attributed to this form of involuntary infertility. In view of the phenomenon of "one-child sterility," particularly in relation to postpartum puerperal fever (which Irvine Loudon has found to have been particularly a problem afflicting upper- and middle-class women, who were disproportionately subject at their first births to the attendance of medical professionals lacking in proper antisepsis procedures), it seems likely that a considerable proportion of wives of professional men reporting having had a single birth, only, in 1911 would also be reporting the consequences of involuntary, rather than necessarily volitional, infertility.[56]

There is no reason to think that the importance of involuntary sources of infertility would only have been a feature of Britain's fertility decline, as the chapters in this volume on France and Germany in this period confirm. Though, as shown here, it is difficult rigorously and quantitatively to demonstrate the likely scale of effects involved, at least one French demographer has considered STIs and their infertility effects to have likely been of significance in accounting in part for France's low fertility throughout the modern period until the arrival of antibiotics in the 1940s.[57]

For the past one hundred years or so, studies by demographers and historians have almost completely discounted something that certain contemporaries, themselves drawn mostly from the professional middle classes,

drew attention to and so feared. In their heightened state of concern, some contemporary feminists and medical specialists in the first two decades of the twentieth century undoubtedly overestimated the scale of effects due to syphilis and gonorrhea. However, the evidence presented in this chapter indicates that STIs—notably gonorrhea and chlamydia—probably did play a significant role in the secular fertility decline. This needs to be fully acknowledged and reintegrated into our efforts to understand this epochal transformation in the reproductive beliefs and behaviors of British society.

Appendixes

Appendix A: Trussel and Wilson's and E. A. Wrigley and Colleagues' Estimates of Sterility

There exist two different variants of published estimates of the sterility rates that can be derived from the FRFs of parish populations held by the Cambridge Group for the History of Population and Social Structure.[58] The difference between the two estimates is due to three independent sources of variation in the way they were each calculated, the first two of which have substantive effects. The Trussel and Wilson estimate was drawn from a different permutation of parishes from those used by Wrigley and colleagues, notably excluding the township of Birstall (over twice as large by 1789 as the next two largest parishes in the sample: Gainsborough and Banbury). Birstall was used in the more complex chronological grouping system by Wrigley and colleagues for the period 1600–1789. This is relevant, as it is the only parish that was both urban and fast growing across the eighteenth century, both factors likely to have raised the propensity of its population to suffer from STIs, relative to smaller and more stable rural or market-town communities.[59] Second, Trussel and Wilson based their calculations on all marriages, whereas Wrigley and colleagues based theirs on first-time marriages for both partners. Third, Trussel and Wilson experimented with a weighting device for allocating marriage ages within each quinquennium age of wife at marriage grouping—though they concluded from this exercise that "substantive results are unlikely to be affected."[60]

Despite the inclusion of Birstall, which might be supposed to inflate the proportion of sterile marriages in the Wrigley and colleagues series—if it is likely to have been a parish more prone to STIs than any other—it is the Trussel and Wilson estimates that produce significantly higher (by about

20 percent) proportions of sterility at all female ages at marriage, except the very oldest. This suggests that Trussel and Wilson's inclusion of all marriages may have admixed a certain amount of secondary sterility into their samples through the inclusion of widowers and, especially, widows (divorce in this period was virtually unknown outside the aristocracy), who may have already experienced pregnancy in previous marriages but who had since become sterile. This aspect of their calculations is fully acknowledged by Trussel and Wilson, and it was part of the aim of the subsequent work by Wrigley and colleagues, *English Population History from Family Reconstitution, 1580–1837*, to eliminate this influence.[61]

In a sense, the difference between the two sets of figures represents a more "pure" estimate of age-related biological sterility of couples in the case of Wrigley and colleagues' *English Population* estimates, whereas the Trussel and Wilson figures represent a schedule that includes the normal admixture of second and third marriages due to death or marital separation and remarriage that would be found in an actual population of the sort surveyed at the 1911 census, where the fertility questions related to the wife's current marriage, not to any previous marriages.

It has been concluded that, *provided* the combined effects of rates of spouse bereavement and marital separation were not dramatically different between those marrying in the seventeenth and eighteenth centuries and those marrying in the 1870s and 1880s, it is most justifiable to adopt the higher values of the schedule of rates of sterility published by Trussel and Wilson rather than the slightly lower "pure" estimates produced by Wrigley and colleagues, as being most appropriate for the specific comparative purposes of the exercise being conducted at this point in this chapter. This is to establish how much of the sterility found at the 1911 census among those marrying in the 1870s and 1880s can be attributed to voluntary limitation or to STIs. The conditions of this *proviso* seem to be met, in that while rates of spouse bereavement by 1911 would have been slightly lower for those marrying in the late Victorian decades (life expectancies were slightly higher on average than in the seventeenth and eighteenth centuries), this would have been offset by the fact that rates of marital separation were probably somewhat higher in the decades prior to 1911. Divorce remained rare, but from 1878 the Matrimonial Causes Act permitted magistrates courts to issue maintenance and separation orders, and by the 1900s there were about ten thousand per annum issued.[62]

Appendix B: Adjustments to the Trussel and Wilson Estimates of Sterility to Allow for the Influence of STIs

The likely scale of the contribution of STIs to the sterility rates found in the pre-1850 parish populations analyzed by Trussel and Wilson can be calculated as a derivative from the empirically based estimate that about 1.324 percent of the population of both sexes had probably been infected by syphilis by age thirty-five. There is direct evidence from the 1770s Chester Infirmary records that the two sexes were infected with pox at approximately equal rates, and this gender equality of incidence is broadly borne out also by Kevin Siena's study of the more extensive primary sources for London during the eighteenth century.[63] This indicates that the two sexes were also therefore approximately equally at risk to contract the much more infectious and potentially sterilizing STIs of gonorrhea and chlamydia. Gonorrhea has been found to be typically about four times more prevalent in populations lacking effective treatment for STIs, according to the most appropriate historical evidence available for making this comparison.[64] However, the modal age at marriage for both sexes during these centuries was approximately twenty-five years.[65] To calculate the likely general effect of STIs in accounting for sterile marriages, we therefore need to know the average chances at this time of contracting an STI by age twenty-five, not by age thirty-five. Fortunately, the research done on the Chester Infirmary registers can provide such an estimate, and this indicates that just under three-quarters of the overall chance of infection by age thirty-five in rural Cheshire and northeast Wales had occurred by age twenty-five.[66]

The precise working out of the estimates are as follows. It can be deduced from tables 7A and 7B of Szreter "Treatment Rates for the Pox" that those marrying at age twenty-five in Cheshire in the 1770s had a 73.28 percent chance of having contracted the pox relative to those aged thirty-five (this is the mean chance for both sexes combined), and so the estimated figure of 1.324 percent infection among the Trussel and Wilson English parish register population should be reduced to 0.970 percent (1.324 × 0.7328). Multiplied by 4 (to reflect the higher infectivity of gonorrhea), this equates to a 3.881 percent chance of infection with gonorrhea. Using the sex-differential formulas for the chances of this leading to sterility developed in the text of this chapter, a gonorrhea infection rate of 3.881 percent by age twenty-five implies that 0.19 percent of men would have entered marriage sterile (3.881 percent multiplied by a 4.9 percent chance of sterility). The female marital sterility rate due to STIs (including the effect of chlamydia) would have been 0.47 percent (3.881 percent multiplied by the 12.12 percent chance

of sterility due to the sequlae of both diseases combined). This totals 0.66 percent, both sexes combined, thus reducing the Trussel and Wilson figure of 9.1 percent to 8.44 percent. The equivalent figures, adjusted by reference to the Chester schedule of the age incidence of pox exposure, can also be calculated in the same way for those marrying at twenty to twenty-four and thirty to thirty-four, producing estimates that the Trussel and Wilson figure of 4.6 percent sterile for those marrying wives at ages twenty to twenty-four would have been 4.287 percent in the absence of all STIs, and the figure of 16.6 percent for those marrying wives aged thirty to thirty-four would have been 15.79 percent.[67]

Appendix C: Constructing the Five Male Occupational Groups, Utilizing Both the 1911 Census I-CeM Data and the Seventy-Fifth *ARRG*.

