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Measles and the spatio-temporal structure of modern Japan[1]

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This article explores the spatio-temporal structure of infectious diseases in modern Japan, using measles mortality data from the late nineteenth and early twentieth centuries. Three aspects of the epidemiology of measles are discussed: the synchronization of epidemic waves, seasonality, and age at infection. These epidemiological analyses are connected to, respectively, regional integration, governmental policy on primary school education, and the number of young children in families. In addition, based on the fact that measles did not become endemic in early modern Edo (Tokyo), this article corrects epidemiologists' misunderstanding about the threshold of endemicity and argues that the critical population size varied substantially according to the societal factors of a given community. In so doing, this article suggests that historians can use measles data as a new biometric index for studying human health and socio-economic conditions in societies of the past.

I

This article explores the spatio-temporal structure of infectious diseases in modern Japan in the late nineteenth and early twentieth centuries, using measles mortality data from between 1900 and 1960. In so doing, it attempts to show that measles provides a new biometric index for studying the health status of human bodies and socio-economic conditions of societies in the past, and is a powerful tool that enables historians to analyse the patterns of exposure to infectious diseases. Historians are already familiar with a host of biological indices, which have been employed by socio-economic historians in the last couple of decades with impressive results. Birth and death rates, cause-specific mortality, morbidity, height, Body Mass Index (BMI), and many other indices have enormously enriched our understanding of the past.[2] This article attempts to show that measles data provide a unique insight which opens up new research directions and supplements those insights gained from more established biological indices.

The advantage of measles data as a biometric index lies in the infectious nature of the disease, which spreads from one person to another, and from one community to another community. Measles data highlight the route of infection between individuals, while biological indices hitherto employed are mainly concerned with the health status of individuals. Measles thus clarifies connections between com-

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[2] Literature on the history of health, disease, and death is now vast. For a selection of classic papers, see Rotberg, ed., *Health and disease*.

munities, whereas mortality, heights, and other familiar indices have been profitably used to study the health of communities. In other words, measles is a relational index, revealing the spatial relationships formed among different individuals and geographical units at certain times in the past. In addition to elucidating the spatial features through which infection took place, measles data also clarify temporal elements of a certain community. Features related to time, such as different patterns of seasonality and the synchronization of epidemics among different regions, play important roles in the following account. Being an index of spatial and temporal relationships between individuals and between communities, measles data reveal the spatio-temporal structure of a society in the past.

Much of the usefulness of measles as a biometric index is based on the biological characteristics of the disease. Measles is a simple disease, unlike many other infectious diseases which involve multiple factors and conditions. Indeed, Francis Black, an eminent epidemiologist and an expert biochemist of viruses, wrote that '[the] simplest of all infectious diseases is measles'.[3] Being a viral disease whose only known host is humans, measles spread from person to person primarily by droplet infection. This means that one only needs to think about the encounter between the virus and the person and there is no need to take other complicating factors into consideration, such as vectors or intermediary hosts. Neither does one need to worry about the specific characteristics of the susceptible: unlike diseases such as tuberculosis, measles does not depend upon the health status of the susceptibles. One infective person and one susceptible individual put in reasonably close proximity is often all that is necessary for measles infection to take place. In other words, the number of measles cases in a community is a reliable index of the extent of the community's exposure to the measles virus, or 'the frequency with which pathogens are encountered', to use Landers's apt phrase.[4] This simple infectious mechanism has long been recognized as a great merit within the discipline of epidemiology, where measles has long been the 'model' disease which elicits data most suitable for building a mathematical model for the mechanism of infection.

Another major merit of using measles data as a tool for historical research is that the disease used to be extremely common in wide geographical regions for quite a long period. Its relatively clear clinical manifestations are another merit, for one can be reasonably confident about the accuracy of diagnosis made in the past. Hirsch was thus able to claim that measles was in all probability widely diffused in Asia, Europe, and some parts of Africa by the middle ages.[5] The disease was extended to the Americas in the sixteenth century and to Oceania in the nineteenth century. From then on, measles became a common feature of life virtually all over the world until the 1960s, when the number of cases in developed countries started to plummet due to large-scale programmes of vaccination.[6] In Japan, the disease had been a frequent visitor from overseas since the tenth century at the latest and became endemic relatively late, in the late nineteenth century. In the late nineteenth and early twentieth centuries, the disease caused a considerable

[3] Black, 'Measles', p. 297.
[4] Landers, *Death and the metropolis*, p. 13.
[5] Hirsch, *Hirsch's handbook*, vol. 1, pp. 154–70.
[6] Cliff, Haggett, and Smallman-Raynor, *Measles*.

number of deaths year after year all over the country. Unlike dramatic visitations by highly lethal infectious diseases, which reveal social structures only for limited periods and limited geographical regions, one can expect measles to provide long-term, constant, and reliable data for geographically wide regions.[7]

Measles thus has a simple mechanism of infection and immunity, and measles data is a widely applicable tool which can elicit clear-cut historical insights. The almost universal presence of the disease for a long historical period facilitates chronological analyses and geographical comparisons. These features of measles have attracted historians' attention for some time. McNeil and Crosby have emphasized the impact of measles upon the 'virgin soil' population in their large-scale narrative of the expansion of the frontier of civilization and the accompanying penetration of infectious diseases into every corner of the world, a process which Le Roy Ladurie called 'the unification of the globe by disease'.[8] From the 1980s onward, the periodicity of measles has attracted attention in the context of chaos theory in mathematics, and historical data of the disease have long been the staple material for the analysis of chaos in the natural world.[9] Some historians and historically-minded epidemiologists, most notably Duncan, Scott, and Duncan, have analysed measles mortality in various cities in seventeenth- and eighteenth-century England, employing sophisticated mathematical techniques and informative ecological perspectives.[10] Cliff, Haggett, and other medical geographers have provided masterful accounts of the spatial diffusion of measles both in terms of long-term global history and of the local structure of islands.[11]

Upon the basis of such studies, this article attempts to integrate epidemiological study of measles more fully into social and economic historical perspective.[12] In order to do so, the diffusion of measles will be analysed on three levels, namely, regional, local, and domestic, which roughly correspond to three historical frames of reference, namely, regional integration, the creation of primary schools, and the changing composition of young members within a family. In the context of modern Japan (and of many other countries), these three historical phenomena were driven respectively by urbanization, the state's educational policy, and the people's behaviour in the private sphere. In other words, the hierarchical layers of the diffusion of measles were structured by three distinct but inter-related historical phenomena, each of which has been a major frame of analysis in social and economic history. By dividing the transmission of measles into the three spatial spheres above, I propose to match epidemiological features of the transmission of measles with subjects of enquiry in socio-economic history. In this way, this article develops the 'model' disease for epidemiologists as a biometric index for social and

[7] In this sense, the revelatory power of measles for historians is of a different kind from that of infectious diseases that provoked a major societal response in circumstances of crisis (the most famous being cholera in the nineteenth century). For historiographical discussion of the usefulness of cholera and similar epidemics, see the papers in Ranger and Slack, eds., *Epidemics and ideas*.

[8] McNeill, *Plagues and peoples*; Crosby, *Ecological imperialism*; Le Roy Ladurie, 'Concept', pp. 28–9. Modern studies of measles in a virgin soil population include Neel, Centerwalia, Chagnon, and Casey, 'Notes'; Christensen, Schmidt, Bang, Andersen, Jordal, and Jensen, 'Epidemic of measles'.

[9] For an example of this use of measles in mathematics, see Tidd, Olsen, and Schaffer, 'Case for chaos'.

[10] Duncan, Scott, and Duncan, 'Time series analysis'.

[11] See, inter alia, Cliff, Haggett, and Smallman-Raynor, *Measles*; Cliff, Haggett, Ord, and Versey, *Spatial diffusion*; Haggett, *Geographical structure of epidemics*.

[12] Landers, *Death and the metropolis*, pp. 199–350, makes a similar attempt to use records of smallpox in London.

economic historians to investigate urbanization, education policy, and families' reproductive behaviour.

Some explanation will help to clarify the concept of the layers of the infection of measles employed in this paper. Firstly, measles spreads from one region to another. The term 'regions' can refer to communities of various sizes, but this analysis will focus mainly on prefectures and cities. The spread of measles from one region to another depends on the chance of one infective transmitting the disease to a susceptible in other regions. The chance becomes greater when the number of infectives and/or that of susceptibles are large, and the chance of their meeting is great. Regions with large populations, with a heavy traffic of people between them, form the space in which measles spread easily, while isolated regions with small populations were less likely to be affected by measles. It naturally follows that the growth of cities and concomitant development of traffic around them facilitated the transmission of measles between regions. Urbanization thus affects the regional epidemiology of measles, due to its concentration of a large number of people and the increase in the connectivity between people living in and around the city.

Secondly, within one city, town, or village, measles radiated from those places where a large number of infectives and susceptibles congregated. This hub of infection was the primary school in the context of modern Japan, and thus measles established itself as a children's disease. A series of laws from the 1870s established a nationwide compulsory primary education system for children over six years of age, and by 1905 enrolment, if not attendance, was virtually universal.[13] The creation of the primary school system had a profound impact upon the diffusion of measles in local communities.

Thirdly, measles was transmitted from one child to another within a family. As a highly infectious airborne disease, measles showed a high degree of family aggregation: from one index case in a family, a further case or cases regularly resulted in the same household. The number of susceptible children in a family was thus crucial for the domestic multiplication of the disease. The change in fertility rate, and the reduction of the number of young children per family thus influenced the transmission of measles within the family. The changing composition of the family and its young members influenced the mortality of measles, since the disease is more likely to prove fatal in younger children.

A few words are in order about the sources utilized throughout this article. The main bulk of material is provided by the *Imperial cause of death statistics*, which included monthly numbers of deaths from measles in 47 prefectures and varying numbers of cities during the period between 1906 and 1933. For monthly deaths in the postwar period (1950–9) for 46 prefectures (excluding Okinawa, which was then under American rule) and six cities, handwritten tables held at the Statistical Archive of the Ministry of Health, Labour, and Welfare were consulted. For the periods 1900–5 and 1937–42, only annual numbers of deaths from measles for 47 prefectures and several cities were available in the *Eiseikyoku Nenpō* and *Eisei*

[13] Kindergartens and nurseries, which later had a great impact on children's infectious diseases, made only a negligible impact during the period under consideration. They were very small in number and were mainly found in large cities. In 1905, there were only 240 of them nationally. Enrolment in kindergartens was less than 10% in as late as 1950.

