Health and Disease in Human History: A Journal of Interdisciplinary History Reader

Edited by Robert I. Rotberg

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Nutrition and Disease: The Case of London, 1550–1750

Historians of early modern Europe have often detected a connection between harvest failure and epidemic disease. Helleiner, for example, has written that “subsistence crises invariably engendered epidemic outbreaks” in late medieval Europe. Bowden, speaking of plague in Tudor England, noted that “the striking coincidence of serious plague outbreaks with harvest failures . . . leaves no doubt that these two events were closely related.” Contemporaries confirmed the relationship: “first dearth and then plague” was a common saying of the sixteenth and seventeenth centuries, as Meuvret has pointed out. It is easy to reconstruct the process. Following a harvest failure, food prices rose and the poor became progressively malnourished. They ate less and what they ate was worse, as they devoted their limited incomes to grain—which provided the most calories per penny—rather than to a better-balanced but more expensive diet of grains, meat, dairy products, fruits, and vegetables. Malnourishment lowered resistance to disease, allowing a disease already present endemically to grow to epidemic size or providing fertile territory for an epidemic introduced from outside. The

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2 The relative cost of all foodstuffs in early modern Europe is unknown. A study of relative food costs expressed in caloric terms for Paris in the 1780s shows that it cost eleven times as much to buy a certain number of calories in meat as in bread, three times as much in salt fish, six times in eggs, and so forth. Per calorie, bread was much the cheapest food. See R. Philippe, “Une opération pilote: l’étude du ravitaillement de Paris au temps de Lavoisier,” in J.-J. Hémardinquer (ed.), Pour une histoire de l’alimentation (Paris, 1970), 65.
epidemic in turn was spread rapidly by infected beggars that thronged
the roads seeking food. The two—food shortage and disease—went
hand in hand, during both the inception and diffusion of the epidemic.
This picture is plausible, so plausible as to seem beyond doubt.

Recent studies, however, have questioned one crucial link in this
causal chain. The role played by malnutrition in lowering resistance to
infectious disease appears less sure than common sense would suggest.
Physicians studying starvation in the Warsaw ghetto between 1941
and 1943 and also in Holland in 1945 were struck by the low incidence
of such deficiency diseases as scurvy and rickets and also by the relative
immunity of these starving people to many infectious diseases.\(^3\)
Historians, too, have expressed doubts about the assumed relationship
between malnutrition and disease. The late J. D. Chambers argued that
infectious disease in pre-industrial England had its own dynamic, largely
unaffected by food shortage, and that epidemics slowed, stopped, or
reversed population growth in periods of low food prices and, con-
versely, that demographic expansion often took place during periods
of high prices and undoubted shortage simply because epidemics were
not frequent or deadly.\(^4\)

The question is important, for if Chambers is correct, we would
have at least a partial explanation of why English population stabilized
at times—such as 1350 to 1470 and again from 1650 to 1690 and from
1720 to 1750—when grain prices were low and the standard of living
of the grain-dependent poor was relatively high. The statement of
Sylvia Thrupp that the period “from 1349 to the 1470’s, if it was a
Golden Age, was the golden age of bacteria” would be confirmed.\(^5\)
Disease abounded—and better nutrition in no way protected the poor
from periodic epidemics. The absence of any correlation between
disease and nutrition would also help to reconcile the evidence that
early industrialization led to a lowering of the workers’ standard of

\(^3\) Ancel Keys et al., *The Biology of Human Starvation* (Minneapolis, 1950; 2v.), II, 1011,
writes, in reference to the Warsaw ghetto: “among both children and adults epidemics
were very rare and, when they occurred, ran a benign course.” (Typhus fever should
be excepted from this observation.) See also *ibid.*, I, xv, 20, 448–453; II, 1009–1040;
Emil Apfelbaum, *Maladie de Famine* (Warsaw, 1946); Leonard Tushnet, *The Uses of
Adversity* (New York, 1966); Frederick Hocking, *Starvation* (Sydney, 1969). For the
view that malnutrition increases the virulence of infectious disease, see N. S. Scrimshaw,

esp. chs. 1, 4.

\(^5\) Quoted in *ibid.*, 22, 81–82.
living with the seemingly conflicting data that show a rapid rise in population and, for the first time, an excess of births over deaths in many of the larger cities, including London. It should perhaps be added here that no one denies that malnutrition lowers a human’s resistance to certain diseases, such as tuberculosis, or that certain diseases arise directly from dietary deficiencies, such as pellagra or scurvy. What has been questioned is the interplay between the great epidemic killers of pre-industrial Europe—plague, smallpox, typhus, influenza, to name four—and nutritional deficiencies.

The purpose here is to explore the relationship—if any—between malnutrition and certain specific diseases in London for the period 1550-1750, in the hope of adding to the available evidence. We will pay particular attention to plague from 1550 to 1680 and to other diseases from 1630 to 1750, a period when the authorities systematically identified ailments other than plague for the first time. The beginning date for the plague study is determined by the data on nutrition, which begin only in 1545. The ending date of 1680 marks the disappearance of plague from London. Separate mortality figures for typhus, “fever,” and smallpox were not given until 1629, which explains the starting date for these comparisons. Consumption figures are available from 1657. The study was not carried past 1728 for typhus and fever because they were lumped together after that date. The final cutoff of 1750 for the remaining diseases is somewhat arbitrary; we chose to limit this essay to pre-industrial England.

The two basic sources used in this study are London bread prices and London bills of mortality. Perhaps a word about these and the methodology used is in order.

The bread prices were the maximum permissible retail prices, in pence, that could be charged for a four-pound wheaten loaf, as set by the assize of bread. The price series is virtually complete and runs from 1545 to 1925. Our primary interest, however, is not in prices but in malnutrition. The problem is to relate these bread prices to possible caloric, protein, or vitamin deficiencies among the London

6 The literature on the standard of living during the industrial revolution is extensive; see E. J. Hobsbawm and R. M. Hartwell, “The Standard of Living during the Industrial Revolution: A Discussion,” Economic History Review, XVI (1963), 120–146. For urban births, see Chambers, Population, 103.

poor. We know that bread was the core of the poor man’s diet. Very likely bread purchases were his single most important outlay, exceeding what he spent on clothing or housing. But we do not know precisely how dependent the poor were on wheat bread, as differentiated from other breads or grains. Nor do we know to what extent the poor shifted from wheat to another, cheaper grain in times of dearth, or what other, non-grain substitutes might have been added to their diet. Despite these shortcomings in our knowledge, it is possible to show that London wheat bread prices accurately reflect the general level of nutrition. If these prices rose, the nutrition of the poor deteriorated, especially in periods of prolonged high prices. And it is in these prolonged shortages that one would expect to find a greater incidence of disease, if indeed malnourishment made men more vulnerable to infection.

Let us consider the problem of alternative foods that might have substituted for wheat bread when wheat prices were high. During a dearth, the poor no doubt ate more low-priced grains—oats, barley, and rye, if they were available—and less wheat, which always tended to be more expensive per measure. This tactic stretched their available money but would not have fended off malnutrition during a long, severe dearth. Some years ago, Hoskins argued that the price of other grains (and all other foods) tended to follow the price of wheat. Recently, this contention has been questioned by Harrison, who has shown that there were frequent divergences in grain price movements. But this does not mean that when the price of wheat went up, the poor simply switched to another, more modestly priced grain. All too often, in times of extreme wheat shortage all other grains rose swiftly in

8 Judging from the controls and regulations that all authorities throughout Western Europe set to cover virtually every transaction. No other commodity was so thoroughly regulated. See Jack C. Drummond and Anne Wilbraham, The Englishman's Food, A History of Five Centuries of English Diet (London, 1958), 41.
9 See Bowden, "Agricultural Prices," 601–602, 612. In 1641, Henry Best was selling oats at 14s. the quarter, barley at 22s. the quarter, rye at 27s. 6d. the quarter and white wheat at 35s. the quarter. See J. Thirsk and J. P. Cooper (eds.), Seventeenth-Century Economic Documents (Oxford, 1972), 356.
10 W. G. Hoskins, "Harvest Fluctuations and English Economic History, 1480–1619," Agricultural History Review, XII (1964), 40; C. J. Harrison, "Grain Price Analysis and Harvest Qualities, 1465–1634," ibid., XIX (1971), 138–143. According to our calculations, Harrison constructed his 31-year moving averages by adding the prices for the 15 preceding years, the year in question, and the following 15 years, making a correct total of 31 years. He then apparently divided by 30, instead of 31, which gave him a constant error of 3–4 percent. Anyone wishing to use Harrison’s averages should first check his calculations.
price, as a glance at Harrison's grain price graphs reveals. In general, a shortage of one grain meant the shortage of another, particularly when prices remained high for longer than one year.

The crises of 1555–56 and 1596–97 are cases in point. These two periods beyond doubt saw the worst harvest failures of the sixteenth century. In each year of these two crises, the prices of all grains were very high; sometimes wheat rose more than the other grains; sometimes their increase surpassed that of wheat. In 1555, wheat did not quite reach dearth levels but the average of all grains did, thanks to the incredible price of barley. (Here, "dearth" is specifically defined as 50 percent or more above the thirty-one-year moving average price of the grain in question.) The next year there was a dearth of wheat and oats. Barley prices moderated somewhat but remained higher than in any year prior to 1555. In 1596–97, the same pattern can be quickly discerned; 1596 was a terrible year, with wheat, barley, oats, and rye all reaching unprecedented levels. The next year was slightly improved but again all grains were well above their normal price. Perhaps it bears repeating: In bad years, particularly in a run of bad years, the poor could not turn from one grain to another and find it at a reasonable price, even assuming that it was available.

So much for grains. What about other alternatives, such as beans, peas, or dairy products? The poor stretched their bread grain with beans and peas and apparently consumed some milk, cheese, and butter in normal years. Both beans and peas were very dear during the two crises we have chosen as examples. Prices for dairy products are not available for 1555–56, but in 1596–97 they reached a new high, although the increase was not startling from a percentage standpoint. Cattle and sheep prices remained about average during the two crises but the poor man could not afford to replace his usual grain purchases with meat. Indeed, the relative price stability of cattle and sheep suggests that they were of minor importance in the diet of the poor. Had they been of any consequence, buying pressure would have forced prices higher. In this connection, it is interesting to note that wheat prices were less volatile than prices of the cheaper grains.

11 The following price data are drawn from Peter Bowden, "Statistical Appendix," in Thirsk, Agrarian History, 814–870. The quality designation for "dearth" is that used by Hoskins and Harrison in their articles. See also Bowden, "Agricultural Prices," 626.
bad year, the price of wheat had to rise only slightly to put it out of the
reach of the poor because initially it was expensive. Oats or barley, on
the other hand, started at a lower quantity price and would have had
to move up proportionally more before becoming too dear.

Less is known about other foodstuffs eaten by the poor. Salt fish
evidently played an increasing role in their diet during the sixteenth
century, although it was not available in quantities sufficient to sub-
stitute for bread in times of harvest failure. The same might be said of
fruits and vegetables. The poor in the late sixteenth century were
eating more vegetables than before and a further increase in fruit and
vegetable consumption was reported after the Restoration. But it is
doubtful that either fruits or vegetables were much of a factor in the
average diet. The orchards and gardens near London could not have
sufficiently expanded their output in the short term to offset the shortage
of other foods.

In sum, it seems highly unlikely that the poor had any cheap
alternative foodstuffs in time of grain shortage. Probably the poor
man tightened his belt and cut down on all purchases except basic
grains stretched with peas, beans, and other fillers. As William Harrison
said, the poor were reduced to living on “horsse corne, beanes, peason,
otes, tares & lintels” when the grain harvest failed. If this was true,
they faced a decline in both the quantity (in caloric terms) and quality
(in terms of nutritional value) of their food. Naturally, the degree of
malnutrition would have varied with the length and severity of the
shortage, becoming progressively greater the longer prices remained
high and the higher they went.

The other major source for this study, the London bills of mortality,
also contain certain strengths and weaknesses. Occasional bills survive
for the sixteenth century but usually show only plague deaths in years
of plague epidemics. Beginning in 1629, however, the bills regularly
show yearly total mortality for 130 London and adjoining parishes
from a variety of causes, being arranged under various disease or
accident headings. The headings occasionally were changed, to

14 Drummond and Wilbraham, Englishman’s Food, 29–30, 38–39, 55; Thirsk and
Cooper, Economic Documents, 80.
15 Quoted in Drummond and Wilbraham, Englishman’s Food, 88.
16 The origin of the bills is discussed in Thomas R. Forbes, Chronicle from Aldgate
from 1657 to 1758 inclusive . . . (London, 1759). Some earlier bills are included in this
volume but not the occasional bill for the sixteenth century. The number of parishes
varied, some being added from time to time as London grew.
reflect shifting patterns of disease or changing concerns of the authorities. Because they show only mortality and not morbidity, the bills are useful only for tracing fatal diseases. A widespread epidemic of influenza might have left all London prostrate but if it resulted in no deaths, it would have left no trace on the bills.

More important, the bills pose a problem of reliability. The cause of each death in each parish was reported to the parish clerk by the parish "searcher," often an old woman whose only qualification was her willingness to undertake an unpleasant task for a few pennies in pay. She would visit the house of the deceased, view the body, and perhaps discuss the cause of death with relatives or whomever might offer an opinion as to why the person had died. Because of this rather haphazard method of determining cause of death, certain allowances should be made for error. Do these errors invalidate the bills as a source of medical information? This seems to depend upon the disease under consideration. Certain diseases are easy to identify—smallpox, for instance—and mistakes in diagnosis would have been statistically insignificant. Plague was much feared and its symptoms were well known, particularly in a city like London where it was endemic for many years. However, plague was occasionally misdiagnosed; the jump in typhus deaths during a plague epidemic, for example, suggests that some plague cases were misidentified as typhus, through ignorance or fear of the magistrates. But here allowances can be made. Smallpox, plague, and typhus, can be confidently traced through the bills of mortality.