Professionals are well-suited to this exercise as occupational identity tended not to change with age. Some 6,078 couples have been identified from the I-CeM database, where the husband was enumerated with one of the five selected professional occupations and where their wives were aged forty-five and over in 1911 and married at ages twenty to twenty-four, twenty-five to twenty-nine, and thirty to thirty-four, with a respective duration of marriage between twenty-five to twenty-nine, twenty to twenty-four, and fifteen to nineteen years, respectively (so as to ensure a completed fertility record).

The figure of 6,078 couples compares to that of 6,308 from the published table: Census of England and Wales, 1911, Vol. XIII: Fertility of Marriage, Part II (HMSO 1923), Cd 8491, Table 35. Marriages where the wife's age exceeded 45 years at census. Families and mortality therein, classified by occupation of the husband, duration of marriage, and age of wife at marriage (100–102, 108). There are multiple reasons why a discrepancy between what might be called the "observed" (the aggregate figure of the published 1911 census reports) and the "expected" (the figures calculated from the I-CeM database) will occur. First, some of the records of the original census manuscript appear to have been lost and are therefore not in the digital version; hence the population of England and Wales is reported in the published census as 36,070,492, while the comparable I-CeM figure is 36,031,749. Second, the original census document was handwritten, and because this is the first British census for which the householders' schedules themselves form the "original" census record, rather than a set of officially compiled census enumerators' books, the handwriting varies with each household and can sometimes be difficult to interpret. Thus, even though every best effort was

taken to minimize "error" in the transcription, differences of interpretation will inevitably occur. Detailed checking of the data by hand, including verification against the original census records, confirmed that the transcription of the Hollerith occupation code (a three-digit number written in green ink by officials against each married male householder to assign the marriage's fertility to a single officially defined occupation) was extremely accurate. Given that the I-CeM project coded the occupation textual strings (derived directly from the written statement by the householder on the census document itself), independently of the assigned Hollerith code, a detailed check on this type of transcription error was undertaken and mistranscribed codes corrected (and miscoded occupations also corrected). The fertility information itself (number of years married, number of children ever born, number of children died, and number of children still living) can be cross-correlated. The Census Office eliminated 122,286 couples from the total of 6,136,605 from their analyses on the basis of invalid information, rejecting some 2 percent of all couple records.[68] For the I-CeM data this rejection rate is higher (4.01 percent), due largely to cases where the transcribers left blank the "years married" answer on the original schedule. Thus, it is impossible to replicate the figures published in the census tables precisely. However, more important is not the shortfall of valid fertility records but rather if those that are "missing" display significantly different fertility histories to the valid "observed" records. In this regard the difference is extremely slight: an observed number of births per couple of 2.93 against an expected 2.95. This, alongside all the manual checking on the records analyzed here, indicates that there is no evidence to suggest a substantial parity-specific bias of the sort that would invalidate the analyses of this chapter.

To these 6,078 couples it was necessary to add a further 627 couples. This is because the total numbers of deaths recorded in the seventy-fifth *ARRG*, which is being used here for the parasyphilitic death rates it reveals, were usually classified by the individual's *previous* occupation if he died postretirement (or out of work).[69] Thus, those from the selected occupations but identified as retired in the I-CeM database by self-attribution—many of whom were considerably less than age sixty-five—were also included in the analysis here to ensure comparability between the two sources. Aggregating the retired and active together is also important since, as table 12.4 illustrates, the retired exhibit a disproportionate tendency to childless marriages. In each of the selected occupations, the retired subset record both lower births per couple and higher percentage zero parity than their "active" counterparts, a tendency especially marked in the case of both lawyers (barristers and solicitors combined) and physicians.

Table 12.4. Children born per couple and parity among selected occupations: retired and active.

Occupation	Age of wife at marriage	Duration of marriage	Active			Retired			Difference	
			Births per couple	% of couples with no births	Couples (n)	Births per couple	% of couples with no births	Couples (n)	Births per couple	% of couples with no births
Bankers	20–24	25–29	3.60	7.4	367	3.52	10.3	126	-0.08	3.0
	25–29	20–24	2.51	13.0	539	2.28	17.1	111	-0.23	4.1
	30–34	15–19	1.99	18.2	231	1.41	40.5	37	-0.58	22.4
Clergy	20–24	25–29	4.17	8.8	772	3.28	19.4	36	-0.89	10.6
	25–29	20–24	3.16	13.3	992	1.73	34.6	26	-1.43	21.3
	30–34	15–19	2.24	22.2	640	1.36	21.4	14	-0.88	-0.8
Lawyers	20–24	25–29	3.42	10.1	436	2.84	18.8	64	-0.58	8.7
	25–29	20–24	2.68	13.7	526	1.73	45.5	66	-0.95	31.8
	30–34	15–19	1.92	23.9	284	1.57	23.8	21	-0.35	-0.1
Surgeons	20–24	25–29	3.60	7.4	367	2.31	31.3	48	-1.29	23.9
	25–29	20–24	2.51	13.0	540	2.10	23.8	63	-0.41	10.9
	30–34	15–19	1.99	22.9	384	1.67	26.7	15	-0.32	3.8
Bankers			2.76	12.3	1137	2.73	17.2	274	-0.02	4.9
Clergy			3.24	14.2	2404	2.40	25.0	76	-2.38	4.0
Lawyers			2.76	14.8	1246	2.18	31.1	151	-0.59	16.4
Surgeons			2.67	14.3	1291	2.13	27.0	126	-0.54	12.7

The association with sterility among those retiring relatively young could be due to various factors, none of which are mutually exclusive: selection for poor health generally or for the specific ill-effects on health as well as fertility of STIs; it could also be partly a reverse social selection effect that those restricting their fertility most rigorously—either voluntarily or involuntarily—could afford to retire relatively early. In relation to this latter point, it is also the case that the selected occupations we are studying exhibit greatly varying propensities to retire, as shown in table 12.5. Bankers, many of whom may have had a company superannuation policy, retired earlier and almost universally by age seventy. In contrast, clergy, the majority of whom would not have had any formal pension, seldom retired.[70] Three-quarters of lawyers and nearly two-thirds of physicians were still recorded as active at age sixty-five, yet it may have been that many of these were in reality part-time or practiced only when they wished to. Overall, these patterns and considerations tend to suggest the predominance of social, over morbidity, factors in the decision to retire.

Table 12.5. Proportions retired by age for selected occupational categories

	Percentage retired by age			
Age	Bankers	Lawyers	Clergy	Physicians
55	18	13	3	7
60	43	16	3	26
65	75	24	13	36
70	90	35	16	40

Source: The Integrated Census Microdata Project (I-CeM), http://doi.org/10.5255/UKDA-SN-7481-1

Note: These figures are based on the couples where wives were aged 45 and over in 1911 and married at ages 20–24, 25–29, and 30–34, with respective durations of marriage 25–29, 20–24, and 15–19 years.

Appendix D: Evaluation of the Possible Effect of Differences in Marital Status among Males Pursuing Professional Occupations on STI Rates by Age Thirty-Five

Before the widely differing occupational rates of implied infection with syphilis recorded in the supplement to the seventy-fifth *ARRG* can be confidently assigned any possible influence over marital fertility patterns through derivative estimates of STI infection by age thirty-five, it is first necessary to consider the possible effect due to the fact that these rates published in the

seventy-fifth *ARRG* refer to married, widowed, and never-married men pursuing these various occupations. Unless only very small proportions of men pursuing these occupations never married, it is theoretically possible that a disproportionate number of those recorded in the nation's death registers as dying from parasyphilitic causes were drawn from among the unmarried in each occupation. This could be either because having knowledge of a prior infection deterred them from entering marriage or because it was those men within each occupation who had either failed or opted not to marry who were most at risk to contract an STI, if for instance it was the case that they were more likely, on average, to be exposed to the risks that followed from engaging in commercial sex than were married men.

