EPIDEMICS OF MEASLES IN A SEVENTEENTH-CENTURY ENGLISH TOWN

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Despite the recent suggestion that the demographic pattern between 1540 and 1700 was generally determined less by mortality than by fertility, we still need to know much more about the mortality patterns of this period.[1] The buoyant rate of sixteenth-century population growth begins to drop in the early decades of the seventeenth century and continues to fall until between about 1650 and 1690 the total population is shrinking.[2] It appears that worsening child mortality levels played a prominent part in causing this demographic depression, for although expectation of life at birth fell drastically during this period, expectation at the age of thirty only dropped marginally — it seems that most of the changes in mortality took place at the expense of children, and then not so much among infants as in the earlier years of childhood after the first.[3] This might indicate that these deaths were more directly connected with disease than with environmental factors since children were more likely to die from the direct consequences of most infectious diseases than either adults (with their immunities acquired in childhood) or infants (with their short-term inherited immunities). So any explanation of the worsening mortality rates of the seventeenth century must concern itself primarily with disease among children, and with the first half of the century when the deterioration seems to have been most dramatic. Compared with the Elizabethan period, either the resistance of children to familiar diseases fell, or those diseases became more malignant or widespread, or new diseases appeared which attacked children more severely than adults — or some combination of these factors was responsible.

The problem of identifying these diseases remains unsolved, indeed largely unattempted. In this context, any parish register which consistently identifies causes of death is a treasure. Some registers distinguish plague burials but epidemics of any other disease are very rarely specified. However, in the register of the Lancashire market town of Bolton the term 'pox' is appended to individual burial entries in four major epidemics among children between 1635 and 1656.[4] This fact has already been noted in print with reference to the outbreak of 1642, and interpreted as an epidemic of smallpox.[5] It is the contention of this paper that these were outbreaks of measles. The argument which supports this identification can only proceed when we have examined the basic outline of these four episodes as

presented in Table 1, which shows the seasonality and shape of the epidemics and Table 2, which shows the age distribution of the burials. Figure 1 shows in series B the monthly totals of child burials between 1632 and 1656.

Table 1. Seasonality of measles burials

	Total number of measles burials											
	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug
1635-6	4	6	16	12	4	1						
1642							10	36	16	12	5	
1647-8		1	2	6	23	24	7	3				
1655-6	1	10	17	16	11	9	4					

Table 2. Age distribution of measles deaths

Age	1635-6		1642		1647-8		1655-6	
	Parish	Town	Parish	Town	Parish	Town	Parish	Town
Not traced	7	2	8	4	13	8	29*	18*
0-5 months	3	1	9	3	7	5	2	0
6-11 months	17	11	14	7	22	16	2*	2*
1 year	4	2	26	17	9	5	6*	5*
2	6	1	11	4	8	4	4	2
3	2	2	5	4	6	5	1	1
4			1	1			5	3
5	1	0			1	1	3	2
6-7	2	0	1	0			12	8
8-16			2	0			4	1
Adult	1	0	2	0				
Total	43	19	79	40	66	44	68	42

Notes

*A gap in the baptismal record between July and November 1654 renders these figures eccentric. Many of those untraced must be one year old.

In terms of seasonality, three epidemics extended from autumn into winter (September-January 1635-6, October-April 1647-8 and October-March 1655-6 with peaks in November, January/February and November/December respectively) while the fourth covers spring and early summer (March-July 1642 with a peak in April). The contour of each epidemic is distinctly pyramidal, with a central concentration of burials occupying a period of about two months and a 'tail' on either side extending the duration of the epidemic to a space of four or five months.[6] The age distribution of mortality varies a little between the four outbreaks, but all are children (253) with the exception of one adult in 1635-6 and two in 1642.[7] The bulk of the mortality falls between the ages of five months and three years: in the middle two epidemics only 5 and 1 ½ per cent of the victims were aged four or more. The exception to this generalisation comes in 1655-6, when there was a large group of deaths in the age group four to seven. This inconsistency need not be disquieting, for with any frequently recurring children's disease, the age structure of mortality must reflect the length of time which has elapsed since the previous visitation.

