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The geography of smallpox in England before vaccination: A conundrum resolved

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ABSTRACT

Smallpox is regarded as an ancient and lethal disease of humans, however very little is known about the prevalence and impact of smallpox before the advent of vaccination (c.1800). Here we use evidence from English burial records covering the period 1650–1799 to confirm a striking geography to smallpox patterns. Smallpox apparently circulated as a childhood disease in northern England and Sweden, even where population densities were low and settlement patterns dispersed. However, smallpox was a relatively rare epidemic disease in southern England outside the largest cities, despite its commercialised economy and the growing spatial interconnectedness of its settlements. We investigated a number of factors hypothesised to influence the regional circulation of smallpox, including exposure to naturally occurring orthopox viruses, settlement patterns, and deliberate preventative measures. We concluded that transmission was controlled in southern England by local practices of avoidance and mass inoculation that arose in the seventeenth and eighteenth centuries. Avoidance measures included isolation of victims in pest houses and private homes, as well as cancellation of markets and other public gatherings, and pre-dated the widespread use of inoculation. The historical pattern of smallpox in England supports phylogenetic evidence for a relatively recent origin of the variola strains that circulated in the twentieth century, and provides evidence for the efficacy of preventative strategies complementary to immunisation.

1. Introduction

Smallpox is widely considered one of the most lethal of all human pathogens, and was also the first disease to be eradicated. Vaccination was developed by Edward Jenner at the very end of the eighteenth century (Jenner, 1798), and reduced smallpox to a relatively minor cause of death in Europe by the mid-nineteenth century. However, smallpox still accounted for some 10–15 million cases annually as late as 1967 (Fenner et al., 1988: 175), and was only finally eliminated by a very concerted global campaign that combined mass vaccination with stringent isolation, contact-tracing and targeted ring-vaccination (Fenner et al., 1988: Foege, 2011). Smallpox was officially declared eradicated in 1980, although laboratory samples are still retained.

The precocious success of vaccination means that we know relatively little about the epidemiology of smallpox in unvaccinated populations. This is of more than just historical interest, because most of the global population currently lacks immunity against smallpox. Fears regarding the deliberate release of smallpox by bioterrorists in the wake of the September 11th, 2001 attacks led to a spate of mathematical modelling of the likely consequences of such an event. These models produced widely divergent recommendations, ranging from mass vaccination of national populations, to containment via case isolation and contact tracing. A major reason for the extent of discrepancies in model outcomes was the lack of robust historical evidence regarding the spread of smallpox in unvaccinated populations, which made it difficult to estimate some of the basic parameters of epidemic models (e.g. Eichner, 2003; Gani and Leach, 2001; Meltzer et al., 2001, Appendix 1).

Smallpox is caused by the orthopox virus variola (VARV). Several strains of varying virulence have been identified, and phylogenetic analyses of historical samples indicate that the variants that circulated in Europe before the twentieth century belonged to the most lethal strain, variola major (e.g. Li et al., 2007; Duggan et al., 2016). Smallpox was transmitted person to person (and to a lesser extent via infected objects), and conferred lifelong immunity on survivors. It is therefore regarded as a classic 'crowd disease', dependent on relatively large populations of susceptible hosts for continued transmission. The predicted historical dynamics of such person-to-person, immunising diseases were described best by the historian William McNeill. He argued that as human populations grew and became better integrated through trade and migration then disease introduction would have become

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more frequent and regular, and the accumulation of hosts more rapid, sustaining regular epidemics. As the frequency of epidemics increased and populations coalesced into large unified disease pools then few individuals escaped infection in childhood and most adults would have been immune. By this point smallpox would have become an endemic childhood disease that no longer required reintroduction into the population (McNeill, 1976). McNeill also recognised that this endemicisation process could involve a transitional phase, where immunising diseases such as smallpox had become endemic childhood diseases amongst long-term urban residents, but remained relatively infrequent and epidemic in surrounding rural areas. In this situation adult migrants to towns were often immunologically naive, and fell victim to urban diseases upon arrival, producing bimodal patterns of smallpox infection by age (McNeill, 1980).

The chronology of the endemicisation of smallpox in European populations remains unclear, because we have few records of causes of death before the nineteenth century. Although smallpox is usually described as an ancient disease of humans, both historical and molecular phylogenetic evidence generally supports a relatively recent origin of the variola major strain that circulated between the seventeenth and twentieth centuries (Carmichael and Silverstein, 1987; Duggan et al., 2016; Duncan et al., 2005). In London, by 1700 Europe's largest city, smallpox increased from 4 to 6% of all burials in the mid-seventeenth century to over 10% in the third quarter of the eighteenth century, and the frequency of epidemics increased from roughly four yearly to a biennial cycle over the same period (Davenport et al., 2016; Duncan et al., 1996; Krylova, 2011). Amongst London Quakers smallpox accounted for only 10% of all burials of children aged 2–4 years in the period 1650-99, but 26–29% in the period 1700-99 (Landers, 1993: 154). Thus smallpox appears to have become a more common cause of death over the course of the seventeenth and early eighteenth century in London, and may have become a more frequent epidemic disease outside the capital over the same period (Carmichael and Silverstein, 1987; Creighton, 1894: 434–445).