Nenpō.[14] These core data have been supplemented with small-scale datasets included in articles based on contemporary observation that have been published in medical journals.[15]

It should be noted that the main data are those of the *mortality* of measles. Until 1947, measles was not one of the infectious diseases that, by law, had to be reported, and so its morbidity data were available only in some exceptional instances for much of the period under examination. The argument put forward in this article often uses mortality as a substitute index for morbidity, and such reliance on mortality data creates some potential pitfalls, for death and illness were two distinct historical concepts.[16] It is hoped, however, that this analysis largely bypasses such pitfalls, because it concentrates on clearly visible patterns of the increase or decrease in deaths, instead of the actual figures of mortality, except in section V below. Moreover, Japanese epidemiologists at that time expressed their confidence in using the mortality of measles as a substitute for its morbidity/incidence, from observations of the close parallel of the two indices in other countries.[17] Likewise, none of the leading Japanese epidemiologists in the earlier half of the twentieth century expressed any serious doubt about the quality of data on measles mortality.

II

Before embarking on an analysis of the three layers of infection, it is necessary to establish when measles became endemic in Japan, since the Japanese case of measles' endemicity reveals that the mechanism of endemicity is much more social than hitherto assumed by epidemiologists and historians. The concept of endemic threshold or critical population size was articulated in the 1950s by leading epidemiologists such as M. S. Bartlett and Francis Black.[18] The argument goes as follows. Once a person is infected with measles, he or she acquires lifetime immunity or dies. He or she is no longer part of the susceptible population. The susceptible population thus becomes smaller as the epidemic proceeds. In small communities, epidemics of measles and other infectious diseases 'burn out', because towards the end of the epidemic the measles virus can no longer find a new human host, which is the only known type of host for the virus. On the other hand, large communities can sustain the disease without such fade-outs, because such communities may provide a constant supply of new susceptibles, mainly in the form of newborn babies or immigrants. When the population size of a community passes the critical threshold, measles thus becomes endemic. The critical question is thus whether the community is large enough to provide a constant supply of susceptible individuals. Observing the weekly incidence of measles cases

[14] *Nihon Teikoku Siin Toukei*; Kōsei rōdōshō hozon hyō (manuscript tabs. preserved at the Ministry of Health, Welfare, and Labour, Kasumigaseki, Tokyo); *Eiseikyoku Nenpō*; *Eisei Nenpō*.

[15] The datasets are those of age at infection and are taken from Sumiya, 'Joshi no mashin rikanritsu to nenrei tono kankei'; Ogawa, 'Mashin no jōzai ryūkōchi ni kkeru ekigakuteki tokusei ni nansuru kansatsu'; Takahashi, 'Mashin no riron ekigakuteki kenkyū 3'; Hedrich, 'Monthly estimates'; Wilson, Bennet, Allen, and Worcester, 'Measles and scarlet fever'.

[16] For an exploration of the historiographical implications of the distinction between disease and death, see Riley, *Sick, not dead*, pp. 1–23, 269–74.

[17] See, for example, Kubo, 'Mashin ryūkō no toukeigakuteki kenkyū'.

[18] Bartlett, 'Measles periodicity'; idem, 'Critical community size'; Black, 'Measles endemicity'.

in towns of various population sizes in the UK and the USA in the twentieth century, Bartlett has found that while in large cities cases of measles appeared continuously, in smaller cities measles 'faded out' and were reintroduced from outside. Based on such data, Bartlett calculated the threshold population size at around 200,000–400,000.[19] McNeill picked up this concept and set it in the context of his far-reaching historical framework of civilization and infectious diseases, whose core idea is that the growth of large cities was essential for the establishment of acute infectious diseases in human population. Relying on the works of Bartlett and other epidemiologists, McNeill maintains that a population size of about 300,000–400,000 is the critical threshold of endemicity of measles. When human society and civilization were able to support such a large population of people, it began to harbour measles within it, and these epidemics often diffused to communities of smaller size and devastated their virgin-soil populations.[20]

The general outline of McNeill's argument is sound and solid. However, the actual figure for the threshold population size is highly problematic. Bartlett pointed out that the figure for the critical threshold was flexible. McNeill was well aware that Bartlett's figure is drawn from modern urban societies in the early twentieth century and is subject to change, according to societal factors such as birth rates, patterns of life, and customs that influenced the rate of infection. The flexibility of the threshold figure through such factors was, however, much larger than McNeill seems to have allowed.

Measles in early modern Japan provides one striking example of such flexibility. The endemic threshold in Tokugawa Japan far exceeded the figure calculated by Bartlett and adopted by McNeill. The country had three major cities, all of which exceeded Bartlett's threshold population size. In the eighteenth century, Kyoto had a population of around 300,000 and Osaka had around 500,000, while one million lived in Edo (Tokyo), perhaps the largest city in the world. Kyoto reached the threshold population size calculated by Bartlett, and Osaka and Edo easily exceeded it. However, both descriptive and statistical evidence demonstrate beyond reasonable doubt that measles failed to settle itself as an endemic disease in either city, as Jannetta has pointed out.[21] Despite the presence of the three large cities, each of which reached or exceeded the population threshold calculated by McNeill and Bartlett, measles in Japan had remained a disease that was imported from abroad, engulfed the nation with intervals of 10 to 20 years between epidemics, and died out without becoming endemic in any of these cities. Jannetta regarded it as puzzling that eighteenth-century London, with its population of about half a million, achieved endemicity for measles, while Edo failed to do so despite having double the number of inhabitants. Likewise, the establishment of measles in Boston and Philadelphia in the early nineteenth century, which were cities of considerably smaller population size than Edo, suggests that population by itself did not determine the threshold of endemicity, although European immigration to American cities might have played some role by increasing the frequency of reintroduction of the pathogen from the centre of endemicity.[22]

[19] Bartlett, 'Measles periodicity'; idem, 'Critical community size'.
[20] McNeill, *Plagues and peoples*, pp. 78–9. See also Black, 'Measles endemicity'.
[21] Jannetta, *Epidemics and mortality*. See also Fujikawa, *Nihon Shippei Shi*, pp. 169–209.
[22] See Caufield, 'Early measles epidemics'.

Jannetta is certainly right in maintaining that the sea that surrounds Japan acted as a powerful cordon sanitaire and that *sakoku* (the Tokugawa Bakufu's strict regulation of the trade and human movement between Japan and other countries) protected its population from foreign infectious diseases to a considerable extent.[23] The isolation policy, however, did not explain the failure of measles to establish itself *after it entered Japan*. Indeed, the example of measles in Japan is less a mystery than a reminder of the flexibility of the threshold population size: societal factors, not just the size of population, are crucial for endemicity. As Bartlett and McNeill have already indicated, the threshold was dependent on such societal factors as fertility, population density, the ways in which cities were designed and built, and the patterns of sociability that determined how people met each other. At the moment, no conclusive explanation has been offered as to why a country with a densely packed population of 30 million and with one of the largest cities in the world as its capital repeatedly failed to establish endemic measles. Some speculation is, however, possible, which directs our attention to social institutions for gathering people. For measles to become endemic, it is necessary that the chain of infection is sustained when the epidemic is about to end. Towards the end of an epidemic, many residents have already been affected by the disease and become immune to the infection. The number of infectives has also become smaller. Some societal factors are necessary to sustain the tenuous chain of infection at this stage of the epidemic. The crux of the issue is whether babies or very young children, who are likely to be susceptible to the disease, meet infective individuals. It is likely that the church, where people regularly brought their young family members with them, including those who had been born recently and were still susceptible, acted as the space to sustain the infection. While in London and other cities in Europe churches were a common feature of society and a large number of parishioners regularly brought their very young children to them, Edo did not have a well-established institution with a comparable social and epidemiological function. Perhaps it did not have a social fabric which mixed those who had been born recently with residents of the wider community beyond the immediate neighborhood. This means that the infectives in Edo at a later phase of an epidemic outbreak were less likely to meet susceptibles to whom they could transmit the virus, however large the size of the city's population was. Early modern Japanese cities lacked an institutional social fabric that regularly brought the infectives and susceptibles into close contact, while communities in eighteenth-century London and other European cities possessed such a fabric in the form of churches. This explanation is, however, at present purely speculative and needs an international comparative study of measles endemicity for either proof or disproof.

One of the last non-endemic outbreaks of measles in Tokyo took place in 1885, 11 years after the epidemic of 1874. The outbreak in 1885 showed every sign of being an epidemic imported from abroad, as was the case with earlier outbreaks in the previous two centuries, confirming that the epidemiological pattern discussed by Jannetta persisted into the early Meiji period. The disease was almost certainly imported from the western part of Japan and rapidly moved eastward, with some

[23] Jannetta, *Epidemics and mortality*, p. 144.

time lag due to the eastern region's lack of accessibility.[24] Nagasaki, a major port and the main access point for imported infectious diseases for the last few centuries, saw an epidemic of measles, which resulted in 2,156 officially reported cases of measles between November 1884 and January 1885. At the end of January, the outbreak moved eastward and raged in Kobe, where 100 cases a day were reported. In early February, it moved farther east and started to take on the appearance of an epidemic in Yokohama City in Kanagawa Prefecture, where the emergency committee met and discussed anti-epidemic measures. By the end of February, Yokohama was experiencing more than 400 new cases a week. Tokyo saw sporadic appearances of cases from the end of January, and the peak of the epidemic wave was in mid-March. On 12 March, 1,337 cases were reported.

Within Tokyo, it is possible to reconstruct the diffusion of measles on detailed maps (figure 1).[25] The maps clearly show a concentric diffusion. The disease was first established in Nihonbashi Ward, the most central and densely populated area, and the heart of commerce in Tokyo. It radiated from the centre to its periphery, and thereafter waned in the central part. This diffusion pattern also shows that the 15 wards of the City of Tokyo were closely connected with each other in terms of the transmission of measles. Indeed, since susceptibles had been accumulating for 11 years, the diffusion of the disease was rapid, completing the epidemic wave in about two months; in later years, it often took about 10 months for one epidemic wave to complete itself. The outbreak also reached the surrounding rural areas. There, reports of cases were sporadic, but they still look like an epidemic wave when aggregated, which reiterates the importance of the diffusion from the centre to its peripheries.

One of the most important features of the measles epidemic in the Meiji period was that measles became endemic in Tokyo toward the end of the 1890s. A brief note in a medical journal of the measles epidemic of 1897 suggests that the outbreak had occurred after a 12-year gap.[26] Perhaps shortly after this outbreak, measles established itself in Tokyo.