More difficult to assess would be deaths from "Ague and Feaver" or "Consumption and Tissick," to cite just two catchall headings used in the bills. Myriad ailments could be masquerading under the first and just about any lung disorder could come under the second. The identity of a great killer of children, "convulsions," is obscure, although the name apparently described the symptoms. In spite of these problems, the data in the London bills are probably more complete and accurate than any available elsewhere in England at that time.

Finally, it should be mentioned that London offers one great advantage to our study. The transmission of disease is complex,

17 Charles Creighton, *A History of Epidemics in Britain* (Cambridge, 1891), II, 534: "there is hardly anything more distinctive or more loathsome."
18 See Birch, *Bills of Mortality*, 11. The authorities would isolate a house to prevent the disease from spreading. Needless to say, many householders were anxious to avoid the inconvenience of being cooped up for a long period.
involving the twin poles of exposure to the disease and receptivity when once exposed. We are interested here only in receptivity—the resistance of the host—so that it would be advantageous to reduce exposure to a constant. London offers a closer approximation to this constant than would a rural area where exposure would have fluctuated enormously. This is not to say that everyone in an urban area such as London was exposed at all times to every possible contagious ailment. But from a statistical standpoint, it is as close as we can come to such a perfect constant, enabling us at least partly to isolate receptivity to disease.

The correspondence between plague and London bread prices is set out in Fig. 1. It should be noted that bread prices in this and subsequent figures and tables have been dated not by the harvest year but by the following year, when the prices actually would have prevailed. Thus, the harvest year of 1562 runs from the end of September, 1562, to the end of September, 1563. In a year of shortage, such as 1562–63, most of the privation would have fallen in 1563, rather than immediately following the harvest in 1562. Accordingly, we have dated the year 1563 to facilitate comparisons with disease. As the reader will see, the high prices of 1563 thus coincide with the plague epidemic of that year, rather than being off by one year.

We can find little correlation between high bread prices and plague epidemics in London. Between 1550 and 1670 five epidemics ravaged the city—in 1563, 1593, 1603, 1625, and 1665. A lesser epidemic struck in 1636.\(^\text{19}\) Of these epidemics, only one fell in a year of real shortage: 1563, when the price of bread soared 65 percent above normal. In the plague year of 1625, prices were 20 percent above normal. Apart from these, prices in epidemic years were average or below average.\(^\text{20}\)

What might be termed the "reverse" correlation is even less apparent. Years of death did not lead to plague epidemics, even though the disease smoldered endemically in the city. Periods of

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\(^{20}\) "Normal" is here defined as the 15-year moving average price of bread, i.e., prices for seven years back, seven forward, and the year in question, added together and then divided by 15. There are too many gaps in the material to use a 31-year moving average as did Hoskins, "Harvest Fluctuations," 43.
Fig. 1  London Bread Prices and Dates of Plague Epidemics

price in pence per 4-lb. loaf

1670
1660
1650
1640
1630
1620
1610
1600
1590
1580
1570
1560
1550
1540
1530

10.0  9.0  8.0  7.0  6.0  5.0  4.0  3.0  2.0  1.0

1665
1636
1625
1593
1603
1563

YEAR
terrible shortage in the 1550s, the late 1590s (when real wages fell to their lowest point), the late 1640s, and the early 1660s did not trigger epidemics, although the poor must have suffered severe malnutrition. During all of these food crises, except possibly during the dearth of the 1550s,21 plague was endemic to the city. If deteriorating dietary levels meant falling resistance, and if resistance in turn was a factor, plague epidemics would have broken out. But none did. In the grave food crisis of the late 1640s, plague diminished in intensity as the price of bread rose. The first of five bad years, 1647, saw considerable disease in the city, but as the subsistence crisis worsened, the numbers dying of plague declined dramatically, as Table 1 shows:

<table>
<thead>
<tr>
<th>YEAR</th>
<th>PLAGUE DEATHS</th>
<th>BREAD PRICES IN PENCE PER 4-LB. LOAF</th>
</tr>
</thead>
<tbody>
<tr>
<td>1644</td>
<td>1,492</td>
<td>5.1</td>
</tr>
<tr>
<td>1645</td>
<td>1,871</td>
<td>4.8</td>
</tr>
<tr>
<td>1646</td>
<td>2,436</td>
<td>5.3</td>
</tr>
<tr>
<td>1647</td>
<td>3,597</td>
<td>6.8</td>
</tr>
<tr>
<td>1648</td>
<td>611</td>
<td>9.0</td>
</tr>
<tr>
<td>1649</td>
<td>67</td>
<td>8.6</td>
</tr>
<tr>
<td>1650</td>
<td>15</td>
<td>9.0</td>
</tr>
<tr>
<td>1651</td>
<td>23</td>
<td>7.4</td>
</tr>
<tr>
<td>1652</td>
<td>16</td>
<td>6.4</td>
</tr>
<tr>
<td>1653</td>
<td>6</td>
<td>4.8</td>
</tr>
</tbody>
</table>

In short, the London evidence accords with the statement of the French demographer and medical doctor, J.-N. Biraben, that "famine does not seem to increase the virulence of plague by diminishing, for example, an individual's resistance. The plague is a sufficiently grave disease that all those who contract it, even the well-nourished, have little chance of recovery."22


22 Annales de Démographie Historique, 1968 (Paris, 1968), 15. Statistically, the correlation coefficient between the independent variable, London bread prices, and the dependent
Turning from plague to other diseases, we find a somewhat confused picture in the period 1630–1750. Bread prices did not continue to rise as they had throughout the sixteenth century. Extreme short-term fluctuations continued, however, and must have brought great distress to the poor, even though the secular price trend was no longer disadvantageous. The periods that would have known the greatest malnutrition—and therefore a heightened incidence of disease if a connection exists between the two—were in the years 1647 through 1651, 1658 through 1663, 1693 through 1699 and 1709 through 1710. Isolated bad years, such as 1631, would not have been as great a threat to health as these periods of prolonged high prices.

If we look in detail at Fig. 2, which charts the deaths from “spotted fever,” that is, typhus, against the bread prices, we find no rise in deaths during the terrible late 1640s. There was a greater incidence of disease in the dearth of the late 1650s and early 1660s, but deaths were low in the dreadful year of 1662 (the 1661 harvest year), when prices reached their high point for the century, 59 percent above normal.23 The death totals attributed to typhus in 1636 and 1665 should be viewed with suspicion; probably they were plague deaths mistakenly attributed. It is possible that the two killers were present at the same time. If so, there was no connection with the price of bread, which had fallen well below its average.

Later in the seventeenth century a correlation between bread prices and typhus mortality becomes more apparent.24 The years 1674, 1694, 1698, and 1710 saw both high prices and high mortality. It is difficult to see why a difference exists between the first part of the period and the last, that is, why no price-mortality correlation is discernible until about 1660. It seems unlikely that the disease changed its character and suddenly began to affect only malnourished individuals. During the sixteenth century the disease had been recognized as a close companion of famine; it was one of the so-called “famine

variable, plague deaths, for all 50 years from 1629 through 1681 when both figures are available is −.059. This is statistically insignificant at the 5 percent confidence level. All prices were adjusted one year forward, as in the graphs, to correspond to the year when most of the harvest results would be felt. We would like to thank the Economic Resources Laboratory and the Computer Center, both of San Diego State University, for their help with these computations.

23 See footnote 20 above.
24 For the 89 years from 1629 through 1728 when both price and typhus mortality data are available, the correlation coefficient is .144. This is statistically insignificant at the 5 percent confidence level.
Fig. 2 London Bread Prices and Typhus ("Spotted Fever") Mortality 1665-1929 deaths

deaths (in absolute numbers)

1730
1720
1710
1700
1690
1680
1670
1660
1650
1640
1630

YEARS

bread price

mortality

Price in pence per 4-lb. loaf
fevers.” Possibly the answer lies in the years during the 1630s and 1640s for which we have no mortality figures. Typhus has two characteristics that bear on the question: The disease confers immunity on its survivors, and children sicken but seldom die from it.

If there was a widespread epidemic a few years prior to the death of 1647–51, the pool of infectables might not have grown to the size necessary for another epidemic. This would have been particularly true if large numbers of children had been immunized by the earlier epidemic. If this conjectural explanation has any validity, it suggests that normally there was a correlation between nutritional levels and the spread or virulence of typhus, but that the lack of sufficient numbers of susceptible persons made this connection inoperable in the dearth of the late 1640s.

Typhus mortality seems to have tapered off after 1700 and the keeping of separate death figures was discontinued for the disease after 1728. The decline in the per capita incidence of the disease occurred earlier, for the number of typhus deaths remained surprisingly constant from 1630 to 1728, at a time when the city grew substantially. Quantitatively typhus was not an important killer at any time, except in the suspect year, 1665.

Smallpox mortality (Fig. 3) seems to have had no correlation with bread prices. High prices and heightened mortality sometimes coincided, as in 1674 and 1710, but these appear to have been random. The course of smallpox deaths was a jagged, sawtooth affair, with minor epidemics occurring regularly about once every three years. If, for some reason, deaths were fewer than usual for several years, a larger epidemic invariably followed. This seems to explain the heightened death tolls in 1674 and 1710. Like typhus, smallpox conferred immunity on those fortunate enough to survive, but, unlike typhus, children

25 Typhus was the one epidemic disease usually present in the ghettos of occupied Poland. See Isaiah Trunk, *Judenrat* (New York, 1972), 143–172. Famine may be associated with typhus because famine discourages cleanliness, which in turn encourages body lice, the carriers of typhus. Famine can also promote crowding (for example, when beggars thronged into a city seeking charity) which helps to spread the disease. Resistance to typhus may not depend upon on the nutrition of the host.


27 For the 111 years from 1629 through 1750 when both price and smallpox mortality data are available, the correlation coefficient is −.145. This is statistically insignificant at the 5 percent confidence level.
Fig. 3  London Bread Prices and Smallpox Mortality

in pence per 4-lb loaf

bread price

mortality

YEARS

1630  1640  1650  1660  1670  1680  1690  1700  1710  1720  1730

deads (in absolute numbers)

0  1000  2000  3000  4000
had no special resistance to it. Since almost everyone in London was exposed in childhood to smallpox, only children provided a source of unexposed victims. At regular intervals an epidemic attacked those children who had not previously been exposed. As the graphs indicate, smallpox mortality was quantitatively important, running almost ten times that of typhus. The searchers had no difficulty differentiating between smallpox and plague, for in neither 1636 nor 1665 were smallpox deaths elevated, as typhus deaths had been.

"Ague and fever" was another major killer of the time. Probably both influenza and malaria would have fallen into this category. Aside from these two, it is impossible to say exactly what diseases seventeenth- and eighteenth-century physicians included under that heading, and it is extremely unlikely that the searchers had any specific idea of what diseases to include or exclude. Whatever collection of ailments the terms encompassed, "ague and fever" killed more people in the average year than smallpox. As with typhus, the correlation between "ague and fever" mortality and bread prices is nonexistent in the 1640s and early 1650s, but thereafter some correlation can be found, although it is far from consistent. As Fig. 4 reveals, there is a divergence of the two after 1710. The mortality from ague and fever rose slowly throughout the period, paralleling the increase in the city's population.

In the 1650s and 1660s, "consumption and tissick" was the greatest killer in London, carrying off between three and four thousand persons per year. "Tissick" apparently is an antiquated spelling of phthisis, another name for pulmonary consumption, or tuberculosis. Fig. 5 sets out the correlation between consumption and bread prices from 1650 to 1750. Consumption and other pulmonary ailments, such as emphysema, are generally acknowledged to be extremely sensitive to nutrition. A correlation between deaths and bread prices would be

29 Chambers thought it noteworthy that the "seven ill years" in the 1690s were relatively healthy years (Population, 91–94). In London, the generally low level of mortality from the great epidemic diseases in the 1640s seems equally surprising. The prolonged bad years from 1647 through 1651 provoked no heightened mortality from typhus, smallpox, or fever, although the degree of malnutrition probably was greater than at any other time in the seventeenth century.
30 The correlation coefficient for all 89 years from 1629 through 1728 for which figures are available is −.020. This is statistically insignificant at the 5 percent confidence level. E. A. Wrigley, "A Simple Model of London's Importance in Changing English Society and Economy 1650–1750," Past & Present, 37 (1967), 45, offers the following round figures for London's population: 200,000 in 1600; 400,000 in 1650; 575,000 in 1700; 675,000 in 1750.
Fig. 4  London Bread Prices and “Aague and Fever” Mortality

Price in pence per 4 lb. loaf

Deaths (in absolute numbers)

YEAR
Fig. 5  London Bread Prices and Mortality from Consumption

Deaths (in absolute numbers)

Bread price

Mortality

Price in pence per 4-lb. loaf

Year
expected, particularly following a prolonged dearth, such as that of the 1690s. But here again, any correlation is questionable. The graph of consumption shows deaths rising gradually until about 1692, then falling for about thirty years before once again ascending, this time to new heights. What may have prompted the decline in consumption mortality in the first three decades of the eighteenth century? Prices were both more volatile and also absolutely higher than later in the 1730s and 1740s when consumption deaths again increased. Possibly weather played a role; winters may have been milder in the years of low mortality. Further research needs to be done to isolate each environmental factor. But all in all, it is hard to see any connection between consumption and bread price levels in this one historical instance.

It would be a mistake to push our evidence too far. The identification and classification of disease was far from sure. The 1665 mortality figures shown for typhus, fever, and consumption all suggest that plague deaths were falsely attributed to each of these. Such mistakes hardly inspire confidence in the evidence. On the other hand, the various diseases follow approximately the pattern one would expect, aside from any correlation with bread prices. The pattern for smallpox, for example, was the logical one for a disease that virtually all Londoners were exposed to early in life—small epidemics, recurring every few years, and striking only the previously unexposed children. The picture of consumption mortality, too, is as expected: only small variations from year to year in the death toll from this endemic, wasting disease.