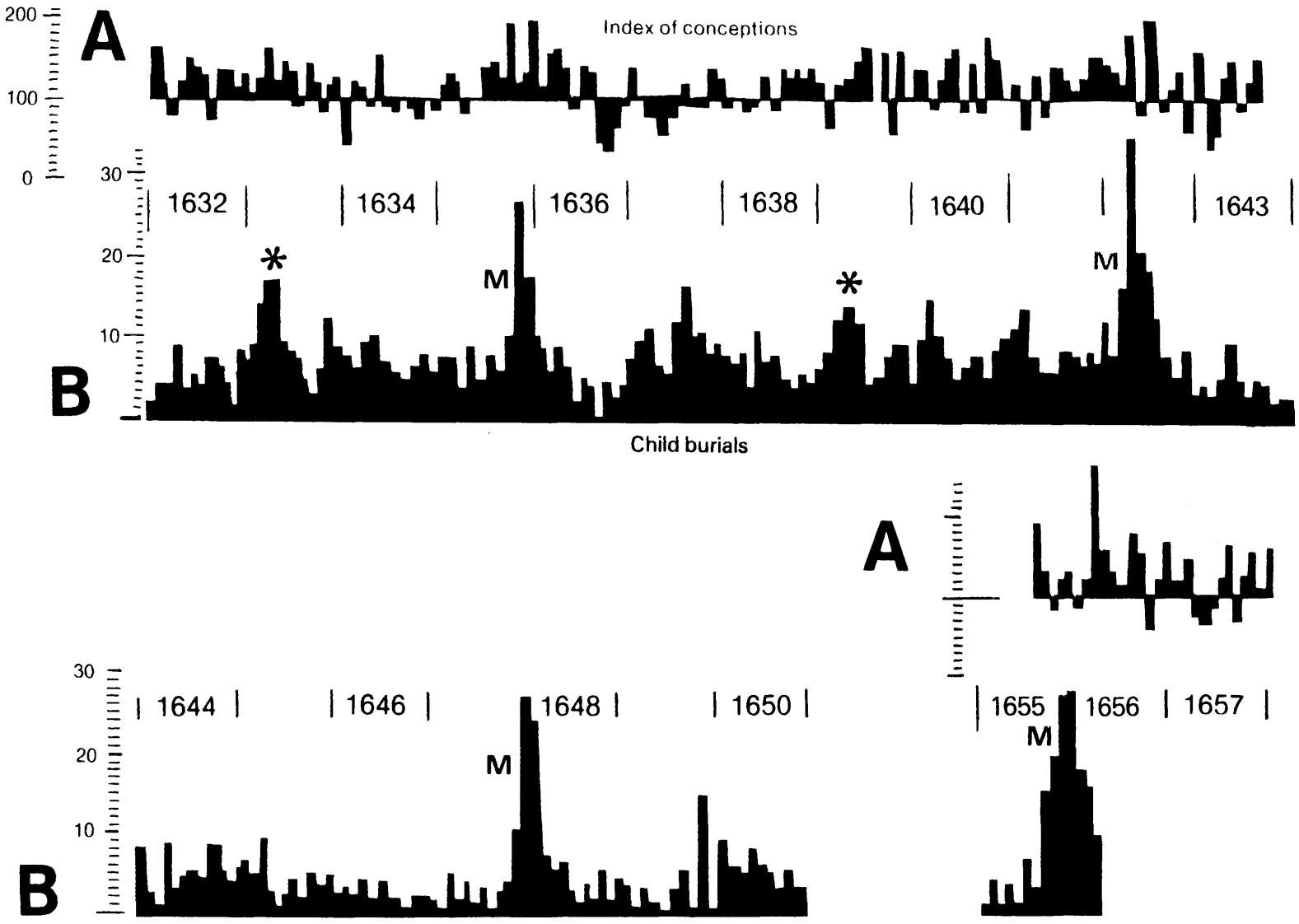

Figure 1. Bolton child burials and conceptions by months 1632-57.

Series A = Conceptions per month compared to seasonally adjusted mean.† Series B = Total number of child burials per month.