While there is no directly relevant evidence with which to evaluate this possibility systematically (since the death registers of England and Wales cannot be searched systematically, unlike those of Scotland), it is nevertheless possible to assess this factor indirectly with evidence from the I-CeM database, from which can be derived statistics of the extent to which men pursuing these occupations in 1911 remained unmarried at different ages. Utilizing the I-CeM database, table 12.6 confirms that men in all these professional occupations tended to be significantly less likely to be married than the national average at all ages in 1911. The second row of table 12.6 shows that just over 50 percent of men had married before age thirty in the general population, as against less than 25 percent in these professional occupations. Just over three-quarters of men aged thirty to thirty-nine in the general population at the 1911 census were ever married, whereas this was the case for only five out of eight of these professional men. However, part of this is clearly related to the distinctive and different age distribution of men recorded in professional occupations at the census as can be seen by comparing columns (h) and (j). There were barely half the proportion of these occupations' totals engaged in their professional occupations at age twenty to twenty-four, compared to the national average (8 percent as against 14.6 percent) and still about one-fifth less at ages twenty-five to twenty-nine.

Furthermore, the detail on display in table 12.6 offers no evidence of a consistent correlation among these four occupational categories in this respect, particularly where the two most important age groups, twenty-five to twenty-nine and thirty to thirty-nine, are concerned. These two age groups are the most important, partly because, unlike ages twenty to twenty-four or higher ages above age thirty-nine, they each contain proportionately the largest number of individuals (11.8 percent per five-year age group) as can be seen from column (h). Second, and even more important, the differential behavior of older age-at-marriage groups cannot have exerted influence on

Table 12.6. Proportions of ever-married males by age in selected professional occupations and England and Wales, 1911.

(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)	(j)
	Proportions of ever-married men by age group						Age distribution (%)	
Age	All England and Wales	Bankers	Lawyers	Physicians	Clergy	Four professions combined	Four professions combined	England and Wales
20–24	14.6	2.0	4.9	3.2	5.3	2.3	8.0	14.6
25–29	50.8	24.1	22.1	22.0	19.5	22.6	11.8	14.2
30–39	76.9	62.5	59.1	62.9	64.5	62.5	23.6	25.7
40–49	86.0	79.2	78.6	82.0	81.2	80.5	22.8	19.5
50–59	89.2	83.4	83.2	87.0	86.9	85.4	17.4	13.4
60–69	90.4	88.9	85.7	90.1	90.3	89.1	10.5	8.3
70–79	92.0	92.5	89.0	89.7	91.2	90.9	4.7	3.6
80+	93.0	96.8	90.1	91.4	92.0	92.7	1.3	0.8

Source: The Integrated Census Microdata Project (I-CeM), http://doi.org/10.5255/UKDA-SN-7481-1

Note: Columns (h) and (j) each sum to 100 percent and give the percentage size of each male age group for the four professional categories combined (h) and for all men in England and Wales (j), regardless of marital status (ever married and never married combined).

the chances of acquiring a syphilitic infection by age thirty-five, which is the key "dependent variable" in this discussion of whether marriage propensities could have influenced STI differentials. At ages twenty-five to twenty-nine, it is in fact the clergy who are the least likely to be married, and the bankers who are most likely to be married among these professional occupations; yet they are at the opposite extremes in table 12.2, with bankers over five times more prone to have had a syphilitic infection by age thirty-five than clergy. The marriage patterns at the second most important age range, age thirty to thirty-nine, are also far from convincing in differentiating these four occupational categories according to their propensity to die from para-syphilitic causes of death. Bankers exhibit exactly the average proportion of unmarried men among this set of four professional categories, very similar to that of doctors, even though bankers' STI rate was the highest and over twice as high as doctors. At this age group it is the lawyers who are least likely to be married, rather than the bankers, yet table 12.2 shows that it was the latter who had a somewhat higher likelihood of contracting syphilis by age thirty-five. It is concluded therefore that there is no evidence here of a systematic relationship among these professional occupational categories between proportions of males remaining unmarried and propensities to die from syphilitic causes of death.

Appendix E: Considerations in Setting an Estimate for the Extent of Additional Marital Infertility Due to Chlamydia in Populations in England and Wales prior to 1914

A major review of 2013 that provides an international survey of eighty clinical epidemiological studies—including many conducted in India and other countries where population health and lack of comprehensive health service conditions are somewhat more akin to those prevailing in Britain's past before 1914 than can be the case for studies conducted among the populations of the OECD countries today—concludes, "Chlamydial PID is the single most important preventable cause of infertility. Approximately, three per cent of women with chlamydial genital tract infection develop infertility."[71]

However, the most rigorous attempt to evaluate the relationship between chlamydia infection and infertility in the British population today has produced a much lower overall headline figure, finding that the chance of a single chlamydia infection, either symptomatic or not, resulting in PID is as high as 17.1 percent but that only one in thirty-four infections (0.51 percent) then result in sterility (TFI).[72] This is of course a far lower rate of progression from PID to sterilization than the three in ten chance from a

gonorrheal PID episode found in the Lund cohort study 1964–88, cited in the chapter's main text. But note the following point about the enhanced effectiveness of treatment for PID in the United Kingdom today, compared with Swedish populations recruited from the mid-1960s onward, which is partly contributing to this much lower progression rate. There are additionally a number of other important considerations, which need to be taken into account when comparing this 0.51 percent figure with the 3 percent figure from the 2013 international survey and when assessing these different estimates as a guide to likely sterility outcomes in pre-1914 British populations—which perhaps suggest that they are not quite as far apart as they may initially seem.

First, while the 0.51 percent estimate did, very helpfully, take into account the absence of treatment for the 76 percent of cases estimated to be asymptomatic, it should be mechanically inflated when applied to a pre-1914 populace to allow for the 24 percent of symptomatic cases in the contemporary United Kingdom that did receive treatment before the onset of PID. Therefore, for comparability to a pre-1914 population, the 0.51 percent figure should be adjusted upward by 31.58 percent (24/76) to a value of 0.671 percent. Second, this figure relates to a population enjoying modern Western treatment even before onset of PID. The authors estimate that 42 percent of all PID is in fact diagnosed and treated in the general population today before it can progress to TFI. The Lund study showed that women treated for TFI within three days of diagnosis had a 280 percent reduction in their risk of infertility, indicating that the complete absence of treatment for chlamydia in the pre-1914 population must have very significantly raised—indeed multiplied by a factor of about 2.8—the 0.671 percent figure, which is derived from modern populations generally receiving extensive treatment. This produces a figure in the region of 1.88 percent.

Third, a range of untreated coinfections, most notably gonorrhea itself but also the many other STI and non-STI conditions suffered by the pre-1914 populations, are likely to have exerted an additional multiplicative risk on the chance of any single chlamydia infection resulting in both PID and TFI, though exact research findings on the statistical scale of these effects are lacking.

Fourth, in an entirely untreated population of married couples, which is the focus of this chapter's analysis, it is not at all clear that infection with chlamydia can be treated as a sequence of discrete events, as has been possible in the contemporary methodology used by Malcolm Price and colleagues, and which is indeed fundamental to the way in which its findings are presented. With no treatment available to either partner, the risks of

female infection leading to sterility in such circumstances are more akin to a compounded sequence of several infectious events, within the terminology of the Price and colleagues' study, "The Natural History of Chlamydia trachomatis." In these circumstances a woman exposed to unrestricted sex with a *C. trachoma*–positive partner (who would not have known of his typically asymptomatic condition relating to a disease at that time unknown) is most likely to have been exposed to an excess risk of the infection progressing to salpingitis and TFI for one of two reasons, according to the current models of the different processes that lead to variability in individual susceptibility to TFI, reviewed by Shruti Menon and colleagues.[73] One possibility is that such women would have been susceptible to a relatively rapid onset form of PID due to accumulating a high vaginal loading with multiple chlamydial inoculations from their individual partner before any immunity had developed in their own bodies and would then be at a high risk of developing PID once the immune response did react strongly to this high load (the tissue damage that causes PID and ultimately TFI is a byproduct of the body's immune response to the presence of chlamydia). Another possibility is that chlamydia can also produce PID from a more indolent pattern of repeat reinfection over a prolonged period due to a chlamydia-positive male partner in a stable relationship, since it has been shown in 2017 that male asymptomatic chlamydia infections can persist for over two years if untreated.[74] Katy Turner and colleagues have shown that, even with the levels of treatment available today, partners are chlamydia-positive in 60 percent of cases.[75]

   In the present state of research, these factors are not amenable to calibrating in mathematical form to offer precise adjustments to the figure of 0.671 percent or that of 1.88 percent, but it is clear that they each have the effect of significantly increasing that figure and so indicate far less discrepancy with the figure of 3 percent offered in the 2013 international review than would appear to be the case at first sight. Consequently, the empirically based figure of 3 percent, equal to almost exactly one-third of the female infertility rate attributed to untreated gonorrhea, is adopted here as probably an appropriate approximation, in the current state of knowledge, of the risks of female sterility that applied in the epidemiological and health care conditions prevailing among the British married populations at any time before the Great War, when general disease and bodily hygiene conditions were so different than they are today among early twenty-first-century OECD populations.