We have suggested that disease mortality levels corresponded to bread prices in two instances: typhus and “ague and fever.” Certainly neither correspondence can be said statistically to be established, but only in these two was there any correspondence. As we noted before, typhus was an insignificant disease from a demographic standpoint. “Ague and fever” was much more important but it too was dwarfed

31 Tushnet, Uses of Adversity, 54, 60. The correlation coefficient between bread prices and consumption mortality was —.066 for the 94 years from 1657 through 1750. This is statistically insignificant at the 5 percent confidence level.
32 The deaths attributed to consumption may, of course, have been caused by pneumonic plague. Both diseases involve the coughing of blood, although the course of pneumonic plague is much more rapid than that of consumption.
33 See notes 24 and 30, above. The statistical correlation of fever mortality to bread prices was actually slightly negative. Any positive correlation appears only on the graph.
by the myriad of other diseases which show no correspondence to bread price levels. The greatest killer in the eighteenth century was "convulsions," a childhood disease (or group of diseases) that killed a progressively greater number of children each year, at a rate unaffected by long-term falling bread prices.

Environmental factors appear to have been less important than one might expect in the course of London mortality. The bread price level was not determining, although these prices were a suitable index of nutritional levels. Another environmental factor that should have played a considerable role was the density of population, or the crowding factor. As London grew, mortality from epidemic disease should have increased disproportionally, because the chances of exposure were greater. But this, too, seems doubtful. Plague disappeared, and typhus declined in importance.

The relative unimportance of environmental factors in London suggests that it was possible for disease to have been extremely virulent in the fifteenth century, despite relatively low population density and relatively good diet, and quite mild during the early industrial revolution, despite increased population density and perhaps deteriorating nutrition. Largely independent of environmental factors, the course of disease may have to be treated as an autonomous influence on population growth.

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34 For reasons that remain unclear.
35 In another place, we have argued the complement to this view: that starvation, unassisted by disease, could determine population change. See "Disease or Famine? Mortality in Cumberland and Westmorland, 1580–1640," *Economic History Review*, XXVI (1973), 403–432.
Appendix A  London Mortality, 1629–1750®

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® The absence of figures indicates the absence of data. Zero mortality is shown as zero. The headings have been modernized. In the bills of mortality, the headings change from time to time. For example, what we have labelled typhus was called “purples and spotted fever” in 1629, “spotted fever and purples” in 1657, “spotted fever” in 1670, “spotted fever and purples” in 1673, then again “spotted fever” in 1675, only to merge with “ague and fever” in 1729 in a grouping including fever, “malignant fever, spotted fever and purples” in 1729.
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<td>1724</td>
<td>—</td>
<td>84</td>
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<td>3,262</td>
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<td>59</td>
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<td>1,569</td>
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<tr>
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<td>102</td>
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<tr>
<td>1728</td>
<td>—</td>
<td>94</td>
<td>2,105</td>
<td>4,716</td>
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</tr>
<tr>
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<td>—</td>
<td>—</td>
<td>2,849</td>
<td>5,235</td>
<td>3,544</td>
</tr>
<tr>
<td>1730</td>
<td>—</td>
<td>—</td>
<td>1,914</td>
<td>4,011</td>
<td>3,728</td>
</tr>
<tr>
<td>1731</td>
<td>—</td>
<td>—</td>
<td>2,640</td>
<td>3,225</td>
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<td>1732</td>
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<td>1,197</td>
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<tr>
<td>1733</td>
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<td>—</td>
<td>1,370</td>
<td>3,831</td>
<td>4,601</td>
</tr>
<tr>
<td>1734</td>
<td>—</td>
<td>—</td>
<td>2,688</td>
<td>3,116</td>
<td>4,139</td>
</tr>
<tr>
<td>1735</td>
<td>—</td>
<td>—</td>
<td>1,594</td>
<td>2,544</td>
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</tr>
<tr>
<td>1736</td>
<td>—</td>
<td>—</td>
<td>3,014</td>
<td>3,361</td>
<td>4,554</td>
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<td>—</td>
<td>2,084</td>
<td>4,580</td>
<td>4,441</td>
</tr>
<tr>
<td>1738</td>
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<td>—</td>
<td>1,590</td>
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<tr>
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<td>—</td>
<td>1,690</td>
<td>3,334</td>
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<tr>
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<td>—</td>
<td>—</td>
<td>2,725</td>
<td>4,003</td>
<td>4,919</td>
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<tr>
<td>1741</td>
<td>—</td>
<td>—</td>
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<td>7,528</td>
<td>4,981</td>
</tr>
<tr>
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<td>—</td>
<td>—</td>
<td>1,429</td>
<td>5,108</td>
<td>4,716</td>
</tr>
<tr>
<td>1743</td>
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<td>—</td>
<td>2,029</td>
<td>3,837</td>
<td>4,353</td>
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<tr>
<td>1744</td>
<td>—</td>
<td>—</td>
<td>1,633</td>
<td>2,670</td>
<td>3,865</td>
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<tr>
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<td>—</td>
<td>—</td>
<td>1,206</td>
<td>2,690</td>
<td>4,015</td>
</tr>
<tr>
<td>1746</td>
<td>—</td>
<td>—</td>
<td>3,236</td>
<td>4,167</td>
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<tr>
<td>1747</td>
<td>—</td>
<td>—</td>
<td>1,380</td>
<td>4,779</td>
<td>4,560</td>
</tr>
<tr>
<td>1748</td>
<td>—</td>
<td>—</td>
<td>1,789</td>
<td>3,981</td>
<td>4,487</td>
</tr>
<tr>
<td>1749</td>
<td>—</td>
<td>—</td>
<td>2,625</td>
<td>4,458</td>
<td>4,623</td>
</tr>
<tr>
<td>1750</td>
<td>—</td>
<td>—</td>
<td>1,229</td>
<td>4,294</td>
<td>4,543</td>
</tr>
</tbody>
</table>
Mortality and Family in the Colonial Chesapeake

Colonial Chesapeake society is currently receiving a vigorous reexamination. After a decade of important demographic studies written about New England communities in the seventeenth and eighteenth centuries, social historians have begun to explore the largely neglected areas of population growth, life expectancy, and family structure in early Virginia and Maryland. Some of the research is unpublished, but the intensity and quality of recent work on early Chesapeake society suggest that the findings of these social historians will be of major importance to students of colonial America. When completed, this reassessment of the Chesapeake should underscore the differences between the social history of early New England and the colonial South.

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The author would like to thank Kevin Kelly, Darrett B. and Anita H. Rutman, Daniel Scott Smith, Russell R. Menard, and Allan Kulikoff for their useful comments on an earlier version of this essay.


The scarcity of adequate population sources for colonial Virginia and Maryland, such as parish registers, censuses, and tithable lists, has confined research to only a very few counties along the tobacco coast. Nevertheless, studies completed thus far have already come to some significant conclusions about the key demographic trends in the early Chesapeake. These may be briefly summarized as follows: the combination of a high mortality rate among immigrants and natives—especially the former—and a persistent shortage of females stifled natural population increase in the Chesapeake colonies during the seventeenth century. As a result, population growth in early Virginia and Maryland depended almost entirely on the influx of immigrants whose numbers peaked near the third quarter of the century. It was not until around 1700 that improved life expectancies, an earlier mean age at marriage for native women—thereby increasing the number of childbearing years—and more balanced sex ratios—due in part to a decline in the largely male stream of white immigrants—permitted most regions


in the Chesapeake to achieve significant natural increase for the first time. More important for population growth, slave importations rose precipitously beginning in the late seventeenth century and continued strong in the early eighteenth century. Thus both components of population growth—sustained natural increase and rapid immigration (in the form of black slaves)—emerged at roughly the same time, providing the Chesapeake colonies with steadily expanding populations by 1700.

Clearly the central demographic phenomenon in the early Chesapeake was low life expectancy produced in large part by an endemic malarial environment. In this disease environment men and women struggled with chronic sickness and the prospect of early death. As difficult and short as existence seems to have been in the colonial Chesapeake, there is evidence for at least one area of early Virginia that even higher levels of mortality prevailed.

The demographic history of Charles Parish in York County, Virginia, suggests that the expectation of life for men was considerably lower than in other areas of the Chesapeake and far below what has been discovered for colonial New England communities. And in contrast to the basic demographic trend in the Chesapeake of gradually improving mortality schedules by the eighteenth century, Charles Parish’s persistently high death rate created a population that barely managed to reproduce itself throughout the colonial era. In short, the mortality experience of Charles Parish indicates that this part of tidewater Virginia must have been one of the unhealthiest areas in the colonial Chesapeake.

Charles Parish was established around 1645 between the Poquoson River and Back Creek in the eastern end of York County. It was but one of four parishes in the county during the latter half of the seventeenth century and one of three after 1706 when two other parishes in the county were merged. Unfortunately, the absence of tax lists or tithable lists makes it impossible to


5 Charles Parish was called New Poquoson and New Towson until 1692 when it was renamed Charles Parish after the renaming of the New Poquoson River as Charles River in the same year. York County Deeds, Orders, Wills, etc., no. 9, (1691-1694), 227, Virginia State Library, Richmond.
measure the size of the parish population during the colonial era.\textsuperscript{6} The survival of Charles Parish’s birth and death registers, however, which run without significant interruption from the mid-seventeenth to the mid-eighteenth centuries, permits a unique glimpse of the effect of mortality on the pattern of natural increase in this Virginia parish community.\textsuperscript{7}

Although neither the birth nor the death register for Charles Parish (especially the latter) is free from the problem of underrecording of vital events, there is sufficient evidence to assume that, except for the recording of infant deaths (which will be dealt with later in the essay), underregistration was not so great as to seriously distort an analysis of natural growth in the parish. For example, demographers have suggested that an important test of the accuracy of a set of birth records is the calculation of sex ratios at birth in a given population (the number of male births for every 100 female births recorded). A normal range for the sex ratio at birth runs between 102 and 108, with a typical figure of 105.\textsuperscript{8} In fact, the sex ratio for children listed in the Charles Parish birth register was calculated at 107.0 for the entire period 1660 to 1760, suggesting at the very least that no distortions occurred in the registration by sex. That births were conscientiously recorded in the parish register receives further support from the fact that only 6

\textsuperscript{6} A study of the 1702 tithable list and the 1704 quit rent roll for York County indicates that Charles Parish contained between 300 and 350 tithes in 1702. Using a multiplier of 2.57 suggested by Edmund Morgan for York County in 1699 ("Headrights and Headcounts," 367-368), one can estimate that Charles Parish may have had a population of between 770 and 900. If so, Charles Parish was a relatively small parish in early eighteenth-century Virginia. The mean size of parishes reporting their tithes in 1702 (28 of 49) was 468 tithes, or about 1258 souls (using the 2.57 multiplier). The tithable figures were drawn from "Public Officers in Virginia," 373-377.

\textsuperscript{7} The Charles Parish registers which span the year 1648 to 1789 have records beginning before any other parish in Virginia. Seven clerks and nine ministers served the parish without absence during this century and a half. And except for a two-year gap in the death register in 1715 and 1716 (due to a lost register), there do not appear to be any defects in the records. Before 1660 and after 1760, however, the number of annual vital events in the registers is too small for demographic analysis. The original registers do not exist for births recorded during the period 1648 to 1714 and for deaths recorded from 1665 to 1725. But copies of these earlier registers were made by a Charles Parish clerk in 1716 and 1725, respectively, and are available in photostatic copy at the Virginia State Library, Richmond. For further information on the history of Charles Parish and its registers, see Landon C. Bell’s introduction to the published version of the registers, Charles Parish, York County, \textit{Virginia, History and Registers} (Richmond, 1932), 1-42.

percent of the children named in York County wills as Charles Parish residents are not listed in the birth register. 9

As a later discussion of mortality will indicate more precisely, the Charles Parish death register is less reliable. Emigration and the common failure to register the deaths of infants contributed to the incompleteness of parish death records. The underregistration of deaths may have been offset, however, by the influx of immigrants into the parish whose deaths were added to the death register but whose births were not recorded in the birth register. 10 Moreover, a close examination of the Charles Parish death register suggests that it was kept with considerable care—relatively few deaths (6.2 percent) are listed out of chronological order and no serious gaps in recording are discernible. 11 The uncertain extent of underregistration in the parish records prevents any precise measurement of natural growth but the likelihood that deaths were less diligently recorded than births only underscores the pattern of slow natural population increase described below.

It is clear from the registers that reproductive population increase was rare in colonial Charles Parish. As displayed in Fig. 1, Charles Parish grew almost imperceptibly until the early 1680s. Indeed, from 1665 to 1680 births exceeded deaths (256 to 217) for a net natural increase of only 39 in these 15 years, a gain of less than 3 per year. Heavy mortality impeded natural growth as the deaths recorded in this period comprised a high 84.8 percent of the number of births recorded during the same years.

Charles Parish failed to achieve any significant natural growth in the first half of the eighteenth century as well. In fact, during the three decades from 1695 to 1724, the ratio of deaths to births grew to 79 percent, up over 9 percent from the previous thirty-year period (Table 1). And as Fig. 1 shows, mortality reached its high-

9 York County Deeds, Orders, Wills, etc., nos. 3–18, photostat, Virginia State Library, Richmond. This 6% figure may be too high, since some of these children named in wills but missing from the birth register may have been born elsewhere before their families entered Charles Parish.
11 Two years are missing from the death register, 1715 and 1716. The number of deaths for these years has been estimated based upon the mean number of annual deaths during five-year periods surrounding the missing records: 1710–1714 and 1717–1721.
Fig. 1  Births and Deaths in Charles Parish, Virginia, 1660–1760
(Three-year running average)

Births

Deaths
est level in the parish’s history during 1717 and 1718. In these two years 117 deaths were recorded compared to only 58 births. Most of the deaths (62.4 percent) occurred between November and the end of March, indicating that a particularly deadly winter epidemic swept through the parish in these years. In the final thirty-year period, 1725–1754, parish mortality remained high with deaths comprising 72.4 percent of the recorded births, about the same proportion of deaths to births as in the latter third of the seventeenth century (Table 1).