Notes

M – specified 'pox' epidemics. * = suspected unspecified measles epidemics. Series A omitted 1644-50 due to under-recording in the register.

† Baptismal totals for each month of the year were reduced to a mean by taking a moving average of eleven consecutive years, from which the two highest and the two lowest values were removed. These means were plotted on a graph for each month of the year and further smoothed by eye. These smoothed means were compared with the actual baptismal totals and the result expressed as an index number for each month with the mean set at 100. Thus a baptismal total of half the mean appears as 50 and one of double the mean at 200. Finally the baptismal totals were transposed back by nine months to approximate to the time of conception. This method has the usual defect of under-estimating the total number of conceptions by suggesting that the unknowable losses which take place between conception and baptism (mostly miscarraiges and stillbirths) were never conceived in the first place.

An understanding of these epidemics also demands a knowledge of the topography of the parish, for the ecclesiastical parish of Bolton at this date consisted of a market town of 2,000-2,500 people[8] set within an extensive rural area of about fifty square miles whose scattered townships and hamlets contained over half the total population of the parish.[9] Thus the totals derived from the register are an amalgam of urban and rural experience which has to be disentangled: fortunately the register specifies the township from which most entries originate.

We may now return to the problem of whether these epidemics were caused by smallpox or measles. The chief problem here is that we lack any specialist study of the status of smallpox in the seventeenth century written later than Creighton's classic general work of 1891.[10] The generally accepted picture is that smallpox was a relatively mild and familiar disease of childhood in the earlier sixteenth century which by the later seventeenth century had changed to be not only much more fatal but also to be generally epidemic rather than endemic, killing adults and children. Exactly when and how this change took place is obscure, but by the eighteenth century we know much more.[11] Smallpox in large towns (perhaps with 15,000 people or more) is seen as a general disease of childhood, with a mortality which varies from a low to a moderate level; few children survived to maturity without being exposed to it, so native adult deaths were a small minority of the total, although recent migrants from the countryside died in much larger numbers. But in smaller towns and in the countryside epidemics were infrequent and many people survived to adulthood without exposure and fell victim to the disease as adults. Perhaps Razzell tends to exaggerate the universality of the disease in pursuit of his specialist argument, for the evidence he presents makes it clear that there must have been very many adult sufferers: many towns similar in size to Bolton — Brighton, Diss, Lewes and Tenterden for instance — arranged mass innoculations for between 49 and 59 per cent of their populations, which must indicate that a very substantial proportion of adults had acquired no childhood immunity.[12] In the countryside the immune adult population was even smaller, and where we know the age of sufferers in epidemics outside the largest towns, there is always a substantial proportion of adults — for example 48 per cent in Godalming and 40 per cent in Aynho.[13]

Other characteristics of smallpox are that it spreads slowly and usually from household to household.[14] It can often be present for long periods of time in epidemics whose 'shape' tends to be sporadic and lacking much central concentration, sprawling over as much as two years, though some cases were more concentrated and pyramidal in the Bolton pattern.[15] Outside the largest towns, epidemics recurred irregularly and rarely more frequently than every seven years; often twenty years or more would separate them, especially in the countryside.[16] Smallpox can occur at any season, but there does seem a tendency for the winter outbreaks to be sporadic and shapeless while the summer ones were more common and more concentrated, especially in the market towns.

This is the pattern in the eighteenth century: how much of it can be extrapolated back into the seventeenth century? The epidemics Creighton notes between 1591 and 1642 were more summer than winter affairs, and there is plenty of literary

evidence of upper class victims, usually adults and often young adults or adolescents.[17] Between 1605 and 1641 epidemics appear to become more frequent, feared and fatal, though much of the evidence comes from London and the disease seems to have been much less common in the provinces.[18] If smallpox was less common in the seventeenth century than it was to be later, we should expect more adult deaths because fewer people were exposed to it in childhood; if, as the evidence of the London Bills of Mortality suggests, the virus was becoming more virulent, then the death toll in epidemics would be rather lower than it was to be later; both these suggestions seem consistent with the available evidence.