Notes

1. Richard Soloway, *Birth Control and the Population Question in England, 1877–1930* (Chapel Hill: University of North Carolina Press, 1982); Susan Kent, *Sex and Suffrage in Britain, 1860–1914* (Princeton: Princeton University Press, 1987); Timothy Jones, *Sexual Politics in the Church of England, 1857–1957* (Oxford: Oxford University Press, 2013), esp. chap. 5.

2. For a pioneering medical and scientific examination of these issues, see James Matthews Duncan, *Fecundity, Fertility, Sterility and Allied Topics* (Edinburgh: A and C. Black, 1866; rev. ed. 1871).

3. Émile Zola's 1885 novel, *Germinal*, is of course a celebrated literary example, and see Fabrice Cahen and Adrien Minard, chap. 11, in this volume, on persistent French fears. On relevant literature in Britain, see Angelique Richardson, *Love and Eugenics in the Late Nineteenth Century: Rational Reproduction and the New Woman* (Oxford: Oxford University Press, 2003).

4. The literature is legion. For an early highly relevant contribution, see William Bynum, "Darwin and the Doctors: Evolution, Diathesis, and Germs in Nineteenth-Century Britain," *Gesnerus* 40 (1983): 43–53; for an overview, see Peter Bowler, *Evolution: The History of an Idea*, 3rd ed. (Berkeley: University of California, 2003); on germ theory, see Michael Worboys, *Spreading Germs: Disease Theories and Medical Practice in Britain, 1865–1900* (Cambridge: Cambridge University Press, 2000).

5. Louisa Martindale's *Under the Surface* (London: National Union of Women's Suffrage Societies, 1908) may have been the first to do so. See Lucy Bland, "Marriage Laid Bare: Middle-Class Women and Marital Sex, c. 1800–1914," in *Labour and Love: Women's Experience of Home and Family, 1850–1940*, ed. Jane Lewis (Oxford: Basil Blackwell, 1986), 123–46; and, more generally, Bland, *Banishing the Beast: English Feminism and Sexual Morality, 1885–1914* (London: Penguin Books, 1995).

6. On the first and the third of these inquiries, see Simon Szreter, *Fertility, Class and Gender in Britain, 1860–1940* (Cambridge: Cambridge University Press, 1996), chaps. 4, 5; on the second, see Harvey Simmons, "Explaining Social Policy: The English Mental Deficiency Act of 1913," *Journal of Social History* 11, (1978): 387–403; and Leon Radzinowicz and Roger Hood, *The Emergence of Penal Policy in Victorian and Edwardian England* (Oxford: Clarendon, 1990); on the fourth, see John M. Eyler, *Sir Arthur Newsholme and State Medicine 1885–1935* (Cambridge: Cambridge University Press, 1997), 277–94. The Royal Commission on the Poor Laws, 1905–9 and the Royal Commission on Divorce and Matrimonial Causes of 1909–12 were also major inquiries hearing relevant evidence. On the former, see Alan Marne McBriar, *Edwardian Mixed Doubles: The Bosanquets Versus the Webbs, a Study in British Social Policy, 1890–1929* (Oxford: Clarendon, 1987); on the latter, see Lucy Owen, "Divorce: 'Disease or Remedy'? Medical Witnesses before the Royal Commission on Divorce and Matrimonial Causes, 1900–1912" (PhD diss., University of Oxford, 1996).

7. Simon Szreter, "The Prevalence of Syphilis in England and Wales on the Eve of the Great War: Re-visiting the Estimates of the Royal Commission on Venereal Diseases, 1913–1916," *Social History of Medicine* 27 (2014): 508–29.

8. For instance, see Daniel Pick, *Faces of Degeneration: Aspects of a European Disorder c. 1848–c. 1918* (Cambridge: Cambridge University Press, 1989); and Richard Soloway, *Demography and Degeneration: Eugenics and the Declining Birthrate in Twentieth-Century Britain* (Chapel Hill: University of North Carolina Press, 1990).

9. See also Simon Szreter, "Fertility Transitions and Sexually Transmitted Infections," in *Reproduction: Antiquity to the Present Day*, ed. Nick Hopwood, Rebecca Flemming, and Lauren Kasssel (Cambridge: Cambridge University Press, 2018), 443–56; and Tim Bayliss-Smith, chap. 6; Roy Scragg, chap. 7; and Shane Doyle, chap. 8, all in this volume. On interwar claims in regard to black US Americans, see Stewart E. Tolnay, "A New Look at the Effect of Venereal Disease on Black Fertility: The Deep South in 1940," *Demography* 26 (1989): 679–90, 679.

10. Owen, "Divorce"; Kent, *Sex and Suffrage*.

11. Joseph Banks, *Victorian Values: Secularism and the Size of Families* (London: Routledge, 1981), esp. chap. 3.

12. Arguably, it was the eugenicists and their claims that resulted in the interdepartmental inquiry of 1904 and—following from its conclusions—the 1911 census inquiry and the Royal Commission on the Feeble-Minded of 1904–9. See Szreter, *Fertility, Class and Gender*, 238–82.

13. Michael Worboys, "Unsexing Gonorrhoea: Bacteriologists, Gynaecologists, and Suffragists in Britain, 1860–1920," *Social History of Medicine* 17 (2004): 41–59.

14. Juliet Hurn, "History of General Paralysis of the Insane in Britain, 1830 to 1950" (PhD diss., University of London, 1998); Sharon Matthews, "Matter over Mind: The Contributions of the Neuropathologist Sir Frederick Mott to British Psychiatry, 1895–1926" (PhD diss., University of Manchester, 2006).

15. Census of England and Wales, 1911, Vol. XIII: Fertility of Marriage, Part II (HMSO 1923), Cd 8491, xix–xlvii.

16. T. H. C. Stevenson, "The Laws Governing Population," *Journal of the Royal Statistical Society* 88 (1925): 63–90; Stevenson, "The Vital Statistics of Wealth and Poverty," *Journal of the Royal Statistical Society* 91 (1928): 207–30.

17. For a review of this literature, see Szreter, *Fertility, Class and Gender*, 9–66. See Cahen and Minard, chap. 11, in this volume, for a discussion of a similar trajectory toward a scientific consensus to focus exclusively on volitional forms of birth control among demographers in France.

18. There is one partial exception to this statement, which is a series of studies by US demographers in the 1970s and 1980s with the specific aim of estimating what proportion of the recorded fall in black fertility in the United States, circa 1880–1940, could be attributed to the impact of gonorrhea and syphilis. Commencing with Reynolds Farley, *Growth of the Black Population: A Study of Demographic Trends* (Chicago: Markham, 1970) and including Joseph McFalls and Marguerite McFalls,

Disease and Fertility (Orlando: Academic Press, 1984), chap. 19, this culminated in the multivariate statistical treatment by Tolnay, "New Look," in 1989. However, all these studies assumed in common a relatively low level of STIs at the baseline date in the 1880s, without offering any sound reason or evidence for making such an implausible assumption.