Table 1  Natural Increase in Charles Parish, Virginia, 1665–1754

<table>
<thead>
<tr>
<th>PERIOD</th>
<th>TOTAL BIRTHS RECORDED</th>
<th>TOTAL DEATHS RECORDED</th>
<th>RATIO OF DEATHS TO BIRTHS IN PERCENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1665–1674</td>
<td>165</td>
<td>100</td>
<td>60.6</td>
</tr>
<tr>
<td>1675–1684</td>
<td>195</td>
<td>148</td>
<td>75.9</td>
</tr>
<tr>
<td>1685–1694</td>
<td>272</td>
<td>210</td>
<td>77.2</td>
</tr>
<tr>
<td>Total 1665–1694</td>
<td>632</td>
<td>458</td>
<td>70.2</td>
</tr>
<tr>
<td>1695–1704</td>
<td>284</td>
<td>239</td>
<td>84.2</td>
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<tr>
<td>1705–1714</td>
<td>300</td>
<td>126</td>
<td>42.0</td>
</tr>
<tr>
<td>1715–1724</td>
<td>254</td>
<td>302&lt;sup&gt;a&lt;/sup&gt;</td>
<td>118.9</td>
</tr>
<tr>
<td>Total 1695–1724</td>
<td>838</td>
<td>667</td>
<td>79.5</td>
</tr>
<tr>
<td>1725–1734</td>
<td>267</td>
<td>168</td>
<td>62.9</td>
</tr>
<tr>
<td>1735–1744</td>
<td>280</td>
<td>213</td>
<td>76.1</td>
</tr>
<tr>
<td>1745–1754</td>
<td>185</td>
<td>149</td>
<td>80.5</td>
</tr>
<tr>
<td>Total 1725–1754</td>
<td>732</td>
<td>530</td>
<td>72.4</td>
</tr>
<tr>
<td>Totals</td>
<td>2202</td>
<td>1655</td>
<td>75.2</td>
</tr>
</tbody>
</table>

<sup>a</sup> Because of a two-year gap in the Charles Parish death register for the years 1715 and 1716, the number of deaths in this period is an estimate. See note 11.

Source: Charles Parish Registers, Virginia State Library, Richmond.

All of which stands in contrast to what is known thus far about natural increase elsewhere in the early Chesapeake. Most counties in Virginia and Maryland managed to achieve rapid natural growth by the 1690s. And there is some indication that at least in the early eighteenth century other Virginia parishes did not experience the withering mortality that prevailed in Charles Parish. Two extant lists of births and deaths in Virginia parishes

12 Walsh and Menard, “Death in the Chesapeake,” 220.
for 1714 and 1725 reveal that recorded deaths made up 38.3 percent of the births in all parishes in these two years, while Charles Parish showed a much higher mortality with 69.9 percent of its births cancelled by deaths.13 Because of the unknown quality of death registration in other parishes, these figures are only rough estimates of the varying mortality levels in early eighteenth-century Virginia. But they do suggest that rapid natural increase may have come more slowly to Charles Parish than to most regions of the early Chesapeake.

Although the pattern of natural increase in Charles Parish does not quite fit that of the colonial Chesapeake, its mortality experience diverges significantly from the demographic trends reported for early New England towns. The number of deaths in Andover, Massachusetts, for example, never exceeded 24 percent of the births recorded for any period until the eighteenth century—about one third of the ratio of deaths to births in Charles Parish.14 Actually, the sluggish growth of colonial Charles Parish more closely resembled the pattern of mortality of the English villages Clayworth and Colyton in the seventeenth century and Barbados and Jamaica in the seventeenth and eighteenth centuries, where burials nearly always outnumbered baptisms.15

What explains the near failure of natural increase in Charles Parish during most years of the colonial period? The shortage of women in early Virginia was partly responsible for limiting growth. Estimates of sex ratios during the seventeenth century indicate that men outnumbered women by as much as three or four to one, resulting in a large pool of unmarried men whose potential fertility could not be used for the colony’s growth.16 Indeed, an examination of the headright entries for seventeenth-

13 The lists are in C.O.5/1317/127–129; C.O.5/1320/74, Colonial Virginia Records Project, University of Virginia. The figure 75.9% for Charles Parish represents the mean proportion of deaths to births between 1705 and 1725.
14 Greven, Four Generations, 89.
16 Morgan, American Slavery, 407.
century York County shows that from 1620 to 1660 male immigrants settling in Charles Parish outnumbered female settlers by four to one and in the county as a whole the sex ratio (the number of males for every 100 females) was 460. In the subsequent forty years, from 1660 to 1700, the sexes became more balanced among the immigrant population, as the sex ratio for Charles Parish dropped to 268.9, while that of the county fell to 273.7.\textsuperscript{17} Despite this drop in the surplus of men (due mainly to an abrupt decline by the late seventeenth century in the predominantly white male immigration to Virginia), men continued significantly to outnumber women in the early eighteenth century.\textsuperscript{18}

The chief hindrance to growth in Charles Parish, however, was the unusually low life expectancy in the area. The high death rates also had an important impact on the life of the family. Data on life expectancy in Charles Parish was obtained from a variety of sources. The reconstitution of all families listed in the birth and death registers was an essential beginning. But because of name confusion in the records (children often being given the same first name as their parents or other relatives), information was gathered from a number of local and provincial records—such as wills, deeds, inventories, land patents, rent rolls, court order books, and genealogies—to insure the proper identification of individuals in the parish. Unfortunately, no marriage register has survived for Charles Parish (thereby preventing one from tracing the careers of women in the records after marriage); consequently, the analysis of adult life expectancy was confined to the men of the parish.

Children were probably the most susceptible to the parish disease environment. Although the persistent problem of under-registration of infant deaths (children dying before age one) masks the full extent of infant and childhood mortality (ages 0 to 14) in the parish, the existing evidence suggests that a large proportion of children did not survive to adulthood.

The central difficulty in estimating infant mortality is that most children who did not survive their first year died within the

\textsuperscript{17} Headright records for York County are in Nell M. Nugent, \textit{Cavaliers and Pioneers: Abstracts of Virginia Land Patents and Grants} (Richmond, 1934), I; Virginia State Land Office, Land Patents, Books 5-9, passim.

\textsuperscript{18} Robert V. Wells, \textit{The Population of the British Colonies in America before 1776} (Princeton, 1975), 154. Both the headrights and county servant importation records for York County suggest that by the early 1680s white immigration to the county had come almost to a standstill.
first few weeks of life and received private burials which were usually not recorded in the parish death register. To develop a good estimate of infant mortality, then, one must try to establish the proportion of early infant deaths omitted from the death register. Louis Henry has created a useful technique for estimating unrecorded infant deaths through an analysis of birth intervals. His method assumes that if a child dies in his first year, the interval between this and the next birth will be smaller than if the child had lived beyond the first year. Using his formula, I calculated that 50 percent of all infant deaths were not recorded in the Charles Parish death register. Thus, two sets of infant and childhood death rates are presented in Table 2—one computed from the raw data and one computed after the mortality figures for the first year were increased by 50 percent.

The unadjusted infant death rates in Charles Parish of 66 per 1000 for males and 92 per 1000 for females during the last third of the seventeenth century are extremely low compared to New England and England during the same period. Despite the apparently healthier climate of seventeenth-century New England communities, Andover and Ipswich, Massachusetts, had higher infant death rates of 115 and 112 respectively. In the English village of Colyton infant mortality was between 118 and 147 in the second half of the seventeenth century. Clearly the adjusted infant death rates in Charles Parish of 132.2 for males and 184.6 for females, although probably still too low, more accurately reflect the parish’s high mortality during the seventeenth century.

The death rates for children in the age group 1 to 4 are of particular importance for understanding mortality in Charles Parish. In most populations infants die in much larger proportions than the children who survive their first year. But as the Rutmans have recently discovered, inhabitants of the early Chesapeake endured a severe malarial environment which created important age-specific mortality rates. In this disease environment, infants frequently received from diseased mothers a short-term immunity to malaria which allowed many to survive infancy only later to succumb to

19 Wrigley, “Mortality in Pre-Industrial England,” 564–566.
21 Wrigley, “Mortality in Pre-Industrial England,” 571.
the disease as small children when their immunity had worn off.\textsuperscript{22} Thus, we find Charles Parish boys aged 1 to 4 dying at almost the rate of male infants during the last third of the seventeenth century, while girls in this age group experienced a higher death rate than female infants (Table 2).

\begin{table}[h]
\centering
\begin{tabular}{lrr}
\hline
\multicolumn{3}{l}{\textbf{Table 2} Life Table Death Rates of Infants and Children in Charles Parish, 1665–1734\textsuperscript{a} (per 1000)} \\
\hline
\textbf{MALES BORN 1665–1699} & Raw Data & Adjusted \\
\hline
0–1 & 66.1 & 132.2 \\
1–4 & 107.0 & 117.9 \\
5–9 & 28.3 & 32.1 \\
10–14 & 38.4 & 57.1 \\
1–14 & 133.3 & 145.4 \\
\hline
\textbf{FEMALES BORN 1665–1699} & Raw Data & Adjusted \\
\hline
0–1 & 92.8 & 184.6 \\
1–4 & 149.0 & 191.2 \\
5–9 & 67.5 & 84.6 \\
10–14 & 57.1 & 60.3 \\
1–14 & 174.9 & 198.0 \\
\hline
\textbf{MALES BORN 1700–1734} & Raw Data & Adjusted \\
\hline
0–1 & 91.9 & 183.8 \\
1–4 & 85.5 & 100.9 \\
5–9 & 58.5 & 74.4 \\
10–14 & 75.2\textsuperscript{b} & 107.7 \\
1–14 & 128.1 & 145.2 \\
\hline
\textbf{FEMALES BORN 1700–1734} & Raw Data & Adjusted \\
\hline
0–1 & 90.4 & 180.8 \\
1–4 & 100.0 & 157.5 \\
5–9 & 59.9 & 80.2 \\
10–14 & 130.0\textsuperscript{b} & 229.7 \\
1–14 & 133.8 & 151.1 \\
\hline
\end{tabular}
\end{table}

\textsuperscript{a} The correction made for the underregistration of infant deaths follows a method suggested by Louis Henry in his \textit{Manuel de démographie historique}, (Paris, 1970), 22–25.

\textsuperscript{b} Because an unusually large number of persons dropped from observation during these years, the death rate is highly inflated for this age group.

source: Charles Parish Registers, Virginia State Library, Richmond.

Of course, those children who survived their early contacts with malaria and other childhood diseases could expect increasingly improved chances to live to maturity. This is reflected in the declining death rates for children 5 to 9 and 10 to 14 in the seventeenth century. (The eighteenth-century figures for children 10 to 14 are much too high due to emigration from the parish). Overall childhood mortality (1 to 14) in Charles Parish reached about

\cite{22} Rutman and Rutman, “Malaria in the Early Chesapeake,” 38; Peter H. Wood, \textit{Black Majority: Negroes in Colonial South Carolina from 1670 through the Stono Rebellion} (New York, 1974), 90.
145.4 per 1000 for males and 198 for females in the seventeenth century. In contrast, childhood mortality rates for colonial New England communities did not exceed 95 per 1000.\textsuperscript{23}

Movement out of the parish was likely to be greatest among children aged 15 to 19, so it is difficult to determine mortality rates for this age group or for all the childhood years, 0 to 19. But if one assumes that the mortality rate was the same between 15 to 19 as between 10 to 14, then of all the children born between 1665 and 1699, 315 per 1000 died before reaching the age of 20. Thus, mortality before adulthood remained about as high in the early decades of the eighteenth century: of those children born between 1700 and 1734, 311 per 1000 did not survive to their twentieth birthday. That these figures represent a relatively high level of mortality is evident from a comparison with rates calculated for a New England community. In Andover, Massachusetts, the chances of surviving to maturity were substantially greater than in Charles Parish—and until well into the eighteenth century. Of children born in Andover between 1670 and 1699, 170 per 1000 died before reaching age twenty. Indeed only during the years 1730–1759 when the throat distemper was raging in Andover did mortality rates begin to equal those that had prevailed in Charles Parish since the latter third of the seventeenth century.\textsuperscript{24}

Even those who reached maturity in Charles Parish could not expect to live long lives. Evidence on the age at death exists for


\textsuperscript{24} Greven, \textit{Four Generations}, 189–190. Little is known as yet about infant and childhood mortality elsewhere in the Chesapeake. In a recent study of mortality in seventeenth-century Charles County, Maryland, however, Walsh and Menard have used model life table estimates to suggest that a large proportion of children (46.6\%) failed to reach maturity. They developed their "preferred estimate" on the assumption that 80\% of infant deaths were unrecorded because of late birth registration—estimated as an average delay of six months from birth. The Charles Parish materials allow us to modify these estimates. Beginning around 1715 both the date of birth and baptism are often given in the registers, permitting the calculation of an estimated mean interval between birth and registration. What one finds for Charles Parish is an average time lag of four weeks between birth and registration (64.1\% of the births were recorded within four weeks and 97.0\% within eight weeks). If the pattern of birth registration in Charles Parish were common for most of the early Chesapeake, then it appears that Walsh and Menard may have exaggerated the extent of underregistration of infant deaths (by overestimating the delay in registration); consequently, their estimates of infant and childhood mortality are probably too high. See Walsh and Menard, "Death in the Chesapeake," 221–222.
only 118 out of 240 males who were born between 1665 and 1699 and who are known to have survived to at least age twenty. These incomplete data suggest that men were struck down by death long before they had reached an advanced age.

Table 3, Column A shows the life expectancy for those men whose age at death is known. The average lifespan for the 118 men who survived to at least age 20 was only 39.9 years. But since these men comprise only about one half of all males known to have reached adulthood in Charles Parish, it is possible that their mean life expectancy is unrepresentative of most men in the parish. Thus, for a more comprehensive view of adult male mortality, it is necessary to account for the experience of the remaining 122 men for whom the age at death is not known.