It will be evident from all this that we should expect a smallpox epidemic in a market town like Bolton with its extensive rural surroundings to kill substantial numbers of adults, to spread slowly in a sporadic way over a long period of time, to occur much less frequently than every three years and in mid-summer as often or more often than in the winter.[19] It will be noted that in all these respects, the main distinguishing features which a register-based study can reveal, the Bolton epidemics are not at all characteristic of smallpox, indeed are nearly as unlike that disease as one might reasonably expect.

An additional point of relevance is the fact that before the later seventeenth century there was only a limited awareness that the principal fatal diseases which produce raised rashes — smallpox, measles and scarlet fever — were in fact separate diseases.[20] The prevailing view was that they were all basically 'fever' and that the differing skin symptoms were indicative of the severity of the disease rather than pointers to the presence of quite different diseases. Thus the early London Bills of Mortality give a single category to 'smallpox and measles' and medical writers of the time join the two as if they are essentially one.[21] So when the parish clerk of Bolton uses the term 'pox' he is not deliberately avoiding making the identification of measles; he merely means 'a fever involving eruptive spots'. It was not until Sydenham produced his classic work based on the London epidemics of the 1670s that the medical profession began to draw clear distinctions between the two diseases, although some laymen had throughout the century made common-sense distinctions between the two.[22]

Since scarlet fever has been named, it would be as well to exclude it as the Bolton culprit at this point. What little we know of the disease in the seventeenth century suggests that it was not then at all the major killer that it would be in the nineteenth century.[23] Pepys writes that his serving maid 'is fallen sick of the meazles we fear or at least of a scarlett feavour', indicating that measles was regarded as the more serious of the two.[24] It affects chiefly the age group three to ten, and is rare under one year, while it is not highly infectious: neither of these characteristics fits the Bolton epidemics. The only other major epidemic disease which produces a rash is typhus. This may be easily excluded because it is very much more fatal among adults than children, so we should expect reverse of the Bolton age distribution of mortality if typhus were the culprit.

So we are left with measles. Where it occurs regularly it is rare under the age of five months and its highest mortality lies in the age group nine months to two-and-a-half years.[25] The attack rate can be high up to the age of five but the case fatality rate

drops strikingly over the age of three. This pattern is influenced by the frequency of epidemics, which recur in a two- or three-year cycle in towns and rather less often in the countryside; since most children are infected in each epidemic, age-specific mortality rates must reflect the period of time which has elapsed since the previous outbreak.[26]

Measles spreads through a community much more readily than smallpox and infects most of the susceptible population in each epidemic, conferring immunity for life. Where there is no childhood exposure to measles, as in isolated rural communities or islands, all ages catch the disease and up to 20 or 25 per cent of the population have died.[27] Because the disease spreads so readily, epidemics are of short duration, exhausting the pool of susceptibles in three to six months. It is essentially a cold season complaint, peaking at the end of March but occurring at any time between September and the following July: in a group of nineteenth-century epidemics two-thirds ran from about October to February, though many were over by Christmas, while the remainder began in late winter, typically February, and continued until July: some did not begin until April or even May.[28]

It will be apparent from these details that they fit the characteristics of the Bolton epidemics very well. Seasonality is exactly right, the age distribution of mortality is exactly what one would expect, as is the frequency, contour and duration of the epidemics. It will be assumed from this point that the identification of measles as the disease causing these mortalities has been established beyond reasonable doubt, although of course absolute certainty must always elude us.

Figure 1 shows measles epidemics as they affected the parish as a whole; they appear to occur at intervals of about seven, five and eight years between 1635 and 1656. The geographical distribution of mortality varies considerably, for the penetration of the virus into the rural hinterland was very uneven. Mortality was fairly uniform in the market town itself, but of the four outbreaks in the seventeen rural settlements, one township recorded no death, four were hit on one occasion, three in two epidemics, six townships in three and only three settlements were affected on all four occasions. In 1642 and 1647 the disease begins in the town and spreads into the countryside, as one might expect, but the reverse is true of 1635 and in 1655 it begins in country and town simultaneously. The longer periods without exposure to the virus enjoyed by some of the townships is reflected in the fact that victims here were often older than in the town — all three adult burials came from the countryside — and also by the greater proportion of infants aged five months or less: a mother normally passes sufficient antibody to her unborn child to protect it for the first six months after birth, so these cases indicate that the mother had no immunity to pass on.[29]