III

The measles epidemic of 1885 seems to have created havoc all over the country, as can be gleaned from the perusal of numerous reports issued by local governments. There is little data about the disease for the rest of the nineteenth century until 1900, when the systematic collection of prefectural mortality data started. In the intervening 15 years, the epidemiological outlook of measles changed drastically: nationwide epidemics, which had swept the entire country in a single wave from the south-western part of the country to the north-eastern part, were no longer observable. With the establishment of the endemic centres of measles and the subsequent shortening of the hiatus between epidemics, there was no longer an

[24] The account of the eastward diffusion of measles is reconstructed from reports of the disease which appeared in *Yomiuri Shinbun*, 24 Jan. 1885–5 May 1885, particularly 24 Jan. 1885, 28 Jan. 1885, 21 Feb. 1885, 10 March 1885, 28 March 1885. Regional details of the diffusion can be glimpsed in prefectural reports, such as Shimane's *Eisei Tsuho*, pp. 82–4.

[25] The data for the map are taken from the daily reports of the number of patients in *Yomiuri Shinbun*, 12 Feb. 1885–12 April 1885.

[26] Anon., 'Mashin no shūrai'.

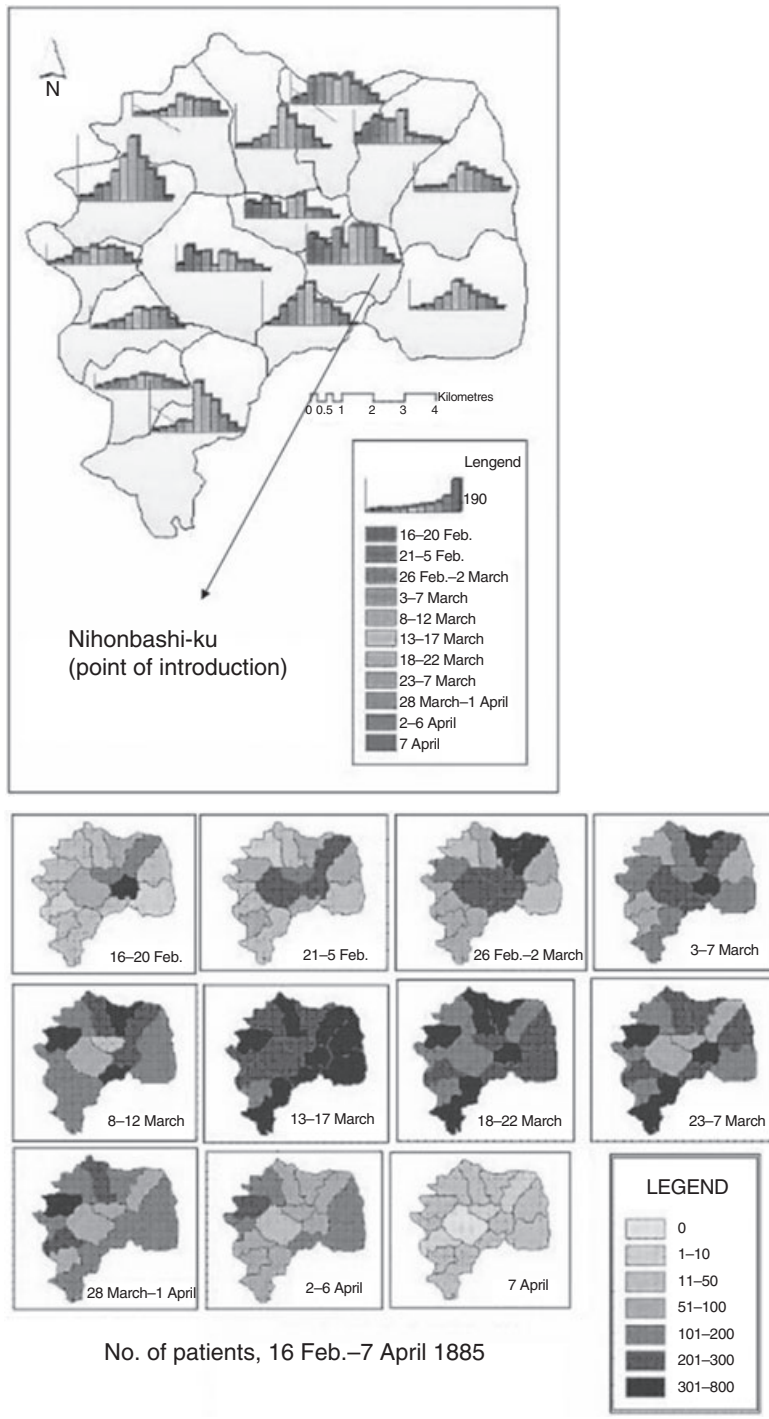

Figure 1. *Number of measles patients in 15 districts of Tokyo, 1885*
Sources: Yomiuri Shinbun [*Yomiuri Newspaper*], Feb.–April 1885.

Table 1. *Measles mortality (per 1000), Tokyo, Osaka, Kōchi, and Okinawa, 1900–10*

	Tokyo	*Osaka*	*Kōchi*	*Okinawa*
1900	9.2	5.8	1.1	0.0
1901	5.3	1.6	1.1	0.0
1902	5.4	3.5	1.8	0.2
1903	1.4	0.4	1.9	0.2
1904	4.6	1.6	0.2	0.0
1905	10.6	3.3	2.7	5.8
1906	4.5	2.1	5.5	15.3
1907	8.6	6.1	0.8	0.6
1908	5.4	2.6	0.6	0.0
1909	11.6	7.4	6.1	0.0
1910	3.0	1.8	23.6	0.0

Source: Eiseikyoku Nenpō.

abundant number of susceptibles that would have enabled an outbreak to achieve a nationwide sweep with a single stroke. Instead, regional differences in the epidemic pattern became manifest.

Table 1 represents the mortality of measles from 1900 to 1910 for four prefectures (table 1). There were two urban areas where measles became endemic, namely, Tokyo and Osaka. These two prefectures with large cities had already exhibited a rhythm of epidemic cycles, which was becoming shorter and more manifest, with high waves and strict regularity. In the 1910s, the two-year cycle had become prominent in both prefectures. Clearly the measles virus had been endemic in the two large cities of Tokyo and Osaka, whose populations in 1900 were about 1.5 million and 0.88 million respectively. Among Japanese prefectures at that time, Tokyo and Osaka were exceptionally urbanized, and the residents of the metropolises comprised respectively 70 per cent and 52 per cent of the prefectural population. Numerous mathematical models of measles infection have demonstrated that its periodicity is greatly influenced by the size of community and the speed with which susceptibles are newly supplied to the community.[27] Accordingly, Tokyo and Osaka were the first prefectures that came to exhibit the regular and the shortest cycle of two years between outbreaks.

Two prefectures on the periphery, on the other hand, show very different patterns from those of Tokyo and Osaka. Kōchi, isolated by steep mountains from the other three prefectures in Shikoku Island, had a pattern with sporadic and pointed peaks, reminiscent of earlier imported epidemics. Okinawa, a prefecture that is separated by sea from mainland Japan and certainly the most isolated of Japanese prefectures, had one major outbreak in the year 1905/6. For the rest of the years, measles was almost completely absent.[28] Between those two extremes of central Tokyo and Osaka and peripheral Kōchi and Okinawa, there were many intermediate prefectures which exhibited irregular but relatively frequent epidemic

[27] Scott and Duncan, *Human demography and disease*. Scott and Duncan and others have argued for the importance of nutrition, suggesting that measles periodicity was linked to the cyclic movement of wheat prices. There is, however, another possible explanation for the synchronicity of the measles epidemic and wheat prices: scarcity of wheat forced people to increase their mobility in order to search for resources, which is a phenomenon often observed in times of famine. See also Duncan, Duncan, and Scott, 'Dynamics of measles epidemics'.

[28] On measles epidemics in Okinawa, see Inafuku, *Okinawa shippei shi*, pp. 226–44.

peaks. Some other rural prefectures, such as Kagawa, had a regular and stable three-year cycle.[29] In short, in the late nineteenth and early twentieth centuries, divergences appeared in the epidemic pattern of measles among regions, in contrast to its former uniformity. In other words, different regions experienced the epidemic with different frequencies.

The situation was comparable to what many historical epidemiologists and demographers have found in different countries and time frames.[30] Using data on smallpox mortality in England in the seventeenth and eighteenth centuries, Duncan, Scott, and Duncan have found that large conurbations such as London harbored endemic smallpox which flared up into a major outbreak every two years, while medium-sized rural towns saw outbreaks every five years, which was the amount of time necessary to build up a susceptible population that could allow an epidemic to occur.[31] The epidemiology of twentieth-century Japan was, however, markedly different from that of early modern England: while in the latter the contrast in periodicity between urban and rural regions was relatively stable and persisted at least for 150 years, the former witnessed a rapid change in less than 30 years. It is therefore necessary to examine some aspects of the rapid restructuring of the spatial diffusion of measles in Japan in the early twentieth century.

Within one prefecture, a large city epidemiologically integrated its peripheries, which is discernible by the comparison of the rhythms of outbreaks in the regions of the prefecture. Figure 2 shows the number of deaths from measles in the City of Osaka and surrounding districts from 1914 to 1933.[32] The figure shows a progressive epidemiological integration of the peripheries by the City of Osaka. By 1914, Higashinari and Nishinari, two districts which were adjacent to the old city area of Osaka, had already been integrated into the epidemiological system of Osaka City: the mortality of the measles of the two districts synchronized with the two-year cycle of the metropolis. Mortalities of the remoter districts remained uncorrelated during the 1910s, showing irregular increases and decreases. By the late 1920s and early 1930s, however, all districts within Osaka Prefecture fell into the dominant rhythm of the City of Osaka.[33] The growth of industry and the congregation of workers' families in and around the City of Osaka must have contributed to the epidemiological regional integration.

This synchronization does not mean that every district in Osaka Prefecture was urbanized, nor does it mean that every remote village in the peripheral counties had measles outbreaks every two years. It should be borne in mind that the regularity of the epidemic wave of counties around Osaka City was the effect of

[29] One epidemiologist classified epidemic patterns of 46 prefectures into six groups. There is an obvious contrast between those prefectures containing large cities (Tokyo, Osaka, and Kanagawa) and rural prefectures. See Suwa, 'Mashin no ekigaku hoi'.

[30] In London between 1647 and 1837, the inter-epidemic interval of measles gradually shortened from five years, to two, to three years; Duncan et al., 'Dynamics of measles epidemics'.

[31] Duncan, Scott, and Duncan, 'Modelling the different smallpox epidemics'.

[32] Satomi, 'Osaka fuka ni okeru mashin shibō'.

[33] The synchronization of the measles mortality of a metropolis and its surrounding regions does not mean that every village or settlement in the surrounding regions exhibited the regular periodicity of the metropolis. A county (*gun*) covered a wide area, containing many villages and settlements, some of which were highly isolated and hence experienced measles outbreaks only sporadically. The growth of population in core towns within a county connected to the metropolis might have contributed to the periodicity when aggregated. See Takahashi, 'Mashin no riron ekigakuteki kenkyū 3'.