Where smallpox was recorded as a cause of death, then such records provide unusually rich information about the incidence of the disease. Although historical cause of death records must always be treated with great suspicion, smallpox was relatively distinctive in its symptoms and was considered by contemporaries to be confused only with chickenpox, which was rarely lethal (e.g. Buchan, 1774: 161; Creighton, 1894: 530). Estimates of case-fatality rates associated with variola major range from 10 to 30%, meaning that a sizeable proportion of all those infected can be detected in burial records where smallpox burials were noted. Smallpox was sufficiently virulent to kill adults as well as children, and was apparently relatively insensitive to host nutritional status (Dixon, 1962; Fenner et al., 1988: 196). The relatively small differences in case-fatality rates by age mean that the age structure of smallpox burials provides some indication of the age profile of those infected (Creighton, 1894: 520, 618; Nishiura et al., 2008). Because smallpox infection conferred lifelong immunity on survivors, then where adult victims were rare it is likely that most adults had already encountered smallpox in childhood. The age of smallpox victims therefore provides some indication of transmission patterns within a population.

In Sweden, where causes of death were reported for the population as a whole from 1749, smallpox accounted for 8.3% of deaths nationally in the period 1774-95 (when smallpox was first reported separately from measles) (Sköld, 1996: 549). In Finland, it accounted for 11% of deaths between 1776 and 1800 (Pitkanen et al., 1989: 98). In Sweden as a whole, 95% of smallpox deaths occurred at ages under ten years in the period 1788-92, and less than 10% of smallpox deaths were aged ten years or more in all dioceses except the very isolated island of Gotland (Sköld, 1996: 580). This pattern is consistent with the circulation of smallpox as an endemic disease of childhood.

Britain was much more densely settled than Sweden, and experienced very rapid urbanisation in the eighteenth century, with the proportion living in large towns (10,000+) rising from c.13% to over 20% in England and Wales in the course of the century (De Vries, 1984: 39). Thus, we might expect that smallpox was, as in Sweden, an endemic childhood disease and major cause of death by the mid-eighteenth century. However, the fragmentary records that exist for England present a conundrum. The few records we have that reported causes of death in the eighteenth century indicate that smallpox was a very major cause of death in some of the larger towns and cities, accounting for 10–20% of all burials in London, Manchester and a number of other northern towns (Davenport, in press). However, although smallpox was constantly present in London and was a childhood disease of urban-born residents, around 20% of smallpox burials were of young adults in eighteenth century London (Davenport et al., 2011, 2016; Landers, 1993: 153-56). Young adults constituted the main source of migrants to London, and the bimodal age pattern of smallpox victims confirms McNeill's prediction regarding the vulnerability of rural-urban migrants (McNeill, 1980). However, it also implies that London's migrant hinterland in this period included significant areas where much of the population grew to adulthood without encountering smallpox. In contrast, in mid-eighteenth-century Manchester in northern England less than 5% of smallpox burials were aged ten or over, despite the similarities in age structure of the populations of Manchester and London (Davenport et al., 2016). These differences in urban smallpox patterns appear to reflect a wider north-south divide in smallpox patterns tentatively identified by Razzell (2003). Using a small sample of burial registers that recorded smallpox burials, Razzell reported that adult smallpox victims were rare in northern England, but common in southern England. He interpreted this pattern to imply major differences in exposure to smallpox between the two regions (Razzell, 2003: x - xiv).

To investigate these intriguing patterns further we exploited the recent explosion in digital data for genealogists to search millions of burial records for mention of smallpox. Although very few parish registers consistently recorded causes of death, incumbents often noted deaths from smallpox, as well as plague and violent deaths. We identified 225 burial registers from the period 1540–1799 that reported smallpox burials and included some indication of age of victims. We then used the age patterns of smallpox victims to infer the frequency of epidemics, and tested four main theories regarding the geography of these patterns.

2. Methods

2.1. Data

To identify smallpox burials we searched c.7 million burial records donated by family history and genealogical societies in electronic form (see Table S1 for sources and geographical coverage), as well as commercial genealogical databases, using variants of the search terms ‘smallpox’ and ‘variola’. We also searched published sources of data for specific parishes identified in the secondary literature as reporting causes of death. Where smallpox burials were recorded then we evaluated the quality of recording of age information. Some registers recorded exact age in years, and some gave explicit age indicators such as ‘infant’, ‘child’, ‘young man’. The majority recorded relationships that could be used to infer adult or child status, such as ‘son of’, ‘daughter of’, ‘wife of’, ‘widow of’ (e.g. Table S2). We classified all descriptors of the types ‘son of’, ‘daughter of’ and ‘child of’ as child burials, and all marital and occupational descriptors as adult. Single women and adult males of any marital status were rarely ascribed a relationship, and their adult status was inferred either from occupational or status information (e.g. ‘labourer’, ‘gent’), or from the lack of any descriptor. This procedure could obviously create an adult bias if many entries lacked information for reasons other than age or marital status, and so only registers where more than 60% of all entries contained relationship indicators for the decades surrounding the period of smallpox