Immersed in the age structure of measles mortalities there lie clues to epidemics which are not specified in the register. The evidence needs careful interpretation because since case fatality rates fall sharply over the age of three, a lack of burials over this age may not indicate an absence of infection. This said, it is clear from the last recorded epidemic of 1655-6 that while there were many victims aged seven there were far fewer aged eight, indicating that the older children had been protected by their infection in 1647-8 and that there had been no extensive outbreak

between the two recorded ones. Pushing this argument back through time, nearly every child of four or more seems to have been immune in 1647-8, indicating an unrecorded epidemic in 1644-5; the register reveals no peak in child mortality at this time so a less virulent strain of the virus may have been active. The ages of the victims of 1642 indicate an unrecorded exposure three years previously and the child burials show tempting peaks in March to May 1639, a likely season. Mortality then was considerable, but not on the scale of the recorded epidemics.

If there is validity in this approach, the outbreak of 1635-6 was preceded by an unrecorded one about two years before, and there is a burial peak in the winter and spring of 1633. Another interesting burial peak can be seen in the late spring of 1629, but it would be stretching the point too far to identify this one with measles; however, we are left with an attractive hypothesis which gives us seven epidemics of measles between 1633 and 1655, the first six occurring at fairly regular three year intervals, which is what one would expect from modern medical experience. With this pattern of regularity, variations in the level of mortality must be caused by different degrees of virulence of the virus, an uneven spread of infection through the rural area and also by differences of season — since most of the victims would have died from lung infections, mortality would be highest in midwinter: perhaps there were fewer infant deaths in 1642 because the epidemic was postponed until April and May.

The modern history of measles shows that it passes through phases of heightened and then lowered virulence, these phases tending to take about thirty years between peaks.[30] It is quite possible that in the 1630s the disease entered a phase in which it became both more common and certainly more fatal. The presence of adult victims in the countryside in 1635-6 and 1642, and their absence thereafter might indicate that it had been possible before 1634 for some children to escape contact with the disease, although the number of adults involved is so small that this argument may not be valid. And the very fact that the compiler of the register was moved to note the outbreaks might indicate that they, or at least their fatality levels, were novelties. Against this is the possibility that the compiler had some strong personal interest in the disease, perhaps derived from a bereavement prior to 1635.

During modern serious epidemics, such as Glasgow in 1807-12 or during recent famines in West Africa, the case fatality rate of measles has reached 15-20 per cent or more.[31] We cannot begin to calculate rates for the whole parish of Bolton because the disease did not affect all the rural townships, but we can work on the market town alone. Here in 1635-6 at least 31 per cent of those aged six to eleven months died and in 1642 at least 24 per cent of the one year olds were killed:[32] these figures are minima, for we have assumed that all those in the baptismal register would have survived until the measles epidemic, but some of them would have died from other causes before the epidemic began. If one looks at the whole age range of six to thirty-six months then in 1635-6 some 7.5 to 9 per cent of the total baptised was lost, rising to 16-18 per cent in the outbreaks of 1642 and 1647-8. These are again minima, for by the third year after baptism one would expect a substantial cumulative loss from other causes which might put the case fatality rate up to the formidable level of 20-25 per cent or more.

In the three specified epidemics between 1642 and 1656, Bolton market town lost on each occasion the equivalent of about 60 per cent of one year's average baptismal total. This might indicate that over these fifteen years measles killed about 12 per cent of those born. These figures ignore the unrecorded minor epidemics which we have suggested might raise the frequency of recurrence to a three-year pattern; they also assume that every measles death is identified in the register in the specified instances.[33] Taking these factors into account, measles may have killed about 15 per cent of the children baptised in the town between 1633 and 1656. If this is so, then the disease was responsible for the deaths of about one third of all the children buried in this period.[34] The major role of measles in child mortality is reflected in the fact that the epidemics under review account for all the prominent peaks in child burials in the period 1625-1660 in this register.