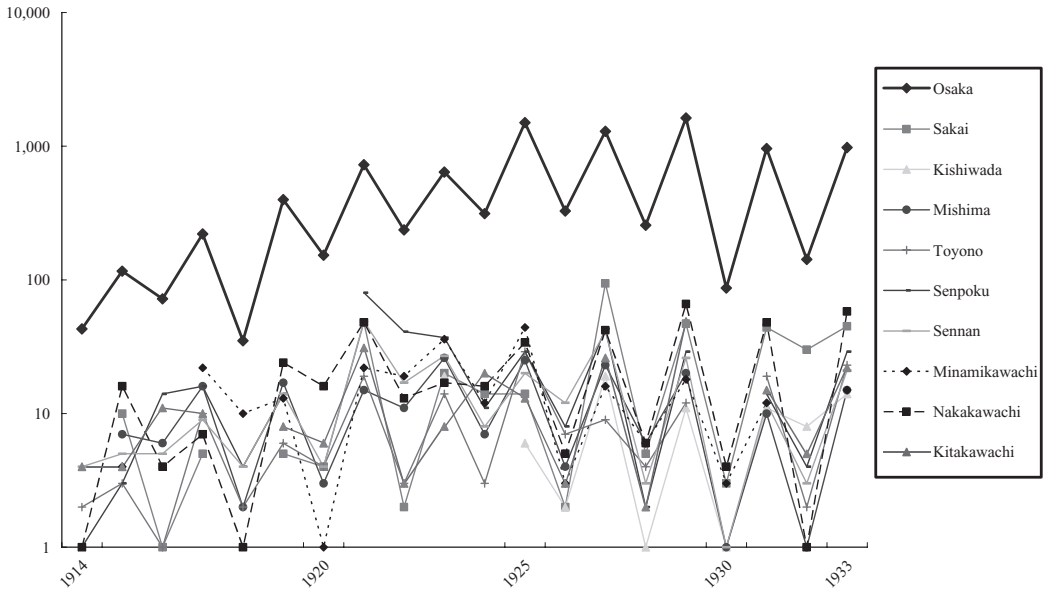

Figure 2. *Measles deaths in the city of Osaka (top) and neighbouring cities and counties*

Source: Osaka Prefecture, *Osakafu Tōkeisho* [*Statistical Report of Osaka Prefecture*], (1914–33).

the aggregation of epidemics in villages and towns.[34] Although small towns in the districts were experiencing growth, many regions in Osaka still remained rural in their general outlook in the 1930s. Nevertheless, the synchronization meant that those rural areas that could not by themselves sustain endemic measles were integrated into the 'near population' of the central metropolis, where measles was endemic and had outbreaks every two years.[35] Thus, in a sense, we can observe the unification of Osaka Prefecture by a viral disease.[36]

The City of Osaka and its surrounding regions were epidemiologically integrated through the progress of urbanization. Those semi-rural areas within the regime of the City of Osaka were exposed to the urban rhythm of measles epidemics. Although the precise dynamics of such integration remain to be investigated, its epidemiological mechanism is fairly clear: the development of corridors which connected the metropolitan centre and surrounding regions and the growth of the population both at the centre and in its environs played a crucial role. Population growth increased the number of infectives and susceptibles, and improved transportation increased the chance of an encounter between the two groups. The rapid population growth of the City of Osaka and its surrounding regions and the construction of roads and railways contributed to regional inte-

[34] The effect of aggregation in forming a regular wave of measles epidemics was shown in a paper based on a detailed survey of 31 towns and villages in an area in Saitama Prefecture. See Yosano, Mori, and Shōnari, 'Mashin no chiriteki ekigaku'.

[35] Epidemiologists were well aware that relatively remote and rural areas exhibited an 'apparent endemicity'. Ueki, Horiuchi, and Sugiyama, 'Mashin no ryūkō ni kansuru ekigakuteki kōsatsu'.

[36] The much larger phenomenon of the unification of the globe by microbes was famously discussed by Le Roy Ladurie, 'Concept'.

gration in terms of measles epidemics. Similar phenomena of the epidemiological integration of urban peripheries was observed in other major cities. The City of Tokyo's integration of the rest of Tokyo Prefecture took place in the 1910s. Annual numbers of deaths from measles in Yokohama City and the rest of Kanagawa Prefecture started to synchronize around 1930, and those in Kobe with the rest of Hyogo Prefecture in the mid-1930s.[37] The major cities created around them an epidemiologically homogeneous spatial unit, nearly every part of which had the same annual rhythm, following the rhythm of the centre.

The dominant rhythm of the Cities of Tokyo and Osaka extended beyond their respective prefectural borders and reached surrounding prefectures. From the 1920s, such inter-prefectural integration became visible around Tokyo and Osaka (figures 3a and b). Tokyo integrated Kanagawa from an early stage, and Kanagawa's dis-synchronization for five years between 1923 and 1928 was quickly corrected. By the mid-1930s, Tokyo brought into its epidemiological dominion the neighbouring prefectures of Chiba and Saitama, which were, generally speaking, still rural regions and were unlikely to achieve the large oscillation without Tokyo's influence. On the other hand, Yamanashi, another prefecture bordering on Tokyo, was not integrated during the period under consideration. This was almost certainly because the corridor that connected Tokyo and Yamanashi was underdeveloped and there was no chain of human habitation connecting the capital and the mountainous prefecture.

Likewise, Osaka integrated surrounding prefectures from the 1910s (figures 3c and d). There are some notable features in the process of the integration. Nara, which was connected to Osaka by railways, came under Osaka's epidemiological dominion from the 1910s. Ironically, relatively urban Hyogo turned out to be the major obstacle, for Hyogo's rhythm was dictated by the City of Kobe, which had a population of 700,000 in 1930 and had a two-year rhythm, which alternated with that of Osaka City between 1922 and 1928. In 1929, when Osaka had an exceptionally severe epidemic of measles, Kobe finally yielded. This episode confirms that the number of infectives played a crucial role in regional epidemiological integration. Afterwards, the City of Kobe and the prefecture of Hyogo remained in the epidemiological unit of Osaka. The growth of industrial and residential cities and towns that lay on the corridor between Osaka and Kobe in the 1920s must have contributed to the conditions that made this integration possible. Thus by the 1930s, the City of Osaka dictated the epidemiological rhythm of the three prefectures of Osaka, Nara, and Hyogo. Among other prefectures that bordered on Osaka, Wakayama, with its small population and relatively tenuous transport links with Osaka, remained outside its epidemiological unit. Urban Kyoto, somewhat like Kobe between 1922 and 1928, retained its distinct three-year cycle of measles epidemics and maintained its glorious isolation.[38]

Concentration of a large number of people in large cities, and the development of transport links between those centres and their peripheries, had profound epidemiological impacts. Large cities, particularly Tokyo and Osaka, moulded the

[37] For reasons that are not clear, Nagoya did not bring its peripheries into stable and long-term synchronicity during the period under examination.

[38] The cycle of three years was not exclusive to Kyoto. The city of Dairen in China exhibited a regular three-year cycle of measles epidemics between 1912 and 1932. See Morita, 'Dairen ni okeru mashin no toukeiteki kansatu'.

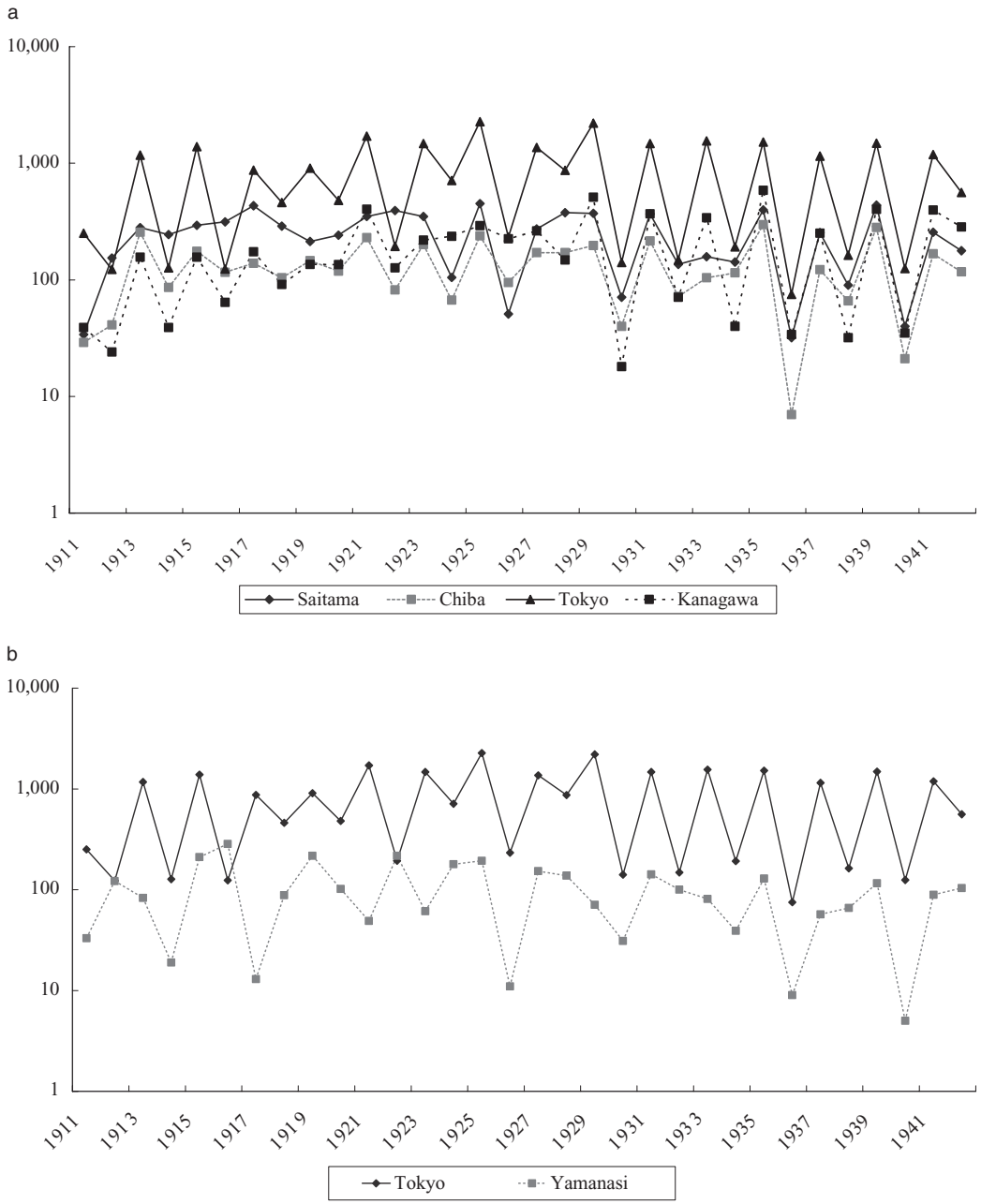

Figure 3. *(a) Number of measles deaths, 1911–1942: Tokyo, Sataiama, Chiba, and Kanagawa*
(b) Measles deaths: Tokyo and Yamanashi
(c) Measles deaths: Osaka, Hyogo, and Nara
(d) Measles deaths: Osaka, Kyoto, and Wakayama
Sources: Eiseikyoku Nenpō; *Eisei Nenpō.*