19. Prince A. Morrow, *Social Disease and Marriage: Social Prophylaxis* (New York: Lea Brothers, 1904), 106. Morrow also referred to "the expressive German phrase *ein kinde sterilität*" (sic). While *Einkindsterilität* was, indeed, associated with gonorrhea, it was also associated with other sequlae and infections following a first birth, notably puerpereal fever, all of which were also subsumed under the terms "secondary sterility" and "acquired sterility"—for instance, in the discussion in Enoch Heinrich Kisch, *Die Sterilität des Weibes*, 2nd ed. (Vienna: Urban und Schwarzenberg, 1895), 302–4. A subsequent 1910 summary in an authoritative German handbook for medical students pointed out that, while an infection with gonorrhea did not necessarily lead to sterility, as was proven by the gonorrheal eye infections of newborns, the opportunity for gonococci to ascend beyond the cervix in the postpartum period when lochia was produced could result in *Einkindsterilität*; and this was something that could also be provoked by unwise medical "interference" in the postpartum period (an oblique reference to other dangers, such as puerperal fever). Albert Döderlein, "Die Gonorrhoischen Erkrankungen der Weiblichen Geschlechtsorgane," in *Kurzes Lehrbuch der Gynäkologie*, ed. Otto Küstner, 4th ed. (Jena: Fischer, 1910), 436 and 443. We are grateful to Christina Benninghaus for information on these German sources (pers. email comm., February 2, 2019). The exact way in which, in women who had never had a birth, gonorrhea so often gained access from the cervical area to the fallopian tubes, without typically infecting the intervening endometrium, continued to puzzle medicine for decades. In his subsequent handbook, Pelouze cogently argued that since it could be shown that blood commonly retrofluxes through the fallopian tubes at menstruation, that this would carry the gonococcus upwards within the uterine system, explaining why salpingitis could be common even in 'nulliparae' women who had never given birth. Percy S. Pelouze, *Gonorrhea in the Male and Female: A Book for Practitioners*, 3rd ed. (Philadelphia: Saunders, 1941), 313–25.

20. Michael Anderson, "Highly Restricted Fertility: Very Small Families in the British Fertility Decline," *Population Studies* 52 (1998): 177–99, 193, 196. Anderson has returned to this theme but has not presented any substantial body of additional literature from the pre–Great War period: Michael Anderson, *Scotland's Populations: From the 1850s to Today* (Oxford: Oxford University Press, 2018), 292–93. He has added a citation from Beatrice Webb expressing conflicted attitudes about childlessness, though of course in her own case she did not marry Sidney Webb until she was thirty-four, following a long, unconsummated infatuation with Joseph Chamberlain: Pat Jalland, *Women, Marriage and Politics, 1860–1914* (Oxford: Oxford University Press, 1986), 221. As has been noted, as late as 1938, a whole generation later, there was still active concern being expressed in the *British Medical Journal* over

the undesirability of forms of contraception resulting in the possibility of sterility of marriage; see Caroline Rusterholz, "Testing the Gräfenberg Ring in Interwar Britain: Norman Haire, Helena Wright, and the Debate over Statistical Evidence, Side Effects, and Intra-uterine Contraception," *Journal of the History of Medicine and Allied Sciences* 72 (2017): 448–67, who shows that the concern that contraception could lead to sterility was still common in medical circles at that time, citing the debate in the *British Medical Journal* in 1938 between George H. Alabaster, Joan Malleson, and Margaret C. N. Jackson.

21. Szreter, "The Prevalence," 525, tables 2, 3.

22. The Cambridge Group's parish family reconstitution files are described in full and extensively analyzed in E. A. Wrigley et al., *English Population History from Family Reconstitution, 1580–1837* (Cambridge: Cambridge University Press, 1997). See also Simon Szreter, "Treatment Rates for the Pox in Early Modern England: A Comparative Estimate of the Prevalence of Syphilis in the City of Chester and Its Rural Vicinity in the 1770s," *Continuity and Change* 32 (2017): 1–41.

23. There is no known information for ascertaining age at first sexual intercourse for men in this period, beyond that of the age at marriage. The assumption made here is consistent with the findings of the first relevant national survey, which was conducted by interview in 1990–91 and which found that the oldest males interviewed (aged forty-five to fifty-nine, born 1931–45 and therefore nineteen years old between 1950 and 1964) had a median age at first intercourse of nineteen years (a figure that fell a further two years to age seventeen among those aged sixteen to thirty-four at the time of the interviews); see Kaye Wellings et al., *Sexual Behaviour in Britain: The National Survey of Sexual Attitudes and Lifestyles* (London: Penguin, 1994), 41, table 2.2. Median age at first intercourse for men born five decades earlier, 1881–95, is extremely likely to have been several years older than nineteen, since the historically low illegitimacy rates in the Edwardian period indicate that for most of the population premarital intercourse was not an extensive practice and the average male age at marriage was as high as twenty-seven years. It was the period 1945–60 that witnessed a sharp rise in the proportion of younger men (age twenty to twenty-four) entering marriage; see B. Jane Elliott, "Demographic Trends in Domestic Life," in *Marriage, Domestic Life and Social Change*, ed. David Clark (London: Routledge, 1991), 85–108, fig. 4.2a. There is no evidence for a widespread norm of sexual initiation for young men before marriage with commercial sex workers, as appears to have been the case in France (see Cahen and Minard, chap. 11, in this volume). Clearly, this was a distinct possibility for some young men, despite the lack of social approval for it, and certainly studies both of prostitution and of the military in this period indicate that such premarital sexual activity did occur at least in these sections of the population, though it remains too opaque to evaluate its quantitative extent; see Julia Laite, *Common Prostitutes and Ordinary Citizens: Commercial Sex in London, 1885–1960* (Basingstoke: Palgrave Macmillan, 2011); and Alan Skelley, *The Victorian Army at Home: The Recruitment and Terms and Conditions of the British Regular, 1859–1899* (London: Croom Helm, 1977).

The assumption made here that the male risk of contracting STIs began on average from age twenty onward (initially, of course, at age twenty, relating to only a fraction of the male population, and thereafter growing in numbers year by year) is made in light of the knowledge that it is approximately six years before the average male age at marriage at this time and five years after the minimum age for entering service in the Royal Navy, with entry in the army supposed to be age eighteen minimum, while entry into the merchant navy was less regulated and could be even younger. Of course, in the absence of national service or conscription, these age markers for military service apply only to minorities of the population, not to all young men, as in some continental European populations at this time.

24. For the Swedish data for 1918–19, see Gunnar Dahlberg, "Venereal Diseases in Sweden, 1913 to 1937," *American Journal of Hygiene* 33 (1941): 51–63, sec. A, table 1 and Szreter, "Prevalence," pp.526–27, including notes 77, 78, and 79. These two years are most comparable in the sense that salvarsan treatments had not yet become a widespread practice among any populace and so would not have affected the relative incidence of syphilis. McFalls and McFalls, *Disease and Fertility*, 473, argues that the number of persons infected with gonorrhea, relative to syphilis, might be only half the four to one observed infection rates, because people could be infected multiple times with gonorrhea, and only once with syphilis. However, this fails to acknowledge the massive under-reporting of gonorrhea because it is asymptomatic in 75 percent of female and 45 percent of males: Richard Pattman et al., eds., *Oxford Handbook of Genitourinary Medicine, HIV, and Sexual Health*, 2nd ed. (Oxford: Oxford University Press, 2010), 150. Previously Szreter, "The Prevalence," p. 528, suggested a rate of male gonorrhea 3.25 times the syphilis rate. However, given the scale of asymptomatic gonorrhea, this would more than offset any multiple visits in the Swedish data (it appears no record was kept of this) and so we propose here a fourfold ratio.

25. This assumption is conservative for at least two reasons. First, it would have been possible for husbands and wives in a number of cases to keep reinfecting each other for a time with a gonorrhea (or chlamydia) infection originally brought into the marriage by the husband. Second, some husbands, and indeed some wives, would have engaged in commercial or adulterous extramarital sex after marriage. Both these factors are potential sources for increasing the risks of STI infection, relative to the estimates offered here. They are being deliberately ignored, partly for lack of reliable quantifiable evidence in either case, partly for the sake of simplicity of the model, and partly to ensure the estimates offered here of the impact of STIs on sterility remain conservative, minimal estimates.