However, we cannot make the automatic assumption that measles reduced population growth to this degree, for, like many diseases of young children, measles could stimulate the birth rate to compensate for the deaths it caused. The total of baptisms for the year following the measles epidemics is abnormally high in every case, and the total of 'unexpected' births corresponds roughly to the total of measles burials. We have a ready-made explanation to hand in the fact that if protracted breast feeding acted as a form of birth control, the death of a nursing child would lead to another pregnancy more quickly than would otherwise have been the case.[35] But in fact these baptismal peaks are more difficult to explain than this simple analysis would suggest. An examination of monthly conception totals (fig. 1, series A) shows that in 1635 and 1642 conceptions were already running well above average before the epidemic began, and this is probably also true of 1655.[36] But in all three years there is a striking peak of conceptions which follows the climax of the measles mortality — there is a lag of two months in 1635 and of three months in 1642 and 1656. This seemed to be much more significant, so the month of April 1643 was examined: one might have expected about eighteen conceptions in this month yet the total is thirty-two. But only seven of these thirty-two pregnant wives had lost children in the epidemic. In the case of all these seven the theory of interrupted breast feeding seems to work, for all seven children were aged between eleven and thirteen months at death. But the theory can only explain about half of the excess conceptions, and some of this total could have been unconnected with the epidemic, for birth intervals of twenty to twenty-two months are not unreasonable in normal circumstances.

If we take all the seventy couples which lost children under four years old,[37] we find that twelve wives were already pregnant again before the epidemic started and twenty baptised no more children in the register; of the remaining thirty-eight bereaved parents, twenty took between six months and five years to produce another conception and only eighteen wives became pregnant within five months after the burial of their measles victim. Thus the 'automatic' compensatory process seems to apply to a maximum of eighteen children while the bulge in conceptions would suggest that up to sixty extra children were born in this year. Thus it is clear that although there is a distinct connection between the epidemics and a raised birth rate, most of the 'unexpected' children were not produced by bereaved parents. We are left to speculate that the experience of observing the bereavement of other

parents impressed these couples with the fragility of their children's lives and the wisdom of insuring against future loss by conceiving again.[38] Not enough is known of the practice of birth control in this period to make further discussion very viable. But we should bear in mind that, however caused, these waves of conceptions did not really cancel out the epidemic losses, for there is usually a trough in conception levels after the wave, which suggests that these babies were born earlier than would otherwise have been the case, but were not necessarily additional to the total numbers which would have been born without the agency of the epidemic.

It is clearly not possible to claim that measles was a major child killer throughout Britain on the basis of these Bolton findings alone. the London Bills of Mortality indicate peaks of measles burials in 1632, and (after a gap in the records in 1636-46), 1648, 1652 and 1656.[39] The correspondence with the chronological pattern already shown in Bolton is interesting. The level of measles mortality in London was well below the Bolton figure, with 153 burials in the worst year of this series in 1656; the 795 deaths of 1674 are nearer, but still do not approach the Lancashire situation, though one never knows how many 'smallpox', 'convulsions' or 'fever' burials are in fact measles victims.[40] There is little work available on child epidemics since most of the published material does not differentiate between adult and child burials, but of some West Midland urban registers we have examined from this point of view, there is a significant level of correspondence with the dates of the Bolton epidemics: there were peaks of child mortality in 1635-6 in Shrewsbury, St Mary, Stratford-upon-Avon, Gloucester St Nicholas and Ludlow for instance, while the 1648 and 1655 peaks find parallels in Shrewsbury.[41] Measles is the kind of disease which would spread far and fast, indeed the first Bolton burial in 1647 was a child from Manchester, indicating a possible source of the infection.[42] But it must be pointed out that the figures of monthly burials given by Wrigley and Schofield do not provide much in the way of correlation in the years concerned, perhaps because child mortality is obscured by the addition of adults and because the parishes in the sample are too scattered to pick up these localised attacks.[43] It may also be true that the damp north-west was always more liable to the lung complications which caused most of the measles mortality.[44]