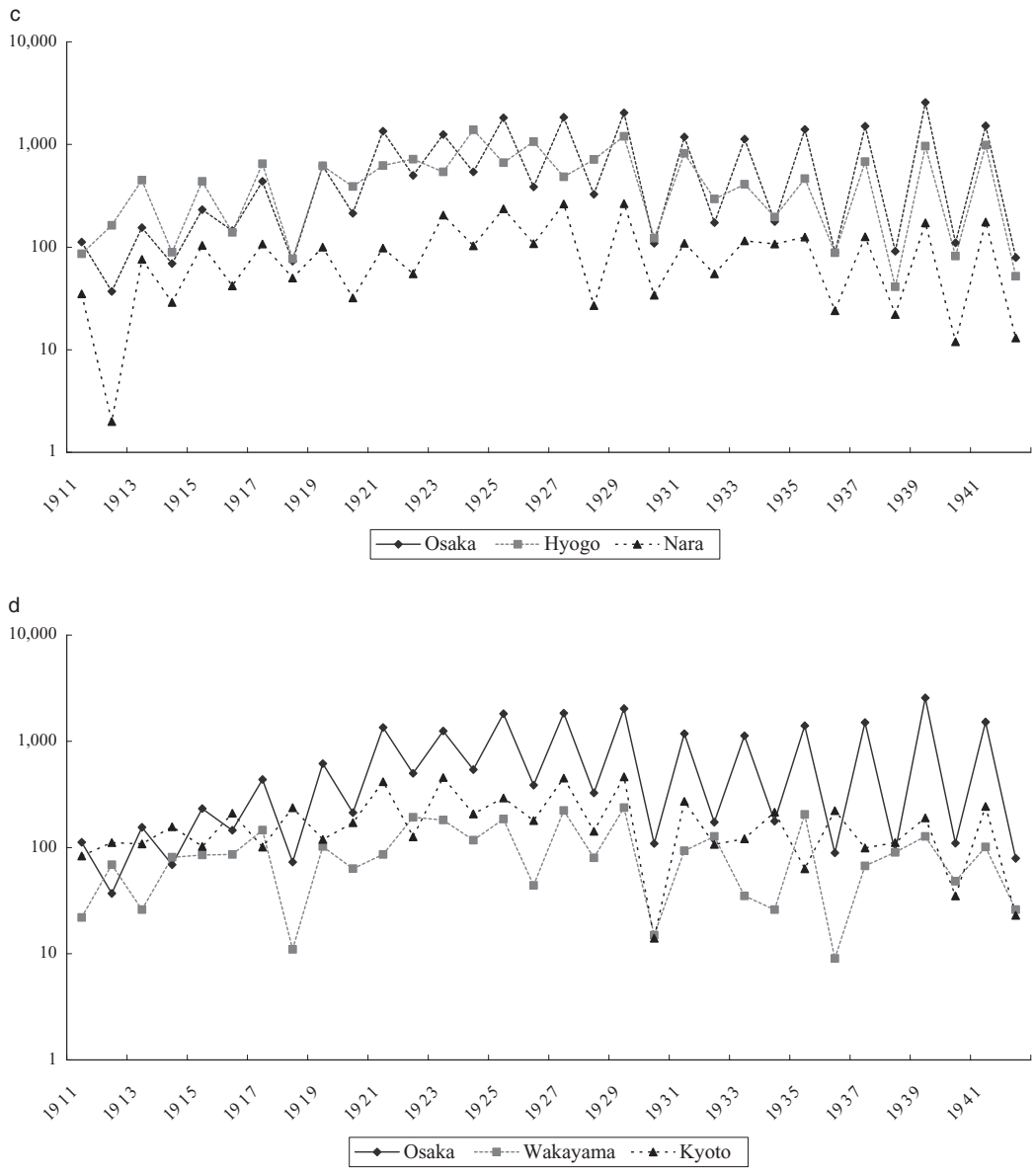

Figure 3. *Continued*

spatial-temporal structure of measles epidemics. The metropolises dictated the temporal rhythm of outbreaks of measles within a wider region beyond their city or prefectural borders. Areas which fell into the rhythm of two metropolises included rural prefectures (Chiba, Saitama, and Nara), as well as urbanized prefectures (Kanagawa and Hyogo), which, interestingly, sometimes showed resistance to the dominion of the two metropolises due to the distinct rhythm of their own major cities. The incorporation of the former category of prefecture into the metropolitan epidemiological unit suggests that closeness to a metropolis where measles was endemic mattered more than the actual extent of urbanization in the

region. A person who lived in a relatively rural part of Osaka prefecture experienced more frequent outbreaks of measles than one who lived in the city centre of Kyoto or other major cities.

Although the above account has emphasized the epidemiological regional integration led by large cities, there are instances in which no single centre of integration can be specified. Rural prefectures did not normally contain a large city that was an overarching epidemiological centre dominating its sub-regions, but the annual pattern of measles mortality in rural prefectures was not irregular. Their sub-regions might not be centrally integrated, but their cities and towns were connected with each other. One example of this type of epidemiological regularity is the case of Kagawa, mentioned above, which did not have any major city but still kept regular and clearly discernible three-year periodicity. Almost certainly, the seemingly urban pattern in a prefecture without a major city was related to Kagawa's geographical structure, namely, the concentration of towns in a relatively small area without any major geographical dividing lines.

IV

So far this article has analysed the epidemiological integration of regions, namely, cities and prefectures. Measles data are also useful for studying the integration of geographical areas of much smaller sizes, such as communities of a village or a town. In this context, schools played a crucial role in the Japanese experience of measles.

Places that drew together a large number of people were obvious hubs of infection. For infectious diseases of childhood such as measles, primary schools were an obvious site for the transmission of the disease. International studies of seasonal patterns of measles confirm the importance of schools: a study of England in 1950–79 has demonstrated that the transmission rate rose at the beginning of school terms.[39] In Japan since 1897, measles was one of the specially designated 'school diseases', which meant that children suffering from it were prohibited from attending school.[40] One local study of an outbreak in both the primary school and the kindergarten in a remote village in Fukushima Prefecture reveals the impact of the closure of the hub of infection: when the primary school closed for 10 days for a mid-term holiday, the chain of infection was interrupted and the number of infected individuals of school-attending age plummeted. In contrast, those who attended the kindergarten, which stayed open during the period, did not witness such an interruption.[41]

The impact of the primary school upon the epidemiology of measles in Japan is best exemplified by one field survey of an outbreak, which was conducted in 1954 in a rural village of Kinushima in Tochigi Prefecture.[42] The village consisted of eight settlements and had a primary school at its centre (figure 4). Some of these settlements were far apart (three kilometres). The first case of measles took place on 2 August during the summer holiday (figure 4a). The schoolboy caught the

[39] Fine and Clarkson, 'Measles in England and Wales'.
[40] Ministry of Health, *Gakkō Hoken Hyakunen-shi*, pp. 89–91.
[41] Wakabayashi, 'Mashin no ekigakuteki kenkyu IV'.
[42] Kunimi, 'Nōson gakudō ni okeru mashin ryūkō'.

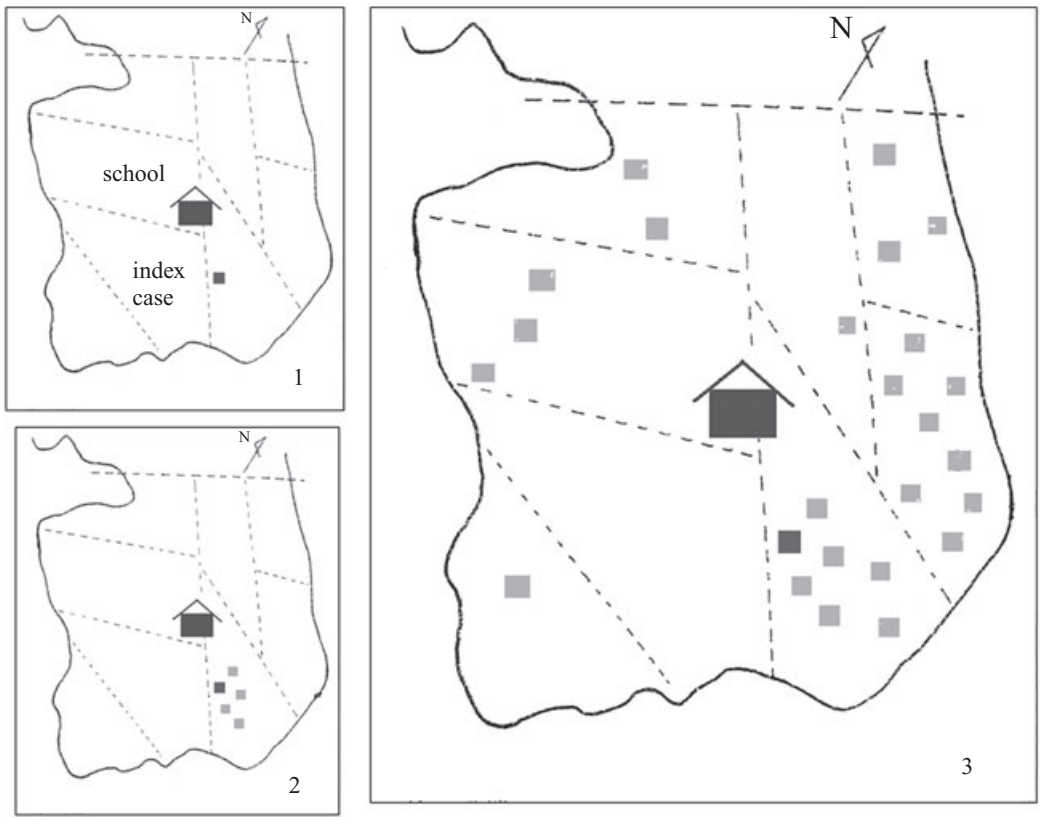

Figure 4. *Diffusion of measles among schoolchildren of a village in Tochigi Prefecture in 1954*

Source: Kunimi, 'Nōson gakkō ni okeru mashin ryūkō'.

disease when he visited another town. Three subsequent cases, both residing in the same settlement as the index case, appeared in the next three weeks (figure 4b). When the school opened on 21 August, one pupil who was still infective attended the school. As a result, a fierce epidemic broke out and infected all the remaining 20 susceptible schoolchildren in the village in four weeks, sweeping seven out of eight settlements (figure 4c). The impact of the school is obvious in this case: during its closure, the index case infected three children in three weeks, while in term-time the single remaining infective case managed to infect 20 children in four weeks. This example also shows that the school did not just multiply the cases of measles, but also diffused the disease geographically. Particularly in rural areas, where the population was sparsely distributed, schools acted as an important epidemiological bridge between distant settlements. Without the school, the measles virus was much less likely to find a host residing in a distant settlement within a village. One epidemiological survey of an explosive outbreak of measles in a remote village on Shikine Island in 1939 revealed that even with the accumulation of susceptible cases 12 years after the previous epidemic, the disease did not diffuse from the residential house of the index case, but used the primary school

as the hub of infection.[43] It was the primary school that integrated settlements within a village and brought them into one epidemiological unit.