26. These estimates are extremely simplified but, overall, robust. Strictly speaking, the empirical pattern of male ages at marriage should be taken into account. For men in England and Wales at this time the average age at first marriage was approximately twenty-seven years, a figure that falls exactly halfway between age twenty and age thirty-four. This means that halving the 10.4 percent estimate down to 5.2 percent for men marrying at age twenty to twenty-four will produce a significant

underestimate of their risks of sterility, since more than half of them were unmarried at all these ages. The figure for age twenty-five to twenty-nine would also be a (more moderate) underestimate, since it also incorporates the underestimate for ages twenty to twenty-four within its construction. The half-and-half assumption (married, unmarried) is a reasonably accurate assumption for this age group, twenty-five to twenty-nine, which evenly straddles the average male marriage age figure of twenty-seven years. Finally, the figure for age thirty to thirty-four would appear to be something of an overestimate, since a majority of men would have been married in this age range, as it is entirely above the national average age at marriage. However, this overestimation effect is partly offset by several factors. First, arithmetically, it also incorporates, as components of its 26 percent figure, both the underestimated figures for ages twenty to twenty-four and twenty-five to twenty-nine. Second, it is more likely that the remaining minority of men delaying their marriages into their thirties would have been greater users of commercial sex than men who married at younger ages, and the chances of any individual becoming infertile was a function of the accumulating absolute number of times they were exposed to a possible gonorrhea infection, not the relative number of exposures per unit of time. Third, although the male singulate mean age-at-marriage average was twenty-seven, a very considerable proportion of those aged thirty to thirty-four were still unmarried, probably at least 25 percent. This is a guesstimate, but it is informed by the fact that we do know that even when they were fifteen to twenty years older, as many as 11.66 percent of men forty-five to fifty-four years old were recorded as still never married at the 1911 census.

A robust estimate of the national average male singulate mean age at marriage (SMAM) can be derived from the data for the slightly over two thousand subregistration districts published on the Cambridge Group's atlas website: "Populations Past: Atlas of Victorian and Edwardian Population," University of Cambridge, accessed February 22, 2019, www.populationspast.org. For the two most relevant dates, the census years of 1891 and 1901, this data produces an overall national average (summing the singulate mean age at marriage values for each Registration Sub-District and weighting them by their population sizes) of 26.96 for 1891 and 27.22 for 1901. The same weighting procedure was used to produce the estimate of 11.6 percent males never married at age forty-five to fifty-four in 1911.

27. A clinical review from 2012 concludes, "Major problems for male fertility may arise particularly in patients with epididymitis as this disease appears to have a greater influence on semen quality and male fertility than an infection/inflammation of the prostate or seminal vesicle. In addition, in quite a number of patients, the diagnosis of chronic epididymitis is extremely difficult as these patients do not feel discomfort and their health is not compromised. Due to a silent nature of the infection/inflammation, epididymitis will only be diagnosed once these patients appear in an andrological clinic consulting for infertility." See Ralf Henkel, "Infection in Infertility," in *Male Infertility: Contemporary Clinical Approaches, Andrology, ART and*

Antioxidants, ed. Sijo J. Parekattil and Ashok Agarwal (New York: Springer Science + Business Media, 2012), 261–72.

28. Percy S. Pelouze, *Gonorrhea*, 240; and McFalls and McFalls, *Disease and Fertility*, 298, citing Stephen Kraus, "Complications of Gonococcal Infection," *Medical Clinics of North America* 56, no. 5 (1972): 1115–25; see 1115 for the figure of 17 percent.

29. Men in the decades prior to 1911—certainly those of a class who could afford it, such as professionals—were significantly more likely to seek treatment for gonorrhea than women, because they were more likely to experience symptoms; however, lacking sulfa drugs or antibiotics, the treatment available was not especially effective and could be both excruciating and itself damaging of fertility due to strictures and other consequences, treatments consisting, as they did, of urethral flushing, dilators, and application of a heated bougie. See J. David Oriel, *The Scars of Venus: A History of Venereology* (London: Springer, 1994), 140–42.

30. Richard Sweet and Harold Wiesenfeld, eds., *Pelvic Inflammatory Disease* (London: Taylor and Francis, 2006), 21.

31. The Lund twenty-four-year prospective cohort study, 1964–88, generated numerous research reports, which are accessibly summarized by Sweet and Wiesenfeld, *Pelvic Inflammatory Disease*, 69–71.

32. Ian N. Clarke and Hugh R. Taylor, chap. 4; Michael Worboys, chap. 5, both in this volume. As Worboys has also observed (pers. email comm. with Simon Szreter, May 19, 2017) of the first decades of the twentieth century, "Only in hindsight can we link inclusion bodies found in genital smears in the 1900s to chlamydia now." At that time "finding inclusion bodies in the genital tract of females and males didn't equate with any disease." But the fact that they were found by the leading scientists of the day confirms that what has since been identified as chlamydia was there in a number of cases examined where gonorrhea-like symptoms were presented. Drawing from page 7 of Phillips Thygeson, "Trachoma Virus: Historical Background and Review of Isolates," *Annals of New York Academy of Sciences* 98 (1962): 6–13, Worboys observes, "In 1910 Ludwig Halberstaedter and Stanislaus Prowazek first found inclusions in female genital epithelium, and in the same year Karl Lindner found them in three cases of nongonorrheal urethritis of men. . . . He postulated that trachoma and 'inclusion blennorrhea' ('genital trachoma' or 'paratrachoma') were caused by the same agent but suggested later that the two diseases might have a relationship like that of variola and vaccinia." Halberstaedter and Prowazek did use the German term *Chlamydozoen* (from the Greek for a type of cloak) in their article title because they observed the nucleus of the affected cells appeared to be cloaked by the affecting agent, which they assumed to be a protozoan, like other high-profile tropical diseases that had then been discovered, such as malaria and sleeping sickness.

33. An in-depth review of the field has identified as many as four "distinct mechanisms or processes of host-pathogen interaction that are commonly referred to in the field to explain the process that underlies the development of infertility following

Chlamydia infection. The processes are not exclusive of each other, and there is evidence for and against a role for each process." Shruti Menon et al., "Human and Pathogen Factors Associated with *Chlamydia trachomatis*–Related Infertility in Women," *Clinical Microbiology Review* 28, no. 4 (2015): 969–85, 977, https://doi.org/10.1128/CMR.00035-15. Such researches have shown, for instance, that at the individual level the pathway from infection to infertility can be dependent on many factors, including which of the dozen or so serovars is involved and what kind of immune response it elicits, the genotype of the host, the presence of coinfections, and the general state of hygiene of the infected host.

34. The most recent US literature metareview is Danielle G. Tsevat et al., "Sexually Transmitted Diseases and Infertility," *American Journal of Obsteteric Gynecology* 216 (2017): 1–9, https://doi.org/10.1016/j.ajog.2016.08.008. The most recent major National Health Service review, which also reevaluated the high-quality data available from the Netherlands study by Land and colleagues, concluded that even with current screening and treatment regimes, chlamydia is probably responsible today for about 29 percent of all tubal infertility (a reduction from the 45 percent figure found in the Dutch retrospective study, due to the superior prospective methodology adopted): Malcolm J. Price et al., "The Natural History of *Chlamydia trachomatis* Infection in Women: A Multi-parameter Evidence Synthesis," *Health Technology Assessment* 20 (2016): chap. 11, esp. 138. The original Dutch study was J. A. Land et al., "Performance of Five Serological Chlamydia Antibody Tests in Subfertile Women," *Human Reproduction* 18, no. 12 (2003): 2621–27, http://dx.doi.org/ 10.1093/humrep/deg479.