Much ink has been spilt on the issue of the 'autonomous' character of mortality — was it influenced by social and economic factors such as food supply, or were these less important than independent changes in the distribution and virulence of diseases? Measles give no easy answers here. There is modern evidence that measles mortality is high where living standards are low, and malnutrition is a major factor in the high mortality of African famine-related epidemics.[45] We know that the early seventeenth century could well have seen chronic malnourishment in north-western England, for we already have studies of famine-related mortality crises in the region in 1596-8 and 1623. Yet we also know that the virulence of the measles virus ebbs and flows in phases which do not seem to be related to external circumstances. It may well be that we have here the first documented phase of a series which is fairly clear after the serious outbreaks of 1670 and 1674, followed by a quiescent stage until 1705. Whether the period between 1630 and 1674 forms one virulent phase, or two with a short intervening lull, is not at all obvious, but the shifts themselves must owe more to the mysteries of microbiology than to simple food

shortage. Whatever the conclusions to this issue are, however, it does seem likely that measles must be assigned a prominent role in the mortality changes of the seventeenth century.

NOTES

1. E. A. Wrigley and R. S. Schofield, **The population history of England 1541-1871,** 1981, pp.242-5.
2. Wrigley and Schofield, pp.207-12.
3. Ibid., pp.230-1, 248-50.
4. Lancashire Parish Register Society, vol. 50, 1913, pp.427-9, 455-8, 473-5, 491-4.
5. C. D. Rogers, **The Lancashire population crisis of 1623,** 1975, p.5.
6. The period from first to last burial in Bolton town itself was thirteen, fourteen and fifteen weeks for the first three epidemics and twenty-three for the last.
   In this carefully compiled register, child burials may be reliably identified by the use of the formula 'son of . . .' or 'daughter of . . .', or by the use of the words 'child' or 'infant'. These terms appear to cover all burials under the age of fifteen or so, and many until marriage has taken the individual from the family home. Mortality among those in late adolescence and early maturity is so low that it may be ignored as a distorting factor, as is illustrated by the absence of anyone over sixteen in Table 2. Precise ages were discovered by comparing the date of baptism, where recorded, with the date of death.
8. Assuming a birth rate of about thirty per thousand.
9. **Victoria County History of Lancashire,** vol. 5, 1911, pp.235f. The rural area produced 62 per cent of the baptisms and 59 per cent of the burials in the register in normal years.
10. C. Creighton, **A history of epidemics in Britain,** 1891. See also C. W. Dixon, **Smallpox,** 1962, pp.191-6.
11. P. Razzell, **The conquest of smallpox,** 1977.
12. Razzell, pp.83-92, 90-1, 141.
13. Ibid., pp.106, 116. Of the Aynho fatalities, seventeen out of twenty-five were aged over twenty (Creighton, vol. 2, p.520).
14. Ibid., p.15.
15. Ibid., pp.124-5.
16. Ibid., p.52. In the village of Trentham there were nine deaths from smallpox spread over the years 1722-32, and only three were aged under five: a typical instance of the sporadic and limited nature of the disease in the countryside. **Trentham,** Staffordshire Parish Register Society, 1906, pp.218-37.
17. Creighton, vol. 1, pp.461-5. The Chamberlain letters contain forty-five references to smallpox affecting members of the upper classes in London between 1602 and 1624. On three occasions his remarks show that he sees the disease as a summer phenomenon which needs unusually warm winter conditions to flourish. **The letters of John Chamberlain,** N. E. McClure (ed.), 1939, vol. II. pp.205, 218, 413.
18. Adolescents migrating from the provinces rapidly succumbed to smallpox when they reached London; Josselin sent four children from rural Essex to London in their teens between 1659 and 1680 and all were infected within a matter of months. A. MacFarlane, **The family life of Ralph Josselin,** 1970, pp.112, 113, 119, 120.
19. Smallpox infects whole families, so one would expect some examples of multiple deaths in single families, which is very rare in Bolton. For a three-year cycle, see below.
20. W. F. Byam and V. Nutton, **Theories of fever from Antiquity to the Enlightenment,** 1981, pp.66-7.
21. Creighton, vol. 1, pp.456-9, 462.
22. J. D. Rolleston, **The history of the acute exanthemata,** 1937, pp. 78-80.