Using a method developed by Walsh and Menard, I have followed the careers of these 122 men in all the available county rec-

<table>
<thead>
<tr>
<th>ACHIEVED AGE</th>
<th>EMPIRICAL TABLE</th>
<th>HIGH MORTALITY</th>
<th>LOW MORTALITY</th>
<th>PREFERRED ESTIMATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>19.9</td>
<td>16.7</td>
<td>23.2</td>
<td>20.8</td>
</tr>
<tr>
<td>25</td>
<td>17.9</td>
<td>14.5</td>
<td>20.7</td>
<td>18.2</td>
</tr>
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<td>30</td>
<td>16.3</td>
<td>13.3</td>
<td>17.6</td>
<td>16.4</td>
</tr>
<tr>
<td>35</td>
<td>14.3</td>
<td>11.6</td>
<td>15.8</td>
<td>14.4</td>
</tr>
<tr>
<td>40</td>
<td>11.7</td>
<td>10.9</td>
<td>13.7</td>
<td>11.8</td>
</tr>
<tr>
<td>45</td>
<td>10.9</td>
<td>10.5</td>
<td>12.2</td>
<td>11.3</td>
</tr>
<tr>
<td>50</td>
<td>9.5</td>
<td>9.2</td>
<td>11.5</td>
<td>9.5</td>
</tr>
<tr>
<td>55</td>
<td>8.3</td>
<td>8.0</td>
<td>10.6</td>
<td>8.5</td>
</tr>
<tr>
<td>60</td>
<td>6.8</td>
<td>6.6</td>
<td>7.3</td>
<td>6.9</td>
</tr>
<tr>
<td>65</td>
<td>5.1</td>
<td>4.7</td>
<td>6.0</td>
<td>5.2</td>
</tr>
<tr>
<td>70</td>
<td>4.0</td>
<td>3.8</td>
<td>4.9</td>
<td>4.0</td>
</tr>
</tbody>
</table>

a includes only those whose age at death is known.
b assumes that unknowns died the day after their last appearance in the records. Unknowns participate only through the age at which they last appear.
c assumes that unknowns lived for ten years after their last appearance and then followed the rate of knowns. Unknowns participate only through the age at which they last appear.
d assumes that unknowns lived until the last day of appearance and then followed the rate of knowns.

This life table was constructed according to a method suggested by Lorena S. Walsh and Russell R. Menard, "Death in the Chesapeake: Two Life Tables for Men in Early Colonial Maryland," *Maryland Historical Magazine* LXIX (1974), 212–213. SOURCE: The prosopography described in the text.
ords and have determined the date at which they could last be proven to have been alive in York County. Since the mean age at which men first appeared in the records was 20.4, age 20 was designated the age when the unknowns were first placed at risk. A high mortality estimate was then constructed on the assumption that these men died the day after their last appearance in the records (Table 3, Column B). A low mortality estimate was also established which assumes that all unknowns lived an additional ten years beyond their last appearance in the records and then followed the mortality experience of those whose age at death is known (Column C). These two extremes in mortality experience are sufficiently broad so that the actual life expectancy of the unknowns fell within this range. Finally, a preferred estimate that assumes that the unknowns lived until the last day of their appearance in the records and then shared the mortality rate of the knowns represents the closest estimate of life expectancy between the high and low assumptions (Column D).

The high mortality assumption is too extreme because it indicates that men upon reaching age 20 could expect to live only another 16.7 years, well below the short life expectancy already established for the men whose age at death is known. Undoubtedly the unusually low figures produced in the high mortality estimate is a reflection of the difficulty in tracing the careers of Charles Parish men in a county that contained several parishes. The low mortality estimate is somewhat more reliable for it corrects for the problem of overrepresentation of youthful deaths in the records. But because few of the unknowns could be followed beyond age forty-five, the life expectancies shown in this estimate are probably more reliable in the earlier years. The preferred estimate that a man could expect to live an additional 20.8 years beyond age 20 is in all likelihood the most accurate estimate of life expectancy in the parish.

Life expectancies were low in other parts of the early Chesapeake, but clearly not as low as in Charles Parish. Life tables constructed by the Rutmans for Middlesex County, Virginia, and

25 Ibid., 212-213. For a general guide to the construction of life tables, see Barclay, Techniques of Population Analysis, 93-122.
26 Unless it can be shown that mortality was higher among migrants than “stable” members of a community, it seems likely that fewer young deaths than old ones were omitted from parish registers. See Wrigley, “Mortality in Pre-Industrial England,” 562.
by Walsh and Menard for Charles County, Maryland, show that in these areas the expectation of life for men was several years beyond that for Charles Parish men. The Rutmans have determined that men who achieved age 20 in Middlesex could expect to live another 28.8 years, while Walsh and Menard have found that native men of Charles County who survived to maturity lived an additional 26 years (Table 4). Apparently, living in Charles Parish meant that most men would die from five to eight years earlier than their contemporaries elsewhere in the Chesapeake.

However uncertain existence was for men in the early Chesapeake, it was not as harsh as that endured by Englishmen in the West Indies. Planters who came to these tropical islands, according to Dunn, "died young, married irregularly, and had too few children to maintain the population." Most men remained single and those who did marry normally died before they could produce large families. The 1715 Barbados census depicts a strikingly youthful society, not unlike Charles Parish, in which only 16 percent of the inhabitants had passed age forty and only 3 percent were over sixty.

The similar experience of colonists in Virginia, Maryland, and the West Indies demonstrates that Englishmen suffered in the hot climate of these islands and the Chesapeake. Contributing to the unhealthy living conditions in Charles Parish were the low, marshy soil, mosquitoes, hot temperatures, and poor drinking water. All of these together could have produced dangerous outbreaks of malaria which seem to have been responsible for much of the heavy mortality in the colonial Chesapeake. However susceptible to disease men were upon their arrival in the parish, conditions such as these increased the likelihood that they would not survive long.

27 Rutman and Rutman, "Malaria in the Early Chesapeake," 48; Walsh and Menard, "Death in the Chesapeake," 213. Due to the severities of the "seasoning" process, immigrants fell victim to disease more easily than natives and consequently had a shorter life expectancy of 22.7 years beyond age 20. Walsh and Menard, ibid., 218, 224.
28 Dunn, Sugar and Slaves, 325, 332.
29 L. G. Tyler, Narratives of Early Virginia (New York, 1907), 21–22, 36–37, 127, 210–211, 220, 423–424; John Duffy, Epidemics in Colonial America (Baton Rouge, 1953), 214–222; Rutman and Rutman, "Malaria in the Early Chesapeake," 36, 38. See also the following letters from Virginia governors in the 1680s complaining about their debilitating health in the harsh Virginia summers and winters: Lord Effingham to King, 1687, McDonald Papers, VII, 262–263; Lord Jenkins to Lord Howard, 1683, McDonald Papers, VI, 272. Virginia State Library, Richmond.
Demographic research into colonial New England communities suggests a rather different mortality experience. In seventeenth-century Plymouth and Andover, Massachusetts, survival to maturity gave men the prospect of living an additional forty-four to forty-eight years—more than twice the average life expectancy of adult males in Charles Parish 30 (see Table 4). Although too little is known about life expectancy throughout early America, it does appear that mortality may have decreased as one travelled northward along the Anglo-American coast from the West Indies to New England in the seventeenth century.

Table 4  Life Expectancy for Men in the Seventeenth-Century Chesapeake and New England

<table>
<thead>
<tr>
<th>ACHIEVED AGE</th>
<th>CHARLES PARISH</th>
<th>MARYLAND IMMIGRANTS</th>
<th>MARYLAND NATIVES</th>
<th>MIDDLESEX COUNTY</th>
<th>PLYMOUTH COLONY</th>
<th>ANDOVER, MASS.</th>
<th>SALEM, MASS.</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>20.8</td>
<td>22.7a</td>
<td>26.0</td>
<td>28.8</td>
<td>48.2b</td>
<td>44.6</td>
<td>36.1</td>
</tr>
<tr>
<td>30</td>
<td>16.4</td>
<td>17.4</td>
<td>20.4</td>
<td>19.4</td>
<td>40.0</td>
<td>39.3</td>
<td>29.2</td>
</tr>
<tr>
<td>40</td>
<td>11.8</td>
<td>13.2</td>
<td>15.6</td>
<td>13.0</td>
<td>31.2</td>
<td>31.8</td>
<td>24.1</td>
</tr>
<tr>
<td>50</td>
<td>9.5</td>
<td>10.3</td>
<td>12.0</td>
<td>7.7</td>
<td>23.7</td>
<td>23.5</td>
<td>19.1</td>
</tr>
<tr>
<td>60</td>
<td>6.9</td>
<td>10.0</td>
<td>9.3</td>
<td>5.8</td>
<td>16.3</td>
<td>15.6</td>
<td>14.5</td>
</tr>
<tr>
<td>70</td>
<td>4.0</td>
<td>5.5</td>
<td>7.0</td>
<td>3.6</td>
<td>9.9</td>
<td>10.3</td>
<td>10.0</td>
</tr>
</tbody>
</table>

* a age 22  
  b age 21


An important consequence of the shortened lifespans of men born in Charles Parish was the distinct shape it gave to their families. With the death of many fathers long before they reached the end of their normal procreative years, the size of most families remained small. The reconstitution of 386 families of men born between 1660 and 1689 discloses that the mean number of children known to have been born to all of the wives of these Charles

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30 Demos, *A Little Commonwealth*, 192; Greven, *Four Generations*, 27, 108. Salem is an exception to the New England communities studied thus far. Still, Salem’s estimated life expectancy for men of thirty-six years from age twenty suggests that it was considerably healthier than Charles Parish. For the Salem figures, see Vinovskis, “Mortality Rates and Trends in Massachusetts,” 198-199.
Parish men was 2.99. Almost two-thirds of these families (63.9 percent) contained fewer than four children and nine out of ten (90.3 percent) had under seven children. A large family of ten or more children was clearly a rare occurrence for only four families of that size (about 1 percent) could be found during this period (Table 5). Families remained small among fathers born between

Table 5  Size of Families of Fathers Born Before 1689

<table>
<thead>
<tr>
<th>NUMBER OF CHILDREN</th>
<th>NUMBER OF FAMILIES</th>
<th>PERCENTAGE OF FAMILIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-3</td>
<td>247</td>
<td>64.0</td>
</tr>
<tr>
<td>4-6</td>
<td>102</td>
<td>26.4</td>
</tr>
<tr>
<td>7-9</td>
<td>33</td>
<td>8.8</td>
</tr>
<tr>
<td>10 or more</td>
<td>4</td>
<td>1.0</td>
</tr>
<tr>
<td>mean</td>
<td>2.99</td>
<td></td>
</tr>
<tr>
<td>median</td>
<td>3.00</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>386</td>
<td>100.2</td>
</tr>
</tbody>
</table>

1690 and 1719. The mean number of children per family rose to 3.65, but the proportion of large families—those with seven or more children—stayed at about 10 percent. Again, families of one to three children predominated as 59.5 percent were of this size (Table 6).

Table 6  Size of Families of Fathers Born Between 1690 and 1719

<table>
<thead>
<tr>
<th>NUMBER OF CHILDREN</th>
<th>NUMBER OF FAMILIES</th>
<th>PERCENTAGE OF FAMILIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-3</td>
<td>147</td>
<td>59.5</td>
</tr>
<tr>
<td>4-6</td>
<td>75</td>
<td>30.3</td>
</tr>
<tr>
<td>7-9</td>
<td>20</td>
<td>8.1</td>
</tr>
<tr>
<td>10 or more</td>
<td>5</td>
<td>2.0</td>
</tr>
<tr>
<td>mean</td>
<td>3.65</td>
<td></td>
</tr>
<tr>
<td>median</td>
<td>3.00</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>247</td>
<td>99.9</td>
</tr>
</tbody>
</table>

Reconstituting families from the parish register can create difficulties, however. A married couple may have begun raising a family elsewhere before entering Charles Parish or may have had additional children after leaving the parish. In either case the size of the family as reflected in the parish register would be underestimated. Thus, a separate study was made of only those families headed by men who were known to have spent their entire lives in the parish. Limited in this way, only seventy-seven families can be analyzed in Charles Parish from 1660 to 1719. Even this restricted sample confirms our initial observation that families grew to only modest proportions. Indeed, the mean number of children born to all the wives of these 77 men was 3.6, roughly the same as that determined for all families reconstituted from the register from 1690 to 1719. Although about a third of these families contained from four to six children, only four could have been considered large, with seven or more children31 (Table 7).

Table 7 Size of Families of Fathers Born Between 1660 and 1719 and Who Remained in Charles Parish Until Death

<table>
<thead>
<tr>
<th>NUMBER OF CHILDREN</th>
<th>NUMBER OF FAMILIES</th>
<th>PERCENTAGE OF FAMILIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-3</td>
<td>42</td>
<td>54.5</td>
</tr>
<tr>
<td>4-6</td>
<td>29</td>
<td>37.6</td>
</tr>
<tr>
<td>7-9</td>
<td>6</td>
<td>7.7</td>
</tr>
<tr>
<td>10 or more</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>mean</td>
<td>3.60</td>
<td></td>
</tr>
<tr>
<td>median</td>
<td>3.00</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>77</td>
<td>99.8</td>
</tr>
</tbody>
</table>

SOURCE: The prosopography described in the text.

31 This analysis of family size was based on the procreative years of the husband rather than the wife because of the lack of marriage records, which prevents tracing the lives of women after marriage. Since the reproductive years of men are not usually limited as are those of women, the average family size indicated here may be somewhat larger than what would be found if the wife’s period of fertility were used. Moreover, if men outlived women in Charles Parish to the same extent as they did in Middlesex County, the period of female fertility would have been considerably shortened. Rutman and Rutman, “Malaria in the Early Chesapeake.” Child-bearing came somewhat less frequently to Charles Parish women than to women in other parts of early America, with a mean birth interval of thirty-two months. Other studies indicate that children were usually spaced from twenty-four to thirty months apart in seventeenth- and eighteenth-century America and Europe. See Wells, “Quaker Marriage Patterns,” 440-441.
Parental death frequently led to remarriage and the merging of two families could sometimes produce rather large households. Unfortunately, the absence of marriage records for Charles Parish precludes a study of remarriage. An illustration from the parish register, however, suggests how the development of large households from remarriage may have been a limited phenomenon. Buford Pleasants had one son, John, by his first wife, Mary. After her death in 1725, he quickly remarried and within two years he and his new wife Elizabeth, had the first of seven children. Infant and childhood deaths, however, reduced what might have developed into a large family of eight children. Indeed, by 1743 when Buford and Elizabeth’s last child, George, was born, only four children were still alive. Thus, while second and third marriages on occasion forged large and complex households for a few years, the constant presence of death made swift and frequent changes in these families and limited most of them to three or four children.