35. The 2013 international review Malhotra Meenakshi et al., "Genital Chlamydia Trachomatis: An Update," *Indian Journal of Medical Research* 138, no. 3 (2013): appendix E, concluded in the section titled "*Chlamydia trachomatis* and Infertility" that in the present state of documented knowledge "the role of *C. trachomatis* in male infertility is not yet proven." This is despite the fact that Allan Pacey and colleagues have shown that a chlamydial infection can damage and kill sperm: Adrian Eley, Allan A Pacey, Massimiliano Galdiero, Marilena Galdiero, Francesco Galdiero, "Can *Chlamydia trachomatis* Directly Damage Your Sperm?," *Lancet Infectious Diseases* 5, no. 1 (2005): 53–57. The negative finding that there continues to be no convincing evidence that the damage to either sperm or epididymis in men caused by chlamydia is sufficient to cause sterility has been confirmed by Paddy Horner, coauthor of Price et al., "Natural History of Chlamydia" (pers. email comm. with Szreter, September 6, 2017). On the likely reasons for this, see the discussion in Simon Szreter's introduction to this volume, p. 15.

36. Note that we are talking here only about the chances of infertility due to STIs among the married female population, which are purely a function of exposure to transmission of a "live" infectious episode from her husband. We are not talking about the husband's chances of STI-induced sterility, which follow an entirely different schedule of probability and which are posited to increase with his age at marriage because of the accumulation of exposure to separate risk incidents, any one of which

may result in his infertility. Note also that the chances of a husband passing on an infection of either gonorrhea or chlamydia remain the same, regardless of his age and even regardless of whether or not he himself has already been rendered sterile by a previous episode of gonorrhea infection. One of the few high-quality studies available, based on using large numbers, interviewing named contacts, and bacteriological testing of infections, confirmed very high rates of infectiousness of gonorrhea, particularly from male to female, in the order of 90 percent (and 74 percent from female to male). A. H. Pedersen and W. D. Harrah, "Follow-Up of Male and Female Contacts of Patients with Gonorrhea" *Public Health Reports* 85, no. 11 (1970): 997–1000. Furthermore, it is relevant that this study included many casual contacts. Even higher rates, approaching 100 percent, would be expected where repeated and regular exposures occur with the infected partner, as in a marriage; see P. V. Marcussen, "Variations in the Stability of Sexual Relations as Explanation of Differences in the Spread of Syphilis and Gonorrhea," *American Journal of Syphilis, Gonorrhea, and Venereal Diseases* 37 (1953): 355–61.

37. To take the example of the group of women marrying at age twenty to twenty-four, the estimate of a 0.886 percent chance of a childless marriage is arrived at in the following way. The female chance is 0.63 percent, while the additional male chance at ages twenty to twenty-four is 0.256 percent, summing to 0.886 percent. The female component remains at 0.63 percent among the two older female age groups, but the added male chance rises to 0.769 percent at ages twenty-five to twenty-nine and to 1.282 percent at ages thirty to thirty-four.

38. On Britain, see Anne Hanley, "'The Great Foe to the Reproduction of the Race': Changing Medical Knowledge and Practice; Diagnosing and Treating Infertility Caused by Venereal Diseases, 1880–1914," in *The Palgrave Handbook of Infertility in History: Approaches, Contexts, Perspectives*, ed. Gayle Davis and Tracey Loughran (London: Palgrave Macmillan, 2017), 335–58; on Germany, see Christina Benninghaus, chap. 10, in this volume.

39. James Trussel and Christopher Wilson, "Sterility in a Population with Natural Fertility," *Population Studies* 39 (1985): 269–86.

40. Szreter, "Treatment Rates," 26.

41. Chester's population in 1774 was 14,713; Gainsborough and Banbury reached 5,112 and 3,810, respectively, in 1801. For the sixteen parishes, see Trussel and Wilson, "Sterility in a Population," 278n18.

42. The family reconstitutions of all twenty-six English parishes compiled at the Cambridge Group for the History of Population and Social Structure, including both the sixteen parishes used by Trussel and Wilson, "Sterility in a Population" and the larger number used by Wrigley et al., *English Population History*, were kindly supplied to the authors by Gillian Newton of the Cambridge Group.

43. The multiplier is calculated as follows. First, the English family reconstitution data were selected by parish and period in accordance with the groupings given in table 2.2, on page 26, of Wrigley et al., *English Population History*. This differs from those selected in Trussel and Wilson, "Sterility in a Population," since Wrigley

and colleagues include the additional parishes of Austrey, Birstall, Bridford, Dawlish, Great Oakley, Ipplepen, Lowestoft, March, and Morchard Bishop, while at the same time excluding data from certain parishes for given periods on the grounds of reliability. Note that Wrigley and colleagues exclude the small parish of Hawkshead, included by Trussel and Wilson, "Sterility in a Population," in its entirety, on the basis that "the level of childlessness among married women was suspiciously high" (29) together with (unspecified) "other reasons for doubt" (30). Following the selection of data, rates for marriage with parity zero (childless) were calculated for completed marriages (but in line with Trussel and Wilson, "Sterility in a Population," allowing for remarriages), where the wife was aged, respectively, fifteen to nineteen, twenty to twenty-four, twenty-five to twenty-nine, or thirty to thirty-four at marriage. Rates were calculated for all parishes combined and for Banbury and Gainsborough separately. The ratio between each set of rates was then calculated, producing the result that for the four age-at-marriage groups, the rate of childless marriages in Banbury and Gainsborough was higher than that observed for all reconstitution parishes, combined, by a factor of 1.73, 1.16., 1.47, and 1.31. The final figure of 1.387 is the average of these four rates weighted, respectively, by the number of observations in each age-at-marriage group.

44. This estimate is from the 1801 census reproduced in Wrigley et at., *English Population History*, 22–23, table 2.1.

45. The figure of 0.424 is the product of the following computation, which weights the ratio of how much more sterility there was in Banbury and Gainsborough (1.387 times more) by the relative population size of these two parishes (8,922), expressed as a proportion of the total population of all sixteen parishes (29,169): $1.387 \times (8,922 / [20,247 + 8,922])$. This is $1.387 \times (8,922 / 29,169)$—that is, $1.387 \times 0.3059 = 0.424$.

46. See note 26 on derivation of this figure from "Populations Past."

47. This is the product of this sum (row 10 of table 12.1, weighted by row 4): $[(236.8 \times 52.1) + (149.3 \times 41.1) + (63.2 \times 28.1)] / (236.8 + 149.3 + 63.2) = (12337.3 + 6136.2 + 1775.9) / 449.3 = 20249.4 / 449.3 = 45.07$ percent.

48. Kevin Schürer and Edward Higgs, Integrated Census Microdata (I-CeM), 1851–1911[data collection]. UK Data Service, 2014 SN 7481, http://doi.org/10.5255/UKDA-SN-7481-1; see also Edward Higgs et al., *The Integrated Census Microdata (I-CeM) Guide* (Colchester: University of Essex, 2013). Further details on the I-CeM database, along with related resources, are available from the I-CeM website at "I-CeM: Introduction," Integrated Census Microdata Project, University of Essex, accessed February 27, 2019, https://www1.essex.ac.uk/history/research/icem/. The creation of the I-CEM database was made possible through funding from the UK Economic and Social Research Council, grant number RES-062-23-1629.

49. Registrar General, 1901–12, "England and Wales Supplement to Registrar-General's Seventy-Fifth Annual Report: Part IV; Mortality of Men in Certain Occupations in the Three Years, 1910, 1911 and 1912," *Online Historical Population*

Reports, 1901–12, accessed February 22, 2019, www.histpop.org. About 130 occupations were separately tabulated.

50. On the origins and limitations of this official scheme, see Szreter, *Fertility, Class and Gender*, 67–282; and Simon Szreter, "Fertility, Social Class, Gender and the Professional Model: Statistical Explanation and Historical Significance," *Economic History Review* 68 (2015): 707–22.