23. Ibid., pp. 50-7.
24. **The diary of Samuel Pepys,** R. Latham, (ed.), vol. 5, 1971, p.318.
25. Useful accounts of measles can be found in A. B. Christie, **Infectious disease: epidemiology and clinical practice,** 1974, and H. S. Banks, **The common infectious diseases,** 1949; cf. Rolleston, pp. 78-88.
26. M. Burnet, **The natural history of infectious disease,** 1962, pp.218f; M. Greenwood, **Epidemics and crowd diseases,** 1935, pp.181f.
27. Christie, p.384; Rolleston, pp.86-8.
28. **Report of the medical officer of the Local Government Board,** Parliamentary Papers, 1895, vol. 51, pp.137-79. This seasonality is confirmed in the seventeenth and eighteenth-century instances, recorded by Creighton, vol. 2, pp.635-6, 641, 646, 653.
29. See the authorities in note 25.
30. Rolleston, pp.80-4.
31. Christie, pp.382, 390, 395.
32. Calculated by comparing the total number of baptisms in the appropriate period with the total number of measles fatalities in this age group. Inaccuracy will be introduced by immigration, which would swell the size of the age cohort above that indicated by baptismal totals and by any other factor which provides measles burials for which there is no matching baptism. It should be noted that about 10 per cent (Table 2) of the burials in the first two epidemics have no matching baptism, which would suggest that the cohort could have been larger than the baptismal totals imply and that the fatality levels proposed in the text are about 10 per cent too high; against this one must set the total mortality from all causes between the date of baptism and the measles epidemic, which would surely be about, or greater than 10 per cent, and would leave the text levels as more likely to be under- than over-estimates.
33. In 1642 and 1648 there seems no room for omissions, but in 1635 and 1656 there are some suspicious clusters of unspecified child burials, but the numbers concerned are too small to be very significant.
34. In this period the total number of child burials is 42 per cent of the total number of baptisms, so the measles deaths account for 29 per cent of this mortality total.
35. Intense lactation inhibits the resumption of ovulation, and protracted breast feeding is often suggested as a major element in the lengthening of birth intervals in pre-industrial societies. A. Perez, P. Vela, R. Potter and G. S. Masnick, 'Timing and sequence of resuming ovulation and menstruation after childbirth', **Population Studies,** Vol. 25, 1971, pp.491-503.
36. For 1648 the baptismal register is too poorly kept to justify analysis.
37. This total includes five children whose baptism does not appear in the register but who can be fairly firmly identified.
38. One diarist of the 1660s was acutely aware of the risks to his children and agonised over their illnesses. 'A child of John Butler's dead and mine yet spared, blessed be God. M. H. Lee (ed.), **Diaries and letters of Philip Henry,** 1882, p.170. See also pp.85-6, 91, 168, 186.
39. Creighton, Vol. 2, p.634.
40. Ibid., pp.634-6.
41. **Shrewsbury St Mary:** Shropshire Parish Register Society 1911; **Stratford-upon-Avon:** Parish Register Society 1897; **Cloucester St Nicholas:** Gloucester County Record Office P.154/15; **Ludlow:** Shrops P.R.S. 1910. There is a very close parallel to the 1635-6 epidemic at Leeds (**Publications of the Thoresby Society,** Vol. 3, 1894, pp.360-6).
42. In 1735 measles could be seen spreading from village to village along the road to Edinburgh, Creighton, vol. 2, p.643.
43. Wrigley and Schofield, p.513.
44. Measles deaths in the north-west were often much higher than in eastern England in the nineteenth century: **Annual Reports of the Registrar General,** e.g. for 1841 and 1848.
45. G. M. Howe, **A world geography of human diseases,** 1977, pp.244-50; W. Butler, 'The fatality rate of measles', **Journal of the Royal Statistical Society,** vol. 108, 1945, pp.259-85.
46. A. B. Appleby, **Famine in Tudor and Stuart England,** 1978. It would be significant if both measles and nutrition could be shown to be markedly worse in northern England; Wrigley and Schofield suggest that the north-west was more liable to harvest failure at this date pp.677-9.