The impact of the opening and closing of schools can also be seen at a regional level, through mortality statistics. Figure 5a represents the epidemic wave of measles in Hokkaido in 1920–2. There is something unnatural in this wave: it seems that the epidemics that had started in late 1920 progressed until September 1921, when the epidemic was abruptly interrupted. The epidemic appears to have resumed its momentum in the next month, reached its second peak in November, and then waned. This splitting of the epidemic wave into two halves, so to speak, can be explained by the influence of the summer holiday that took place during August. (Summer closure of primary schools in Japan is typically between 20 July and 31 August, differing slightly between prefectures.) Using schools as the hub of infection, epidemics progressed during term-time, when a large number of children came into contact with each other at school and chains of infection were sustained. With the arrival of the summer holiday, the infection rate suddenly decreased, because each case could only now infect a substantially smaller number of children than during term-time. When the holiday started, the epidemic was thus halted and the number of cases plummeted, leading to a much smaller number of deaths in September. When the school reopened in September, it was likely that there remained a sufficient number of infective cases and still susceptible individuals. Mixing with one another at school, the remaining infectives and susceptibles resumed the epidemic in the subsequent months until the epidemic faded away. There are numerous similar examples, in which the shape of an epidemic wave suggests that a proceeding epidemic was interrupted in August/September.[44] Naturally, the seasonality of the national aggregate of measles mortality reached its lowest point in September. Some evidence from other countries shows a similar pattern. The seasonality of measles deaths in London and Paris between 1880 and 1910 shows a similar pattern, with the lowest point being in October.[45]

The summer interruption of measles epidemics was a phenomenon observed only in rural prefectures, and was absent in urban prefectures. Figure 5b represents the epidemic wave of monthly mortalities in Tokyo, showing epidemic and non-epidemic years appearing alternately. In epidemic years, a high and pointed peak appears in April or May, while in non-epidemic years the curves are very flat. In Tokyo, as well as in Osaka and other urban prefectures, this pattern was repeated very regularly during the period of observation, with no instance of interruption by the summer holiday as was observed in rural prefectures. The reason for the contrast in urban and rural regions is the speed of the diffusion of the disease.[46] Urban areas have a higher infection rate (the number of new cases that one patient can infect during the infectious period), because of their high population density, well-developed means of transport, heavy traffic of people beyond their neighbourhood,

[43] Kobayashi and Suehiro, 'Izu Shikine tō ni okeru mashin no ryūkōgakuteki chōsa'.

[44] Similar examples that show a sudden drop in mortality in Sept. include the epidemic waves of Hokkaido, 1926–8; Aomori, 1927–8; Tochigi, 1927–8; and Hiroshima, 1918–19.

[45] Brownlee, 'Investigation'.

[46] The speed of the progress of measles epidemics, measured by the length of an epidemic wave, is another index that has potential for historical study. For instance, in Tokyo in 1951, its central and densely populated area had an outbreak of measles that reached its peak in 11 weeks, while in the most rural area this took 21 weeks. See Suwa, 'Mashin no ekigaku hoi'.

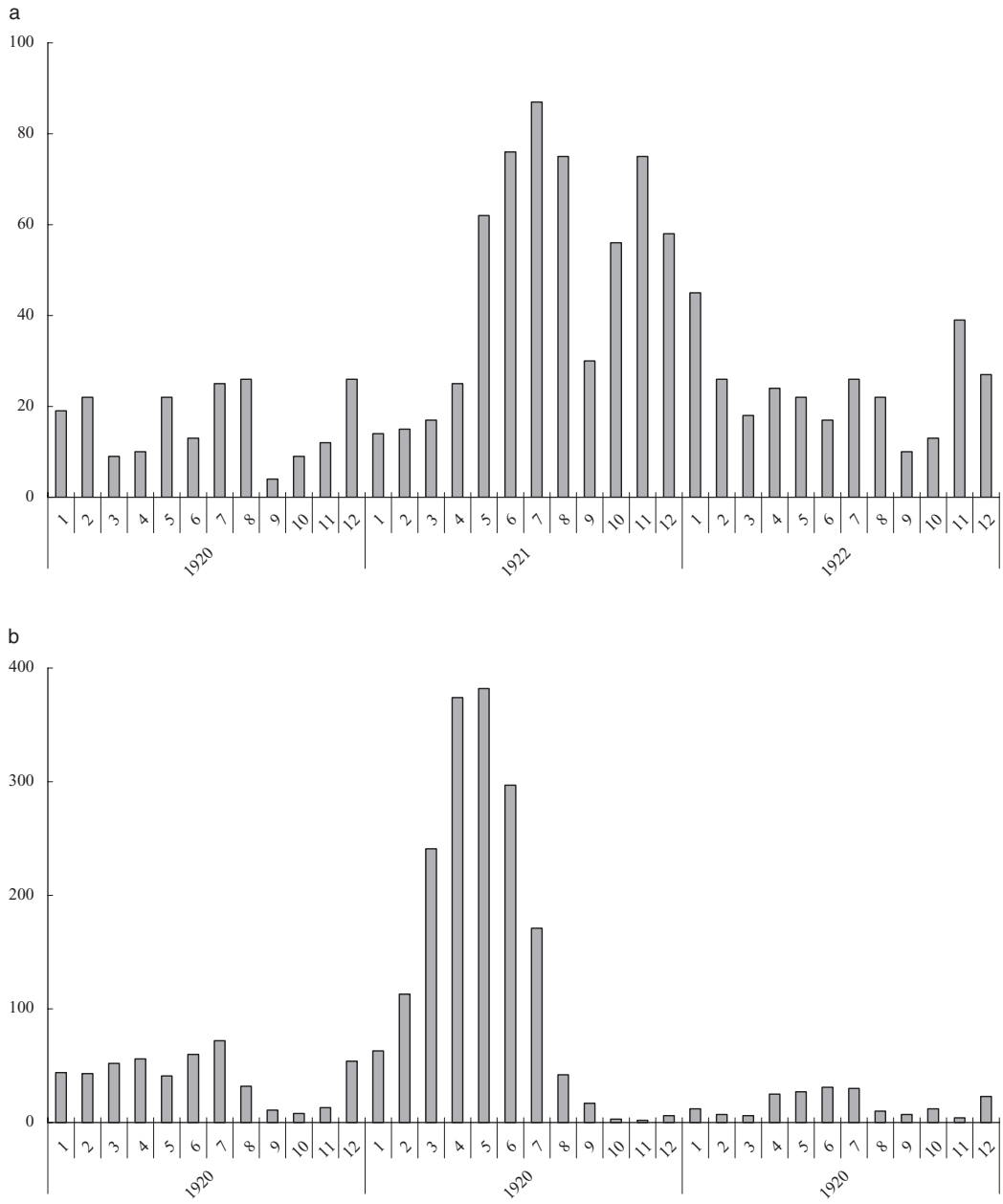

Figure 5. *(a) Monthly measles deaths, Hokkaido, 1920–2*
(b) Monthly measles deaths, Tokyo, 1920–2
Source: Nihon Teikoku Siin Tōkei.

and larger school sizes. An epidemic in an urban area thus progressed quickly, infected many susceptible individuals rapidly, and burned out in a shorter period. In urban prefectures the epidemic starts typically in December, peaks in April or May, and declines in June and July. When the summer holiday started in late July, the

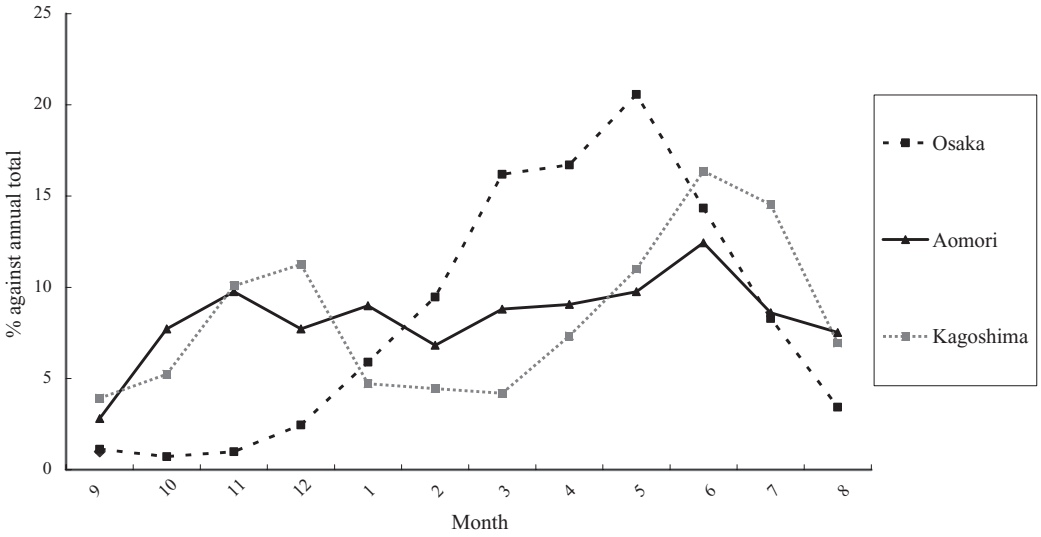

Figure 6. *Percentage of monthly measles deaths, urban and rural prefectures, 1911–20*
Source: Nihon Teikoku Siin Tōkei.

epidemic had already reached its natural end. By contrast, in rural prefectures the infection rate was lower, because of low population density, less well-connected subpopulations, less travel, and smaller school sizes. This meant that an epidemic in a rural prefecture progressed slowly, taking more time to come to an end. The rural epidemic waves were therefore more likely to be interrupted by the summer holiday.