Clearly, the controlling demographic fact in colonial Charles Parish families was the early death of parents rather than of children. With a rather high proportion of persons dying in childhood and adolescence, even those few parents who survived to an advanced age could expect to nourish only one or two children to maturity. Grandparentage was an even rarer phenomenon. From 1660 to 1760 only a dozen men in Charles Parish are known to have lived to see their grandchildren. In light of this, Murrin’s recent suggestion that grandparents were a New England “invention” takes on added plausibility.32

For children early parental loss had an important and immediate consequence: orphanhood. According to the Rutmans, orphanhood was part of the fabric of life in seventeenth-century Middlesex County, where about three-fourths (73.2 percent) of all children had lost at least one parent before reaching twenty-one or the age of marriage. Over a third (36.0 percent) were fully orphaned at maturity. The same phenomenon can be followed in the Charles Parish materials throughout the colonial era.33 During the

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32 See John Murrin’s review essay of several of the demographic and community studies of colonial New England in History and Theory, XI (1972), 238.
33 Orphanhood appears slightly more prevalent in the Middlesex figures probably because the data analyzed by the Rutmans focus mainly on the seventeenth century, when mortality and early parental loss were particularly high.
years 1660 to 1740, fully two-thirds (66.8 percent) of the parish children for whom evidence exists on the death of their parents were at least half-orphaned, while one of seven (13.9 percent) became full orphans\textsuperscript{34} (Table 8).

<table>
<thead>
<tr>
<th></th>
<th>CHILDREN KNOWN TO SURVIVE TO MATURITY\textsuperscript{a}</th>
<th>CHILDREN WITH BOTH PARENTS AT MATURITY</th>
<th>CHILDREN WITH ONLY ONE PARENT AT MATURITY</th>
<th>CHILDREN ORPHANED AT MATURITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middlesex Co.</td>
<td>164 (100%)</td>
<td>44 (26.8%)</td>
<td>61 (37.2%)</td>
<td>59 (36.0%)</td>
</tr>
<tr>
<td>Charles Parish</td>
<td>459 (100%)</td>
<td>152 (33.1%)</td>
<td>243 (52.9%)</td>
<td>64 (13.9%)</td>
</tr>
</tbody>
</table>

\textsuperscript{a} For the Middlesex data, maturity means age twenty-one or the age of marriage, whichever came first; for Charles Parish maturity is defined as twenty-one for men and eighteen for women.


\textbf{Table 8} Orphanhood in Early Virginia

Early parental death not only created an abundance of orphans in colonial Virginia, but also permitted young men to gain their autonomy rather early in life. This is reflected in the relatively young age at which sons married. Although marriage records are not available for Charles Parish, a crude estimate of the average age at marriage for men can be made by establishing a man’s age at the birth of his first child. Given this figure, calculated from the birth register, one can subtract the average interval between marriage and first birth. In a recent paper Menard has suggested that despite a fairly high incidence of bridal pregnancy, most Chesapeake couples experienced an interval of fifteen months between marriage and first birth. Using this fifteen-month interval as a guide for Charles Parish families, one can arrive at an estimated maximum age at marriage for men.\textsuperscript{35}

\textsuperscript{34} Data concerning the date of death for at least one parent were available for 164 families. Of these 164 known deaths, 109, or 66.4%, were fathers, 16, or 9.9% were mothers, and in 39 cases, or 23.7%, the deaths of both parents were known. That so few deaths of mothers are known is simply a reflection of the difficulty in following the lives of women in the records without the benefit of a marriage register.

\textsuperscript{35} Russell R. Menard, "The Demography of Somerset County, Maryland: A Preliminary Report," paper presented to the Stony Brook Conference on Social History (1975). Since the first marriage for a man might have ended in the death of his wife before having a child,
Evidence for sixty-one men born between 1660 and 1699 reveals that they had reached an average age of 25.9 years at the birth of their first child. Although this would suggest that the mean age at marriage was around 24.7, a significant number of men married and started families much earlier than this. In fact, about one half (50.8 percent) had their first child before they were twenty-five, and about one third (34.4 percent) between the ages twenty-one and twenty-four (Table 9).

**Table 9  Age at Birth of First Child of Fathers Born Between 1660 and 1699**

<table>
<thead>
<tr>
<th>AGE</th>
<th>N</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>under 21</td>
<td>10</td>
<td>16.4</td>
</tr>
<tr>
<td>21–24</td>
<td>21</td>
<td>34.4</td>
</tr>
<tr>
<td>25–28</td>
<td>18</td>
<td>29.6</td>
</tr>
<tr>
<td>29 or more</td>
<td>12</td>
<td>19.6</td>
</tr>
<tr>
<td>mean</td>
<td>25.9</td>
<td></td>
</tr>
<tr>
<td>median</td>
<td>24.0</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>61</td>
<td>100.0</td>
</tr>
</tbody>
</table>

During the subsequent forty years, 1700–1739, young men in the parish had even earlier opportunities for marriage and independence. Fewer data are available for these years, but the experience of thirty-seven men discloses that they began raising families at a mean age of 24.8 years, indicating 23.6 as a maximum age at marriage. Twenty-two of the men, or 59.5 percent, had become parents by age 24, or about 22.8 when married, which was almost a 10 percent increase over the previous four decades (Table 10).

Despite the awkward nature of these calculations, it is apparent that from 1660 to 1740 the mean age at marriage was not higher than 24.8 years. For the first four decades of the

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36 A demographic study of 100 gentry families in eighteenth-century Virginia corroborates this pattern of relatively early age at marriage, at least among the elite. From 1695 to 1750 the mean age at marriage for men was 24.4, while for women it was 20.3 years. Karen Dawley, “Childhood in Eighteenth-Century Virginia,” unpub. master’s thesis (University of Virginia, 1973).
Table 10  Age at Birth of First Child of Fathers Born Between 1700 and 1739

<table>
<thead>
<tr>
<th>AGE</th>
<th>N</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>under 21</td>
<td>5</td>
<td>13.8</td>
</tr>
<tr>
<td>21-24</td>
<td>17</td>
<td>45.8</td>
</tr>
<tr>
<td>25-28</td>
<td>8</td>
<td>21.6</td>
</tr>
<tr>
<td>29 or more</td>
<td>7</td>
<td>18.9</td>
</tr>
<tr>
<td>mean</td>
<td>24.8</td>
<td></td>
</tr>
<tr>
<td>median</td>
<td>24.0</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>37</td>
<td>100.1</td>
</tr>
</tbody>
</table>

Source: Charles Parish Registers, Virginia State Library, Richmond.

eighteenth century, in fact, it declined to a maximum of 23.6 years. Particularly persuasive of the early age at marriage in Charles Parish is the fact that over half (54.1 percent) married before age 24. Similar marriage patterns have been discovered in portions of early Maryland. By comparison, men in colonial Andover waited until they were 26 or 27 before marriage, as did Quaker men in the early eighteenth century. Indeed, no region studied thus far in colonial America exhibited such early marital patterns for men as the Chesapeake.37

In a farming community such as Charles Parish, the ability to marry and to establish an independent household depended largely on gaining an inheritance of land or slaves. The age, then, at which a man could marry and become self-sufficient was often closely linked to the timing of his father’s death. Young men in Charles Parish found conditions favorable for their early independence. Since most fathers died in their early forties, sons usually received their inheritance before they had come of age at twenty-one. Moreover, with the normally small number of surviving sons in each family, parents could bequeath rather generous portions of their land and personal property. Consequently, many young men

37 Menard, “Demography of Somerset County”; Greven, Four Generations, 33, 35, 118, 120, 206, 208; Wells, “Quaker Marriage Patterns,” 417. The one region outside the Chesapeake where men may have married as early as they did in Charles Parish was Bristol, R.I., where the mean age at marriage for men before 1750 was 23.9. See Demos, “Families in Colonial Bristol,” 55.
had the wherewithal quite early in life to consider marriage and assert their economic independence.\textsuperscript{38}

The link suggested here between early parental loss and early marriage can be tested by comparing the marital patterns of children who were orphaned before reaching the mean age at marriage with those whose fathers were still alive. As Table 11 shows, the

<table>
<thead>
<tr>
<th>Table 11</th>
<th>Impact of Early Parental Loss on Age at First Marriage for Males in Charles Parish</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MEAN AGE OF MEN AT BIRTH OF 1ST CHILD</td>
</tr>
<tr>
<td>Father living when child reached estimated mean age at marriage</td>
<td>27.2 ($N = 27$)</td>
</tr>
<tr>
<td>Father dead when child reached estimated mean age at marriage</td>
<td>23.5 ($N = 33$)</td>
</tr>
</tbody>
</table>

Source: Charles Parish Registers, Virginia State Library, Richmond.

timing of a father’s death appears to have made an important difference in marriage opportunities, for orphaned children married almost four years earlier than children whose fathers were still alive. Admittedly, the evidence on this question is too small to be conclusive, but the data presented do suggest the strong impact of early parental loss on the marital autonomy of children.

The incomplete and often intractable vital records for Charles Parish have of necessity made this a tentative study of family structure and mortality. More work remains to be done on family life and demographic patterns in other areas of the early Chesapeake before solid conclusions can be drawn. Nevertheless, it may be

worthwhile to consider some possible implications from what is known about this parish.

If the demographic experience of Charles Parish were at all common, then the population of colonial Virginia had a distinctive age structure. Indeed, the oppressive mortality that prevailed in the seventeenth century virtually eliminated the development of an elderly generation, which may have had important consequences for the character of family life and authority in the parish. In the absence of grandparents, uncles, aunts, and family friends became especially important in caring for a rather large population of orphans. Many children, then, encountered more than one set of parents while growing up in Charles Parish. As a result, any tendency toward strong patriarchal authority was probably diverted amid these often abrupt changes in the life of the family.

Moreover, without a watchful, paternalistic group of elders in the parish, the forces for authority and tradition may have been considerably weaker than in New England towns where the influence of old Puritan patriarchs was keenly felt. That Virginia experienced more social and political unrest in the seventeenth century than the New England colonies may in part have been a reflection of the extremely youthful composition of the adult population.39 “Conversation across the generations,” as Laslett has put it, was rare in early Virginia, allowing men of roughly the same age to scramble for power and wealth relatively unimpeded by a sense of tradition or a concern for the opinion of an elderly generation.40

Above all, the demographic experience of Charles Parish indicates that little had changed in terms of family structure and longevity for Englishmen arriving in Virginia after the middle of the seventeenth century. Conditions in the colony could be rather harsh, but most English villages also suffered the effects of high mortality. Men in both England and Virginia lived relatively short lives (to about age fifty in England to age forty-five in Virginia) and very few survived to boast about grandchildren. English and

Virginia families were small, usually no more than a husband and wife and two or three children. In seventeenth-century Virginia, however, land was more abundant and the early deaths of fathers reduced paternal authority in the family and cleared the way for their sons’ early autonomy, several years before young men in England could expect an independent life.41

Kinship and Migration: The Making of an Oregon Isolate Community

The term "isolate" is one that is familiar to the human geneticist, but not necessarily to the historian. When used in genetics the term refers to a small, closed population in which the gene frequencies are different from those found in the general population. Human geneticists have so far developed three general categories of isolates: (1) physical—in the sense of geographical—isolates, (2) ideological isolates, and (3) linguistic isolates.¹

Geographical location has, for example, created an isolate population on the island of Tristan de Cunha. A remote continental location can cause similar isolation, as in the case of Lac St. Jean-Chicoutimi in Quebec Province. Ideological convictions have produced isolates even in circumstances where people exist in easy physical proximity to other social groups. The Old Order Amish are a good example of this phenomenon. Linguistic characteristics have contributed to the isolation of some of the Indian and Eskimo populations in Alaska, who, although they live in similar locations and circumstances, tend not to inter-marry.²

What is particularly significant about the isolate described here is, first, that it exists without primarily conforming to any of

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¹ A clear definition of the term "isolate" is given in L. L. Cavalli-Sforza and W. F. Bodmer, The Genetics of Human Populations (San Francisco, 1971), 352: "relatively small populations that have little or no exchange with other populations." Translated into vocabulary more familiar to historians an isolate is a small community whose members have little to do with those outside their group, and who above all do not marry persons outside their group. See also ibid., 353, Table 7.4.

the patterns cited above, and, second, that its identification was made possible through the application of the techniques of historical demography. The linkage of sociohistorical findings with biomedical data has made it possible both to perceive the existence of the isolate and to investigate its dynamics.

Attention was first drawn to the community when a young man of Dutch extraction was referred by his physician to the University of Oregon Medical School for consultation concerning blistering of the skin upon exposure to sunshine. The condition had afflicted the patient since childhood. His skin would redden easily when exposed to the sun. Small blisters would appear, become confluent, and burst, releasing clear fluid. The affected areas of skin would then heal, but as the process was repeated through further exposure to sun, areas of fissured, thickened skin marred by deposits of deeper colored pigment were left. The involved areas of skin were over the face, neck, forearms, and hands. The process could be prevented by the patient’s staying in the shade or indoors or by his wearing a broad-brimmed hat and gloves. However, prevention became difficult when he began working as a heavy-equipment operator on road construction. He was seen at the Medical School and the diagnosis of sun-sensitive porphyria was made. He was given a variety of sun-screen ointments, which were partially successful in decreasing, but not eliminating his tendency to redden and blister. Because sun-sensitive porphyria is genetical, the patient was referred to the Genetics Clinic for family studies. These disclosed only two other definite cases of sun-sensitive porphyria in his family. Those involved a brother and a male cousin. Parents and other close relatives proved normal on their medical history, clinical examination, and laboratory study. Relief of symptoms was achieved by periodic removal of blood in order to decrease the red cell mass from which the skin-damaging porphyrin compounds emanate.