51. Final Report of the Commissioners, *Royal Commission on Venereal Diseases*, Cd. 8189, 1916. Stevenson did not rely on the cause-of-death category of "syphilis" because of the widely known reluctance on the part of physicians to imperil their commercial and confidential relationship with their paying clients by placing this obscenity on the death certificate of a beloved *paterfamilias*. Instead, Stevenson relied primarily on the evidence of age-standardized death rates from "general paralysis of the insane," locomotor ataxy, and aneurysm, each of which had only recently come to be understood as being due primarily to tertiary syphilis and so as yet carried no widespread stigma. See Szreter, "The Prevalence," 511.

52. Clergy include clergymen in the established church, as well as ministers and priests of other religious bodies (excluding Catholics), but excludes itinerant preachers, scripture readers, missionaries, and lay clerics. Barristers and solicitors exclude law clerks and other legal assistants. Attorneys are included with barristers. Medical practitioners include physicians, surgeons, and other registered practitioners but exclude dentists, pharmacists, dispensers, and herbalists. Bankers include bank managers and other bank officials and clerks. See Higgs et al., *Integrated Census Microdata*, 194–205.

53. Szreter, "The Prevalence," 525, table 2.

54. Szreter, *Fertility, Class and Gender*, 365–440. For other evidence on the importance of attempted abstinence, see Simon Szreter and Kate Fisher, *Sex before the Sexual Revolution* (Cambridge: Cambridge University Press, 2010), 229–65.

55. David Levine, John Gillis, and Louise Tilley, *The European Experience of Declining Fertility, 1850–1970: The Quiet Revolution* (Oxford: Blackwell, 1992). This diversity is further explored as part of the ESRC-funded Atlas of Victorian Fertility Decline project, ES/L015463/1, at the Cambridge Group for the History of Population and Social Structure, Department of Geography, University of Cambridge. For further details, see "An Atlas of Fertility Decline in England and Wales," Cambridge Group for the History of Population and Social Structure, accessed September 12, 2017, www.geog.cam.ac.uk/research/projects/victorianfertilitydecline/.

56. For postpuerperal sterility, see Maeve Kenny's work reported in Worboys, chap. 5, in this volume. See also Irvine Loudon, *Death in Childbirth: An International Study of Maternal Care and Maternal Mortality, 1800–1950* (Oxford: Clarendon, 1992), chaps. 12–15. Additionally, in note 19, above in this chapter, see the warning in the German medical textbook of 1910 against unwise medical 'interference' in the postpartum process, in relation to the risks of provoking *Einkindsterilität*.

57. Jean-Noël Biraben, "Le rôle des maladies sexuellement transmissibles en démographie historique," *Population*, (1996): 1041–57.

58. Trussel and Wilson, "Sterility in a Population"; Wrigley et al., *English Population History*, 384, table 7.11.

59. Compare Trussel and Wilson, "Sterility in a Population," 278n18, with Wrigley et al., *English Population History*, 22–39, esp. 26, table 2.2, and note that Wrigley explained (pers. comm., March 24, 2017) that a note on page 578, which states that Birstall originally lacked FRFs in the case of childless families, can be ignored since these were subsequently supplied and incorporated before the parish was used. Wrigley and colleagues draw on twenty-six separate parish reconstitutions. Trussel and Wilson draw on sixteen parishes listed in note 18 of their article (though this note lists in order of individuals who carried out reconstitution work, and so several parishes are listed twice).

60. Trussel and Wilson, "Sterility in a Population," 278.

61. Trussel and Wilson, "Sterility in a Population," 278.

62. Szreter, *Fertility, Class and Gender*, 290.

63. Kevin Siena, *Venereal Disease, Hospitals, and the Urban Poor: London's "Foul Wards," 1600–1800* (Rochester: University of Rochester Press, 2004), chap. 4, 135–80.

64. Dahlberg, "Venereal Diseases in Sweden," table 1, for data for the years 1918–19, before effective treatment for syphilis became increasingly widespread in the civilian population. For use of this source, see above p. 380 and note 24.

65. Wrigley et al., *English Population History*, 135, table 5.3.

66. Szreter, "Treatment Rates," tables 7A, 7B. Note that both the age incidence and the pattern of broad gender equality of infection among the surrounding rural population was similar to that of the Chester resident populace.

67. Szreter, "Treatment Rates," tables 7A, 7B. Those marrying at age twenty had on average only a 34.74 percent chance of having sought treatment for the pox, relative to those aged thirty-five, and so the figures of 1.324 percent infection should be reduced to 0.460 percent. Multiplied by 4 this equates to 1.84 percent infected with gonorrhea and therefore 0.223 percent of women and 0.090 percent of men sterile at marriage, totaling 0.313 percent, both sexes combined, thus reducing the Trussel and Wilson figure of 4.6 percent to 4.287 percent. Those marrying at age thirty to thirty-four had 89.94 percent of the chance of infection of those aged thirty-five, and so the 1.324 percent infection rate should be reduced to 1.191 percent. Multiplied by 4 (to estimate the gonorrhea rate), this equates to 4.763 percent infected with gonorrhea and therefore 0.577 percent of women (4.763×0.1212) and 0.233 percent of men (4.763×0.049), totaling 0.810 percent, both sexes combined, sterile at marriage; so the Trussel and Wilson figure of 16.6 percent would be reduced to 15.79 percent.

68. Census of England and Wales, 1911, vol. XIII, *Fertility of Marriage*, v.

69. Supplement to the seventy-fifth *ARRG*, pt. 4, p. vii, states that "throughout, the tables and figures refer to the occupied and the retired in the aggregate."

In fact, this was a new departure by Stevenson, as the "unoccupied" (including in that designation the retired) had previously been separated out in the corresponding occupational morality supplements to the sixty-fifth and the fifty-fifth *ARRG*s, relating to deaths registered in 1900–1902 and 1890–92, respectively. We can be sure that, to the contrary, those recorded in the census of 1911 stating they had retired from a named previous occupation were not included in any occupational fertility tables relating to the separate census exercise. This is because we know that with the new Hollerith punch-card system for analyzing the fertility census, all those stating they were retired (with the exception of the armed services and old-age pensioners (OAPs) specifically, which were each coded separately) were given the Hollerith code of 350 ("Retired from Business [Not Army or Navy]"), regardless of any other occupational information they included.

70. A voluntary pension scheme, the Clergy Pensions Institution was created in 1886 and had some 4,000 members by the late 1890s. In 1927 the Church of England Pension Board was created to provide a compulsory pension scheme and the institution was amalgamated into the new scheme. Alan Haig, *Victorian Clergy* (Basingstoke: Croom Helm, 1984), 324–25. In 1911 in England and Wales there were about 36,000 Anglican and nonconformist priests enumerated and about 3,300 Catholic priests.

71. Meenakshi et al., "Genital Chlamydia Trachomatis," 303–16, second sentence in section titled "Chlamydia Trachomatis and Infertility." This is broadly consistent with the results of a Cochrane review (also published in 2013), which found that "chlamydia ascends to the upper genital tract in approximately 10 percent of cases to cause symptomatic pelvic inflammatory disease" while "the probability of tubal infertility in women who have had chlamydia is estimated to be only 1% to 4%"; see Nicola Low et al., "Screening for Genital Chlamydia Infection," *Cochrane Database Systemic Review* 23, no. 12 (2013): 1–15.

72. Price et al., "Natural History of Chlamydia," xxvi. We would like to acknowledge the ready advice and assistance given by Prof. Malcolm Price, Dr. Paddy Horner, and Prof. Tony Ades when they were each consulted on the interpretation of the findings of their 2016 study. Responsibility for the use made of their advice and the judgements made here about historical applications remains of course that of the authors of this chapter.

73. Menon et al., "Human and Pathogen Factors," 969, 985, 977, fig. 1. Thanks go to Paddy Horner for additional interpretative assistance for this appendix.

74. Joanna Lewis et al., "Genital Chlamydia Trachomatis Infections Clear More Slowly in Men Than Women, but Are Less Likely to Become Established," *Journal of Infectious Diseases* 216 (2017): 237–44.

75. Katy Turner et al., "Costs and Cost Effectiveness of Different Strategies for Chlamydia Screening and Partner Notification: An Economic and Mathematical Modelling Study," *BMJ Research* 342, no. 7789 (2011): c7250, https://doi.org/10.1136/bmj.c7250.