One of the outcomes of the differential impact of the summer holiday is that urban and rural prefectures had different seasonal patterns of measles mortality. Figure 6 shows the percentages of monthly deaths of measles in urban and rural prefectures, aggregated for the period 1911–20. The seasonal pattern of urban Osaka and those of rural Aomori and Kagoshima are strikingly different. Seasonal mortality in Osaka had a single peak in the early-to-mid-spring months. Despite the difference in the general outlook, seasonal distributions of measles deaths in Aomori and Kagoshima are similar in that they both have twin peaks, one in winter and the other in early summer. From what has been discussed above, it is intuitively understood that the winter peak in rural prefectures was the product of the revival of epidemic waves after the summer holiday, while urban prefectures did not show any peak in winter because they could complete an epidemic wave before the summer holiday. In other words, the more urban a prefecture was, the smaller the winter peak, which was, so to speak, the leftovers of the epidemic from before the summer holiday. Figure 7 thus shows strong inverted correlation between population density and the percentage of deaths in October, November, and December. The further confirmation of the mechanism of the differentiation of single- or double-peaked seasonality is that urban centres in rural prefectures showed a single-peaked seasonality curve while the rest of the prefecture showed twin peaks. Table 2 shows patterns of differing seasonality in three rural prefectures and their respective capitals. While the three prefectures show typically rural twin-peaked seasonality, their capitals show the pointed single peak, a characteristic of urban regions with speedy diffusion of the disease. Moreover, the seasonal

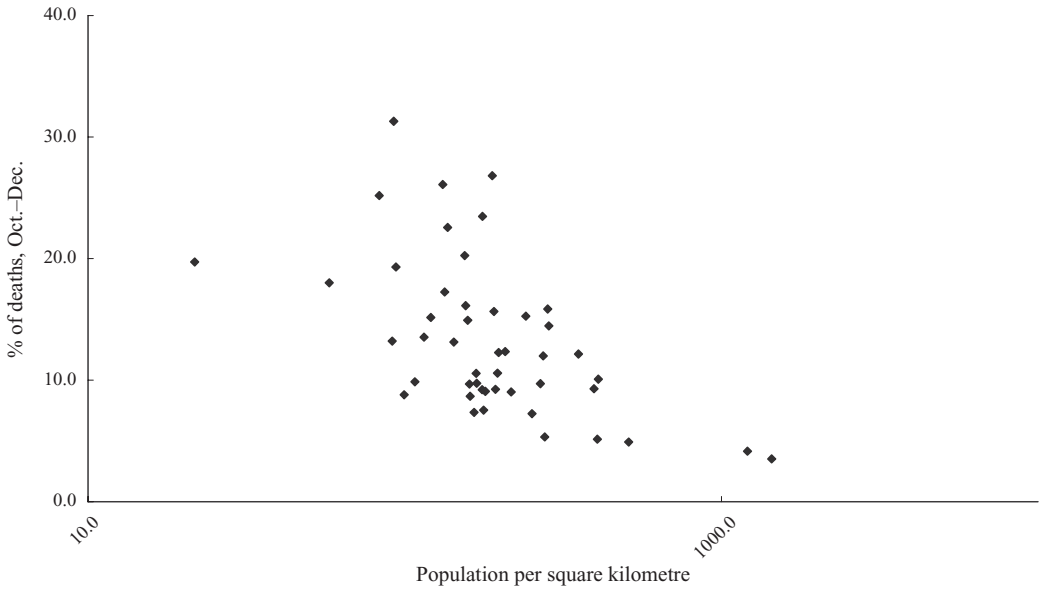

Figure 7. *Population density (1925) and percentage of measles deaths, Oct.–Dec. 1921–30*

Sources: Nihon Teikoku Siin Tōkei; *Eiseikyoku Nenpō.*

Table 2. *Percentages of seasonal measles deaths (rural prefectures and their capital cities, 1912–34)*

	Oct.–Dec.	*Jan.–March*	*April–June*	*July–Sept.*	*Total*
Miyagi Pref.	21.2	27.4	35.3	16.0	100.0
Niigata Pref.	27.0	22.9	29.7	20.4	100.0
Ishikawa Pref.	26.6	22.3	25.7	25.5	100.0
Sendai City	9.2	24.2	43.1	23.6	100.0
Niigata City	11.4	17.4	43.1	28.1	100.0
Kanazawa City	13.1	18.0	33.9	35.0	100.0

Source: Nihon Teikoku Siin Tōkei.

patterns of prefectures went through a gradual change during the period under consideration. In most of rural prefectures, the autumn/winter peak tended to disappear and, consequently, the spring/summer peak became increasingly prominent (table 3). These pieces of evidence confirm that the infection rate was determined by population density, connectivity of sub-regions, and other societal factors, not geographical location or climatic difference per se, and this created differences in the seasonal patterns of measles mortality.[47]

Primary schools moulded the epidemiological spatio-temporal structure of communities. Schools provided a place where cases multiplied through transmission, and they diffused the disease to distant settlements within the school-centred community. A primary school thus integrated children from distant neighbour-

[47] The contrast between single-peak seasonality of urban prefectures and twin peaks in rural ones had been a subject of discussion among Japanese epidemiologists in the 1940s. See, inter alia, Nobechi, 'Mashin no ekigaku'.

Table 3. *Progressive decline in the percentages of measles deaths in Oct.–Dec., urban and rural prefectures, 1900–36*

	1900–10	*1911–20*	*1921–30*	*1930–6*
Tokyo	9.2	6.1	3.5	2.8
Osaka	5.8	6.6	4.1	2.0
Aomori	33.4	40.9	25.2	19.7
Shimane	22.5	23.5	13.5	7.4
Kagoshima	27.2	23.3	16.1	16.7
All prefectures	17.2	18.0	11.4	9.0

Source: Nihon Teikoku Siin Tōkei.

hoods of a village into an epidemiological unit: when the school was closed, one was exposed only to the measles virus of one's neighbourhood, but during term-time, one was exposed to the virus of any place in the village, mediated by the school. Although no hard evidence is available, one can speculate that the Meiji government's establishment of the nationwide system of primary school education contributed to measles becoming endemic after the outbreak of 1885, by increasing the chance that the young and susceptible would be exposed to infectives. It is an irony that primary schools, where children were vaccinated against smallpox and other infectious diseases and given instruction about hygiene, was also the hub of infection.[48]

It should be noted that this hub of infection, and linchpin of epidemiological spatial integration, was a by-product of the educational policy of the modern state. Likewise, a man-made temporal structure (that is, the academic year), established according to the system of the central government, dominated the seasonality of measles mortality. It should be emphasized, however, that the school's impact on the seasonality of measles was not just a product of educational policy, but was mediated by societal factors in the region or sub-regions. Although primary schools kept to a more or less identical schedule all over the country, the seasonality of measles mortality differed markedly among urban and rural regions.

V

This article has traced the trajectory of the measles virus at regional and local levels. It now turns toward the furthermost end of the transmission of the disease: the family.

The family is an important site for the multiplication of measles cases. When several susceptibles exist within a family, many of them catch the disease during an epidemic. Known as 'family aggregation', this phenomenon of infection of measles from a family index case to other susceptible members within the same family has been one of classic subjects of epidemiological studies. Such studies of intrafamilial transmission of measles have been carried out for some time, and recent

[48] Ministry of Health, *Gakkō Hoken Hyakunen-shi*, pp. 8–12.

research into measles in the Third World has established that the mechanism of infection within a family has an important impact upon measles mortality.[49]

To put the insights of these studies briefly: size matters. In a large family with many susceptibles in it, measles mortality is high, while in a small family, the risk of dying from measles is low. Epidemiologists have observed contemporary measles outbreaks in Africa and have found that children in a large family are more likely to die from measles during an outbreak. Departing from earlier excessive emphasis on the influence of nutritional status upon measles mortality, closer attention has been paid to the factor of age at infection, as well as the intensity of infection resulting from the clustering of cases.[50] The risk of death from measles is inversely related to age at infection. If contact with measles is delayed to a later stage in life, mortality is reduced. Reduction in family size thus has an important effect on measles mortality, by reducing the risk of contracting measles at an early, more vulnerable age. A large family also results in a situation where there is often more than one infective in a household, and where, if one susceptible of the family is exposed to the infection of multiple infectives, it leads to intense infection, or intake of a large amount of pathogens.

Some surveys conducted in the past support the argument in line with this model. Epidemiological research in the USA in the period 1929–34 established that the age at which infections of measles and scarlet fever occur is different in families of different sizes. Children of a larger family experienced measles infection at an earlier age of zero to four years, while those who were the only child in a family typically did not catch the disease until they were six to seven years old.[51] Likewise, a serological survey conducted by F. L. Black in New Haven in 1957 reveals that children of large families (that is, children who were one of three or more) became seropositive and had the antibodies to measles considerably earlier than those of smaller families.[52] Schools or kindergartens, which have a set age for admission, provide an explanation for the mechanism by which a smaller family size raised the age at infection and reduced measles mortality. Once susceptibles were infected with measles at school, they carried the disease to their own homes and infected their younger brothers and sisters, who had had a lesser chance of being exposed to the measles virus and were likely to be susceptible to the disease. The young children without elder siblings had a smaller risk of infection, and were likely to be able to avoid measles infection until they themselves went to school. The eldest child was likely to be infected at school or kindergarten: both surveys in the US showed a sudden rise in the infection rate at the age of schooling for the eldest child, while his or her younger brothers and sisters were typically infected at home by his or her elder siblings. In a society whose average family size is greater and which has a primary school system, the age at infection is low and the mortality of measles is high. In other words, fertility influences measles mortality through the temporal structure of schooling and the mechanism of the transmission of the disease from the school to the family.

[49] See, for example, Aaby, 'Overcrowding and intensive exposure'; idem, 'Malnutrition and overcrowding/intensive exposure'.

[50] Aaby, Bukh, Lisse, and Smits, 'Overcrowding and intensive exposure as determinants'.

[51] Wilson et al., 'Measles and scarlet fever'.

[52] Black, 'Measles antibodies'.