The porphyrias are a group of hereditary biochemical disorders involving aberrations in the metabolism of porphyrin compounds which occur with the breakdown of the heme component

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3 The case report is furnished by Drs. Lovrien and Frederick Hecht.
4 The medical practice of blood-letting is a venerable technique which is now regarded as not only outmoded, but harmful. This is one instance where purposeful bleeding of a patient—physiologically—is therapeutic.
of hemoglobin. Despite certain similarities, the various types of porphyria can be distinguished from one another by clinical, biochemical, and/or genetical criteria. Two types of porphyria have been of particular sociohistorical interest: one form, acute intermittent porphyria, involves excruciating attacks of abdominal pain and episodes of aberrant behavior. George III's madness is now thought to have been caused by acute intermittent porphyria since this disease has been diagnosed in some of his living descendants. A second form of the disease, known as porphyria variegata, is also of historic interest. An extensive genealogical study of Dutch settlers in South Africa has been done by Deane, a physician. Finding that a number of his patients had porphyria, Deane proceeded to trace the disease. He identified 1,021 affected persons, all of whom were descended from a pair of seventeenth-century migrants. Which of the two, the husband or wife, had brought the disorder to South Africa is not known, but that the gene had been carried down from one of the two original progenitors is clear. Deane's is a classic study of founder effect.

The genetic mode of transmission for both of these types of porphyria is known to be autosomal dominant. "Autosomal" means that the responsible genes are carried on one of the twenty-two non-sex chromosomes (autosomes). Dominant (and fully penetrant) means that symptoms will be manifest if one porphyria gene is received by a particular offspring. (In the case of autosomal recessive disorders, both parents must carry a gene and it must be present in a double dose in the child for symptoms to be apparent.)

The type of cutaneous porphyria identified in Oregon is clinically reminiscent of the South African variety of porphyria variegata. Insofar as the Oregon patient is of Dutch descent, it seemed initially that the direction of the Oregon study would parallel that of Deane in South Africa. However, careful investigation un-

earthed only two other cases of porphyria among the Oregon patient’s numerous relatives. This finding was incompatible with the dominant (fully penetrant) pattern of inheritance described in South Africa. In turn this led the study in a new direction.

The Oregon porphyria might have a recessive pattern. Insofar as the affected persons were biological relatives, the next logical step was to search for possible consanguinity. That line of inquiry, however, also broke down, for, if there were consanguinity, it was not obvious. What did become clear was that the patient and his relatives belonged to a very large family with a clear sense of its ethnic identity, complex bonds of kinship with other persons of Dutch extraction, and a strong commitment to the Roman Catholic religion. The focus of the inquiry now turned to the possibility of the patient and his relatives being members of an isolate.

To raise the possibility that the original patient might be a member of an isolate community invited a change in methodology, since the community bore none of the classic hallmarks of an isolate. Located in the Willamette Valley, a fertile trough stretching 100 miles from the Columbia River to the city of Eugene, in close proximity to the state’s major metropolis, the community can hardly be categorized as geographically isolated. Nor could ethnocentricity be the basis of isolation for the community encompassed persons of Belgian, German, and even Irish descent, in addition to the Dutch presumed to be so omnipresent.

Ideology offered, if anything, an even less satisfactory explanation for isolation. If religion can be defined as a form of ideology, the most one can say in this instance is that this community has a strong sense of religious identity. Most members of the community are devout, practicing Catholics, and as we shall see shortly, it is the church which serves as the physical and spiritual center of the “town.” However, a distinction must be made between a strong sense of religious identity and isolation.

The importance of the group’s Catholic identity is indisputable. That this identity is the basis of isolation is improbable. In the first place the earliest sedentary European population in the Willamette Valley was Catholic. In the 1820s and 1830s French-Canadian fur traders began forming a small community in an area known as French Prairie. By 1840 the community was sufficiently numerous for a mission to be established under Father Francis Norbet Blanchet. By 1845 Oregon became an ecclesiastical prov-
ince with Blanchet as its archbishop. When the community under study began to take shape in the Willamette Valley in 1875 its Catholic character was hardly an anomaly in a state which had a well-established Catholic hierarchy, schools, and mission system. One would have expected the Catholic character of the group to have assisted in the integration of the new arrivals into the already established population of the area.

Since the community could not be assumed to be an isolate it became necessary to ask explicitly whether or not it was indeed an isolate. It was this need which led to a medicohistorical collaboration. From the medical point of view the usual manner of building a pedigree had proven insufficient to the task. It was clear that the basis for the medical problems observed would not be found within the lines of a single family. A broader study encompassing the total community was needed. Being Catholic, the community had kept careful registers of its marriages, baptisms, and burials. The historical technique of family reconstitution developed by Henry and later adapted to English registers by Wrigley held the promise of proferring an answer to the medical question. As we shall see, the exercise in family reconstitution, pursued from a historical as opposed to a medical point of view, led to the conclusion that the community was in fact an isolate. Further, the same analytical technique uncovered the basis for that isolation in the system of kinship generated by certain idiosyncracies of nuptial patterns.

The start for community reconstruction was provided by the parish registers. Following the general methodology outlined by Wrigley, we transcribed the baptism, marriage, and burial records, and then proceeded to rebuild all the families. It became im-

8 A word should be said here about research procedures. Data was collected for this project by a series of undergraduate seminar students. In three different years groups of ten to twelve students were organized into a research team. Each time the team concentrated its efforts on a particular set of records and wrote a report of its findings. Thus, the first group transcribed most of the parish register which is available on microfilm at the Oregon Historical Society. The second group completed the transcription and expanded the reconstitution process to include information from the Oregon Census of 1880 and land records. The third group utilized migration records and three Wisconsin ms. censuses for 1850, 1860, and 1870.
mediately apparent that the community had two components: those who appeared fleetingly in the parish registers, and those whose lives were traceable through multiple generations. The latter numbered approximately thirty family names, and those we refer to as the “core families.”

Other sources have filled in the skeletal history provided by the parish registers. We have used the Oregon manuscript census of 1880 and the Wisconsin manuscript censuses of 1840, 1850, 1860, and 1870. We have also had access to Dutch migration records, and to unofficial documentation, the most important piece being a history of the parish produced by members of the community at the time of its Diamond Jubilee in 1950. Combining the data from these various sources we now have the rough outlines of the community’s European history, a rudimentary understanding of the group’s first North American experiences in Wisconsin, definite data on the community’s establishment in Oregon, and considerable knowledge of its subsequent history in the Northwest.

The European roots of twenty-eight core families are traceable to Holland, Belgium, or Germany. In twenty-four cases we have definite national identifications. Three families were German, two Belgian, and nineteen Dutch. Of the nineteen Dutch, seventeen family names were traceable to specific towns, all of which were located in the eastern portion of the Province of North Brabant. Of four families for whom we lack definite identifications, family surnames suggest that one was Belgian, one German or Dutch, and two Dutch.

As the map indicates, the geographical origins of the core families were highly concentrated, most coming from a series of towns clustered around Uden. Three other Brabant families came from towns contiguous to the Belgian border. Insofar as that bor-

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9 It is difficult to give an absolute number for the core families, for they dominate in somewhat different ways. Some families are prominently core families if one examines birth records. Others dominate land-holding. Some are demographically unimportant in a numerical and/or economic sense, but are vital as genealogical links. Depending on the focus, we found ourselves concerned with twelve to thirty family names.

10 We have had Dutch migration records furnished us through the generosity of Robert Swieringa, who provided us with the computer lists of migrants from which he is doing a study of Dutch migration to North America in the nineteenth century. We have used his lists to locate the points of origin and migration dates of our core families. Diamond Jubilee, Visitation Church, Verboort, Oregon, 1875 to 1950. Private copy.
der had only been established in 1832, we have speculated that the kin connections between Dutch and Belgians might have preceded the imposition of the political boundary. By the same reasoning it is possible that the Germans were neighbors from across the Rhine. These possibilities are highly conjectural, but they do serve to make the point that the sharp differences one might presume to be reflected by differences in national designations may have been meaningless in the real-life world of these people.

By using the Dutch migration list we have been able to trace the arrival of the nineteen Dutch core families in North America. Their migration began in 1847 and 1848, at which time three Catholic priests sought to encourage people to move into the Fox River Valley of Wisconsin. They recruited a number of North Brabanders, including two future Oregon core families. Other Dutch families arrived in the Fox River Valley in subsequent years, among them the rest of our core families. The last Wisconsin arrival date for a core family was 1870.

In tracing the Wisconsin history of the group we had as our sole source the manuscript census records, and what we have learned leaves us with important questions unanswered. The core families were somewhat scattered along the Fox River Valley, although by 1870 there was a notable concentration of thirteen of thirty-five core-family households in De Pere. Overt signs of their maintaining a separate identity are thin. The core families in all cases had other Dutch households near them. Perhaps one hint that the future Oregonians already had a sense of separate identity is the fact that Kaukana, the site of the most numerous concentration of Dutch households along the Fox in 1850, 1860, and 1870 drew a maximum of three core households, while in De Pere, which had a modest number of Dutch households, half of these were to become Oregon core families.

It is not until we come to the northwestern history of the community that we begin to have a clear understanding of the group's dynamics. A sense of identity emerges from the carefully preserved memory of the founding of the Oregon "town." According to local tradition the community's history began in 1873 when a man named John Verboort travelled from Wisconsin to the Pacific Northwest in search of a suitable location for an agrarian,

Catholic community. Having seen the Willamette Valley, he decided that Washington County would be the ideal geographical site. A second scouting expedition corroborated Verboort’s report, and by the spring of 1875 six families made the move together to the Pacific Northwest.

The six families in question stopped initially in Portland while they arranged the cash purchase of the 550 acre Henry Black Donation Land claim. By the first week of April, 1875, the transaction had been completed, and the families took a train from Portland to the Valley station nearest their destination. For the first months the group functioned in a communal fashion, all twenty-seven in the party living together in the Black House, a ten-room, two-storied structure. A large garden was laid out the produce of which was shared. Even the picking of the wild blackberries which grow so prolifically in western Oregon was carefully supervised and the crop judiciously divided. After the first harvest the land was divided into lots and individual houses were built. The community grew as new families arrived from Wisconsin, and by 1908 community expansion was sufficient to warrant the founding of a daughter parish from among the families of the original parish.

This historical overview serves to emphasize several important points about the community. First, there is no doubt about the cohesiveness of the group during the time it has lived in the Northwest. Second, the group’s identity was already visible in Wisconsin. Third, circumstantial evidence suggests that community identity was already evident in Europe. Each of these observations reveals the common origins of the community and strengthens the possibility that it could be an isolate. However, we have yet to grasp the dynamics which bind the group together. These are the issues to which we must now turn.

Our concentrated analysis has so far centered on Oregon, and on the basis of what we have learned of the community’s northwestern history it appears that there are three phenomena which have served to keep the group united: religion, land, and kinship.

12 There is a strong possibility that family B-4 is related to family A. The husband’s widowed mother in family 4 bears the same married surname as the wife’s maiden name in family 2. We have found no way to prove the linkage but suspect strongly that some relationship did exist (see fig. 1).
We suspect that the community would give greatest credit to the first element, whereas we, since the focus of our interest is on the isolate character of the group, emphasize the last. All three components are, however, interwoven, and from stray clues we think that it has been these same elements which linked the group both in the Old World and the New.

RELIGION The importance of religion as a unifying principle is not to be belittled. Although the community may not exist insofar as the secular organization of the state of Oregon is concerned, the town being unincorporated, it definitely exists in the minds of the inhabitants. Nor is it hard for outsiders to find the community if they know for what to look. The physical center of the “town” is marked by its church, the importance of which is emphasized by its size and solidity. Although the houses of the area are generally modest in size and built of wood, the church stands out in terms of its imposing mass and its building material—red brick.

The centrality of the church, both in a physical and a spiritual sense, is nothing new to the community. According to tradition a major reason for the move from Wisconsin to Oregon was the desire to found a Catholic community. We also know that the guiding force behind the migration was Father William Verboort. His brothers, John and Albert, helped scout the Northwest for a suitable location, and fourteen members of Father Verboort’s family migrated with the twenty-seven person founding group. His unexpected death at the age of forty during the summer of 1876 was a severe blow. Yet the sense of purpose and cohesiveness did not evaporate. Additional families arrived and the church continued to serve as the focal point of the community.

By 1889 the community enjoyed sufficient material comfort and its membership was large enough for a major building program to be undertaken which continued through 1894. Entries in the parish register make clear the wide and active involvement of the parishioners. It was they who provided not only funds, but also labor to erect a house for the priest, paint the church, and place a cross in the cemetery.

However, when we speak of the church as the focal point of the community, this must be understood in a wider context. The church was not just a building, but rather the basic unit of social organization. It included the parochial school—initially limited to
elementary education, but later expanded to include a high school in response to changes in state educational requirements. The community orientation of the school was reinforced by teaching nuns who were frequently daughters of parish families.

Social and recreational activity also clustered around the church, and, to a degree, economic life as well. Beyond the school building stands the community hall. Once heavily used for dances and social gatherings, it is now the one “town” building which appears to be somewhat neglected. It is still used for the annual sausage festival, but the social gatherings of times past are no longer frequent.

The two centers of economic activity stand across the road from the church. These are the grocery store, today a very small enterprise catering exclusively to limited local needs, and the sausage factory. Despite its small size and necessarily limited clientele, the store is still neatly painted and carefully maintained. The sausage factory is a recent addition, and it stands in new splendor, the largest single structure in the town aside from the church. Commercial sausage-making has sprung from the annual sausage festival, a traditional Brabant celebration to which the public is invited.

LAND The strongest physical manifestation of community identity is to be seen in what has happened to landholding in the area. To have an accurate idea of the configuration of the community requires that one take in at least one neighboring parish which was formed early in the twentieth century by the overflow from the original community. The formation of the new parish probably reflects the expansion in landholding which placed the younger generation at an increasingly inconvenient distance from the original parish church. Data about landholding in the second parish have yet to be collected. Nonetheless, within the limits of what has been done, interesting patterns are discernible.

We selected nine sections which corresponded most closely to the boundaries of the original parish, and proceeded to analyze landholding patterns for the core families from 1875 to 1972. We made three observations. The first was that once land came into the possession of community members it tended to stay there. The second discovery was that, in terms of landholding in the nine sections examined, there were two stages of development. The first
stage was completed during the first decade of the twentieth century, while the second took place during World War II. Our third discovery was that within the community confines there was a lively exchange of land among members of the community.