Table 4. *Cumulative percentages of those infected with measles, by age*

Place	*Kagawa*	*Tokyo–central*	*Tokyo–suburb*	*Osaka*	*Baltimore*	*Providence*
Publication date	1944	1958	1958	1955	1933	1939
Type	Cohort	Cohort	Cohort	Cohort	Patients	Patients
Period surveyed	1922–9	1928–57	1928–57	1941–3	1916–27	1929–34
Total number	10,907	16,842	18,634	22,347*	47,383	10,200
Age: 0	4.5	2.1	1.5	0.6	3.6	3.2
1	18.4	11.8	10.8	8.7	11.1	9.8
2	32.9	28.7	25.6	22.7	20.3	18.2
3	45.7	49.8	43.2	38.8	31.1	27.7
4	58.5	65.8	58.1	51.7	42.8	38.5
5	70.6	80.4	72.5	65.7	53.9	51.7
6	84.6	90.6	84.4	75.9	67.7	66.7
7	92.2	95.9	92.2	86.9	80.5	79.9
8	96.1	98.1	96.3		88.9	89.6
9	98.1	99.1	98.3		93.5	94.7
10	99.2	99.7	99.3		96.2	97.3
11	99.6	99.8	99.6		97.7	98.8
12	99.9	99.9	99.8		98.8	99.4
13	100.0	100.0	99.9		99.5	99.7
14	100.0	100.0	99.9		100.0	100.0
15+		100.0	100.0			

Note: * 19,427 had experienced infection at the time of survey.
Sources: Sumiya, 'Joshi no mashin rikanritsu to nenrei tono kankei'; Ogawa, 'Mashin no jōzai ryūkōchi ni okeru ekigakuteki tokusei ni kansuru kansatsu'; Takahashi, 'Mashin no riron ekigakuteki kenkyū 3'; Hedrich, 'Monthly estimates'; Wilson et al., 'Measles and scarlet fever'.

Epidemiological surveys in Japan conducted in the 1950s routinely found instances of measles being transmitted from the school to the family. In a small epidemiological survey conducted in 1947, 53 per cent of the schoolchildren who caught measles transmitted it to their family members; 17.7 per cent of them had caught measles from a family member. They also found that the eldest children were infected at a higher age, with the age of entering primary school being the peak, while the age of infection for younger brothers and sisters was substantially lower.[53] From this evidence, it is possible to formulate a hypothesis that the decline in fertility in the 1920s and 1930s, decades during which the national crude birth rate dropped from 36.2 in 1920 to 26.6 in 1939, contributed to the decrease in measles mortality by heightening the age at infection.

One set of Japanese historical data supports this hypothesis. Table 4 represents the percentage of cases according to age at infection, taken from three surveys in Japan. The survey in Kagawa was published in 1944 and surveyed the age at infection of those who were born in 1922–9.[54] The survey in Tokyo published in 1958 had two parts; one surveyed residents in the central part of the City (Kyōbashi area) and the other studied those in the suburb (Kitatama area). It surveyed those who were born between 1928 and 1957. For the urban area, the median figure of the year of birth is 1943, and 65 per cent of those included in the survey were born after 1940, while for the suburban area, the median figure of the year of birth is 1944 and 76 per cent were born after 1940. The survey in Tokyo thus represented those who were born substantially later than those surveyed in

[53] Sakamoto and Honda, 'Gakudō ni okeru mashin ryūkō ni tsuite'.
[54] Sumiya, 'Joshi no mashin rikanritsu to nenrei tono kankei'.

Kagawa. The survey in Osaka was published in 1955, and scrutinized the age at infection of those who were born between 1941 and 1943. For the sake of comparison, data from Baltimore and Providence are attached. These are taken from surveys of the age of reported measles patients, respectively during 1916–27 and 1929–34.[55]

All three surveys show that measles was a disease of early childhood: more than 90 per cent of those who were surveyed experienced measles before the age of seven. Beneath this shared characteristic of the disease affecting young children, there lies one apparently small but crucial difference between children in Kagawa in 1920s and those in Tokyo and Osaka in the 1940s and 1950s. The accumulated rate of infection shows that children born in Kagawa in the 1920s were more likely to get measles earlier, at the age of up to two years, while those surveyed in Tokyo and in Osaka in the 1940s and 1950s were more likely to avoid infection until they were two or three years old. Admittedly, this evidence is drawn from an imperfect set of data and is far from conclusive. Still, it is reasonable to assume that this set of data supports the hypothesis of the progressive postponement of measles infection in Japan, as the fertility rate became lower after around 1920. There are also impressionistic contemporary observations of the postponement of infection. During one outbreak in a northern port town, one doctor observed the slight increase in the age at infection: while studies conducted in various parts of Japan in 1933–7 had mostly shown a typical age at infection of one to two years, the outbreak predominantly affected those aged two to four years.[56] Japanese epidemiologists were well aware that the age at infection differed considerably among regions in Japan, and between Japan and western countries, as is evident from table 4. Studies conducted in various cities, towns, and villages in Japan in the 1950s had shown that the peak age at infection was observed to be between one and four years, mostly typically around 2.5 years, whereas observations in cities in the US during the 1920s had shown that six to seven years was the peak age at infection.[57]

The hypothesis of the increase in age at infection also fits the picture of measles mortality. From its peak in the 1920s, the mortality of measles started to decline throughout the nation. The steep decline in mortality, which must have taken place during the post-Second World War years when data are not available, was no doubt due to the introduction of antibiotics, such as penicillin, and of sufa-drugs, which prevented death from complications such as pneumonia.[58] The timing of the first mortality decline coincides with that of the fertility decline. The crude birth rate of the nation peaked in 1920 at 36.2 per thousand and then started to decline, first gradually, and then precipitously, with major but short-term increases in the decade that included the wartime crisis. At least part of the decline in measles mortality can be explained through the change in the age at infection, caused by

[55] It should be noted that age at infection was much lower in Japanese surveys than American ones, probably because of the higher birth rate in Japan during the period in question. See also a small-scale survey in Tokyo in 1947, which showed that 109 out of 199 schoolchildren had already experienced a measles infection, and a further 64 had experienced infection during their first year at school; Sakamoto and Honda, 'Gakudō ni okeru mashin ryūkō ni tsuite'.

[56] Oikawa, 'Mashin no ryūkō no kansatsu'.

[57] Wakabayashi, 'Mashin no ekigakuteki kenkyū I'.

[58] For the remarkable effect of penicillin and sulfapyridine, see Oikawa, 'Mashin no ryūkō no kansatsu'.

the decline in fertility.[59] With declining fertility and the decrease in the number of young children in a family, a larger proportion of children enjoyed the epidemiological privilege of being the first child of the family and of postponing their exposure to measles until a later age, when they had developed resistance. Those under the age of two years accounted for less than 20 per cent of infections in Kagawa in the 1920s, but more than 70 per cent of deaths came from this age group. If one calculates ratios of case fatalities at different ages from the data from Kagawa in 1922–9, one finds it declines precipitously with age: getting measles before the age of one year was 2.8 times more lethal than being infected at the age of two years, and infection during one's infancy was fatal about eight times more frequently than at the age of two.[60] A small decrease in the chance of infection at these high-risk ages must have led to a substantial improvement in measles mortality. A calculation of the age-specific mortality of measles in England and Wales for 1945 reveals a similarly declining fatality risk as the age at infection rose: for those under one year old, deaths per 1,000 reported cases were 13.16, while the comparable figure for those aged one to two years was 3.09, and for those aged three to four years was 0.64.[61]

It needs to be stressed that the higher age at infection was one route through which the decrease in fertility led to lower mortality from measles. Aaby has identified the intensity of exposure as the single most important factor that determined the case fatality of measles, on the basis of analysis of data from community studies in Africa in the 1970s and from hospital records in Copenhagen in 1915–25.[62] Aaby maintains that both of these sources suggest that cases of 'intensive' exposure or exposure to a large dose of pathogens were much more likely to lead to severe and fatal forms of measles. Since such intensive exposure happens more often in a family with a large number of susceptible children, a decrease in family size lowers case fatality and mortality of measles. Besides, it is also likely that children of a smaller family experience better nutrition and maternal care, although Aaby maintains that nutritional status does not influence the case fatality of measles as much as has been believed by epidemiologists and historians. Thus it should be emphasized that the postponement of infection was one of the factors that contributed to the decline of mortality from measles in Japan from the 1920s.

[59] Other possible contributory factors might include those that improved general resistance, such as improvement of nutritional status and better medicine. Scott and Duncan (*Demography and nutrition*, pp. 311–12) have probably correctly emphasized the role of nutrition in the decline of measles mortality in England in the nineteenth century. Although one is tempted to look for specific measures, it is unlikely that any single medical innovation helped to lower measles mortality. In the early 1930s, the use of serum for the prevention of measles was still very limited in Japan. Although in the late 1930s, some attempts were made to vaccinate a large number of susceptibles, it was not beyond experimental use: the Metropolitan Police of Tokyo distributed the vaccine for measles only for a handful of susceptibles; 1,501 in 1935 and 2,700 in 1937 (Uruno, 'Mashin no yobō oyobi chryō to ketsueki chūsha'; Iguchi, 'Mashin no yobōhō to narabini sono chiryō kōka').

[60] Kagawa is chosen because the prefecture provides the only set of data (the age at infection *and* age-specific mortality) to calculate the ratio of age-specific fatality. This picture of the high mortality from measles at an early age is confirmed by many studies, both historical and contemporary. A study that compared measles in Nigeria in the 1950s with measles in England before the twentieth century has found that low age of infection, as well as poor nutritional status, contributed to the severity of complications such as bronchopneumonia, laryngitis, and diarrhoea accompanying the disease; Morley, Woodland, and Martine, 'Measles in Nigerian children'.

[61] Reves, 'Declining fertility'; Mercer, *Disease, mortality and population in transition*.

[62] Aaby, 'Severe measles in Copenhagen'; idem, 'Malnutrition and overcrowding'.

VI

This article has tried to demonstrate that the study of measles provides historians with a powerful tool to examine the spatio-temporal structure of infection, by showing the profound and complex changes in the epidemic pattern of measles in Japan from around 1900 to 1960, and by using the concept of the three hierarchical layers of spatial spheres, namely, those of the region, the school-centred community, and the family. With its simple man-to-man droplet infection mechanism and high infection rate, measles data can be exploited with great benefit by socio-economic historians who are interested in the complex mechanisms that determined the health status of societies in the past. Unlike diseases such as cholera and the plague, which have been studied for their ability to reveal societal factors during critical situations or social upheavals, measles data allow historians to reveal these factors in circumstances of gradual change. The findings presented here reveal the complex moulding of the spatio-temporal structure of infection in modern Japan, through the regional epidemiological integration driven by urbanization and the development of transportation, the creation of nationwide elementary education by the state, and the decline in fertility in the private sphere of the family.

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