The propensity for land to remain with members of the core families is illustrated in Table 1 which lists the total acreage held by the core families in 1907, 1937, and 1972. Total acreage remained stable from 1907 to 1937, then increased by 1972.

Close to 1,000 additional acres had been acquired in the nine sections, bringing the community holdings to 70 percent of the total land in the nine sections. The first generation had established a basic territorial claim, with minimal changes taking place during the second generation. Either late in the life of the second generation, or early in that of the third generation territorial organization did alter. The total land area held by the core families increased, possibly as a response to demographical expansion and perhaps as a response to the increased demand for agricultural products generated during World War II.

At the same time the community world was never static. Ownership changed frequently among members of the community and there was a distinct tendency for land near the church to be broken into smaller and smaller fragments. In fact, at the center of the community near the church, there is a proliferation of small parcels of land, a number of which are now utilized by the community’s oldest residents who have retired from active farming. Although the macrocosm either remained static or expanded the microcosm was a kaleidoscope of change.

**KINSHIP** The third and crucial element in fostering community cohesion is kinship. We first became aware of the importance of the

<table>
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<th>1937</th>
<th>1972</th>
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<td>Total land owned by core families</td>
<td>2880.82</td>
<td>3139.45</td>
<td>4070.23</td>
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<tr>
<td>Total number of acres in nine-section area</td>
<td>5760.00</td>
<td>5760.00</td>
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<tr>
<td>% of land controlled by core families in nine-section area</td>
<td>50.00</td>
<td>54.50</td>
<td>70.66</td>
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kin network as we probed the history of the migrating generation. Fortunately community tradition described in minute detail the make-up of the founding group, for the patterns found among them are the same for the entire migrating generation. It is these patterns which are responsible for the development of the community as an isolate.

Figure 1 depicts the migrating group of 1875, and demonstrates that although there may have been six different households, these represented only four families. For example, families 1, 2, and 3 consisted of an elderly set of parents and their four children, two of whom were married and themselves had children. Families 4 and 5 were less complex, but they, too, were extended families. Only family 6 appears as a simple nuclear family composed of parents and young children.

The characteristics revealed by the first group deserve emphasis because, as we later discovered, the same principles affected all subsequent migrants. First, we realized the importance of the kin bonds. These bonds held the group together both vertically through the generations and horizontally among the siblings. The strength of the kin ties was such that migrants represented all stages of the life-cycle, not just the youth who are traditionally regarded as most prone to moving. Lastly, and again this is an effect of kin ties, what appear initially as six nuclear families are interconnected by family bonds which reduce the group to three major social units.

Once the process of family reconstitution was well advanced we discovered that the principles visible among the first group also shaped subsequent migrations. Without the aid of reconstitution as a technique this could not have been discovered because, after the arrival of the 1875 party, the community appears to have lost interest in the dynamics of subsequent arrivals. This may be in part because there were no further transfers from Wisconsin which were organized in the elaborate manner of the founding group. Instead subsequent immigrants trickled in, usually as nuclear families. However, once we established pedigrees for all the families who lived in the community for more than one generation we stumbled onto the underlying pattern of kinship. As Figure 2 indicates, we were able to establish that the families who migrated were linked together in a giant web. For only seven core families were we unable to establish genealogical links, but in five of these
Fig. 1 Migration of Founding Group in 1875

Dotted lines encompass households, each of which is identified by an Arabic numeral. Households are appropriately grouped into family groups, each of which is given a letter. All of the persons depicted arrived in the spring of 1875 with the exception of the male in family A who is identified as arriving in the fall.
Fig. 2  Community Migration: Kinship Ring

We have placed all families whose genealogical connections could be traced on the "kinship ring." Despite the spread of arrival dates from February, 1875 to 1900, it has been possible to establish links between most of the families which arrived in the community.
instances we are convinced that they, too, were related to the community prior to migration. We feel this to be true since these five have been traced to North Brabant. Furthermore, in examining the marriages of their Oregonian offspring we found them intertwined with families who were part of the “migration ring.” Our surmise is that these five families were linked to the web through persons who remained in Wisconsin. Thus, in only two cases do we find a lack of correspondence with the general patterns for the community.

It should also be emphasized that the community migration ring reflects the same feature observed in the 1875 migration: the importance of kinship, both in terms of vertical generations, and in terms of horizontal sibling ties. It also reveals the willingness of these people to move at various stages of the life cycle, a fact which again emphasizes the importance of kin ties.

The construction of the migration ring revealed the importance of kinship in the formation of the Oregon community. It also demonstrated that the community dated its establishment to a time preceding the move to the Pacific Northwest. Furthermore, the ring gave us our first clear evidence that we were looking at an isolate. But kin ties, and with them the isolate, can evaporate quickly unless they are reinforced by each succeeding generation. Reinforcement, in turn, depends upon marital patterns.

The existence of parish records in Oregon has enabled us to examine in depth the marriage practices of the community, and we have discovered the glue which has cemented the group—certainly in Oregon, and possibly in Wisconsin and Holland as well.

There are two marriage practices which are noteworthy in this community. One is what we call “repeat alliances,” defined as the creation of multiple marriage links between given families, whether in the same or in successive generations. The second is a specialized version of the above practice which anthropologists call “sibling exchange marriages.” In this case sets of siblings from two families intermarry.

The marriages of twelve children from a particular family are shown in Figure 3. These children were third-generation Oregonians, their grandparents having migrated to the Northwest. One can see the sibling exchanging phenomenon in the marriages of children 2, 3, and 4 (three sisters who married three brothers from
Fig. 3  Marriages of a Third Generation Family

Marriages of twelve children, third generation ■ symbolizes a realliance. That is to say that in either preceding, succeeding, and/or parallel lines in this family, there were other marriages linking the same surnames. Among the eight realliances, six different surnames are represented.
another family). In addition one can see that eight of the twelve marriages served to re-ally this family to families with which they already have established ties of kinship.

The marriages in another family over three generations illustrated in Figure 4 clarifies how the repeat alliance phenomenon operates. The central family in Generation I consisted of two brothers: I, 1 and I, 2. Brother I, 2 had three sons (Nos. II-9, 10, 11), while brother I, 1 had five sons (Nos. II-2, 4, 5, 7, 8) and three daughters (Nos. II-1, 3, 6). So far as we know the wives in Generation I were unrelated. All eleven members of Generation II married, but rather than eleven new alliances there were but eight. The total number of new family alliances was less than the total number of marriages because six marriages were to pairs of siblings. Four of I, 2's children (Nos. II-2, II-3, II-4, and II-5) married pairs of siblings. I-1's son No. II-7 and I-2's son No. II-9 also married a pair of sisters. Thus, II-7 and II-9 were both first cousins and brothers-in-law.

When we look at the third generation we notice the effects of concentration even more strongly. We have been able to trace the marital history of fourteen of the youngsters of Generation III. Out of their fourteen alliances ties were established with only four families, and of these one was a repeat alliance. The latter occurred with the marriage of III-4 and III-5 who were first cousins. In this case there was legal and ecclesiastical consanguinity and, to cope with the latter, a dispensation was required.¹³ It should be emphasized that such cases appear to be extremely rare. More frequent is the type of situation illustrated by the marriage of III-6. In this case the spouses were not related genetically. However, from a social point of view their marriage served to reinforce ties between two families which were already allied both in Generation II and Generation III. The link in this case was II-6 who was aunt to both III-6 and to her husband. III-6 was the child of II-6's brother (II-5), while III-6's husband was the child of II-6's brother-in-law.

As we can see from the case of this family, marriages based upon repeat alliances or sibling exchanges serve to bind a group of people into a complex biological network. If the practices are per-

¹³ The right to marry a relative is limited both by civil and cannon law. Exceptions to the latter can be obtained in certain cases (for example, for a first cousin marriage) through an ecclesiastical dispensation.
Marriages of a Core Family: Three Generations

Three generations of marriages for a core family. ■ or ○ stand for male or female members of the core family whose marriages are traced over three generations. Generations are noted with Roman numerals. Offspring in each generation are represented by Arabic numerals.
petuated over successive generations the relationships which result become so entangled that the participants cease to understand the nature of their biological relationships. It should be emphasized that this can take place without there being an elevated number of consanguinous marriages in the legal or ecclesiastical sense of the term. However, from a biological point of view these practices, if they persist, do create a gene pool which becomes concentrated. Gradually more and more genes will become unusually frequent, and, in such a situation, the chances of recessive genes pairing increases. In this way one could anticipate the emergence of an isolate population, displaying different gene frequencies from the general population, without the usual factors of geographical, ideological, or linguistic isolation found in isolates hitherto described.

The rapidity with which a biological isolate might arise out of purely social practices would depend upon three factors: the size of the founding population, the intensity with which it practiced restricted mating, and the temporal duration of the mating patterns. Thus, for a very large group of people one would need more generations for unusual genes to become fixed or frequent among the group than one would for a small founding group, assuming the same propensity for both to mate within the group.

For our group we could assume that the migration ring is the founding group. One could envisage an isolate arising from such a founding group, but would it arise within the third or fourth generation? Perhaps, but, in view of the avoidance of legal consanguinity, this is not probable. The appearance of isolate characteristics would instead suggest that the group had existed as a community for more than three to four generations. Indeed historical data tend to support that contention. Since the relationships reflected by the marriage ring were established in Wisconsin we know that there must have been a community of sorts prior to the group’s establishment in Oregon. But genealogical knowledge fails to extend the relationship of the community temporally. To do that we would need some indication that the practices observed in the Northwest predate the move to Wisconsin.

Is there such evidence? At this point we move from what we have been able to unearth as “fact” into a hazier realm in which we have clues only. There is one pattern among the families practicing repeat and sibling alliances which suggests that these habits may predate the move to North America. Overwhelmingly the
families indulging in these practices came from the Dutch Province of North Brabant. In analyzing Oregon marriage patterns we found fifteen families who either exhibited repeat alliances or sibling exchange marriages. Of these, nine came from North Brabant, one from Amsterdam, one from Prussia, and one from Germany. There were three cases where we could not establish a point of origin for the family, but one case at least is certainly Dutch. We traced 335 marriages for these fifteen families. Of these 216, or 66.4 percent, were repeat alliances. Among the North Brabanters 190 of 283 marriages we recorded (70 percent) were repeat alliances. This suggests that the practice may have been peculiar to the European heritage of the families in question, particularly for those from North Brabant.

At first sight this proposition may seem to be far fetched. However, it is compatible with observations made by Moroni in a study of consanguinity in northern Italy. Moroni used as his source the Vatican archives on dispensations. He discovered that the rate of consanguinous marriages increased in the nineteenth century following the promulgation of the Code Napoleon. The basis for this, Moroni believes, was an effort by the northern Italians to protect family property. The Code Napoleon required the equal division of property among all offspring including the females. Applied to land, this meant that a once-handsome family plot might within one or two generations be reduced to a mosaic of useless morsels. To avoid this result, some families ingeniously arranged for intra-family marriages which enabled them to keep the overall holdings intact.

The patterns that we have seen may have been generated by a similar motive. The technique, however, was a bit different. Rather than brothers arranging that their children should marry each other, a decision which required ecclesiastical dispensation, could it be that the Brabanters utilized sibling exchanges or repeat alliances to achieve the same ends—and without the jarring need to bend the rules of their faith?

The chance detection of a case of cutaneous porphyria has led to the identification of an isolate community the existence of which has been studied over approximately 100 years in some

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depth and detail. The major findings may be cited as follows. First, the identification itself has demonstrated that isolates are not necessarily obvious. All isolates are dependent to a degree upon social behavior, but it is now clear that they are not dependent upon isolation of a geographical, ideological, or linguistic nature. They can exist purely on the basis of social practice, and they can follow those practices sufficiently unobtrusively to remain undetected. Anyone seriously interested in isolate communities needs not only to study those groups which are clearly separated from the general run of humanity, but also needs to be alert to the possibility of identifying such communities as they exist within a general population.

Second, the study has unraveled in considerable detail the social patterns and, more specifically, the nuptial patterns which have led to the formation of the isolate. Two definite patterns, realiquence and sibling exchange, have been identified as the basis of biological isolation. The identification of these patterns in turn can be applied for comparative purposes to other already known isolates or to cases where one suspects, but is as yet uncertain, that an isolate does exist.

Third, the study is a demonstration that cooperative research between the fields of biomedicine and social history can be mutually productive. These two fields have appeared to stand far enough apart in terms of their concerns and methods that they have not been envisaged as a fruitful interdisciplinary area. As a result the dialogue between the two areas has been thin, particularly at the historian's end. This state of affairs does seem to be changing for, as an editorial in *The Lancet* commented, there is interplay between "gene frequencies and history."15 In some instances historical knowledge has assisted in the understanding of genetic findings, while in others genetic data have assisted historians in interpreting the significance of archaeological data. The examples cited in *The Lancet* editorial focused on research into important problems of the past. The study reported here not only demonstrates the usefulness of interdisciplinary research linking biomedical and historical research, but also indicates that such collaboration can be utilized in what one might call current history.

In conclusion, it is appropriate to note that it is the mutual interest in social behavior which makes the cooperation of biomedical and historical researchers both possible and productive. Both disciplines are interested in tracing change over time. It is in this area that the community described here fascinates. One cannot but want to know why these particular patterns of behavior arose, why they were perpetuated, and whether in the face of modernization and urbanization the same patterns are apt to persist.
African Mortality in the Suppression of the Slave Trade: The Case of the Bight of Biafra

Studies of the magnitude and causes of African mortality in the Atlantic slave trade have been hampered by the absence of reliable records at both ends of slaving voyages, a problem which worsened in the early nineteenth century as trade from portions of Africa and by a growing number of European nations became illegal and forced the adoption of more clandestine measures. This study partially solves the problem by using the very complete records of that portion of the slave trade which was intercepted by the British navy's West African Squadron. These captured ships were escorted along the coast for adjudication before the binational Courts of Mixed Commission at Freetown in the British colony of Sierra Leone, where the slaves on them were registered, liberated, and resettled. These sources not only provide a record (differentiated by the age and sex of the captives) of the astonishingly high losses between capture and adjudication, but also permit calculation of the factors responsible for these losses. Although the conditions on captured ships were not precisely the same as those on the much larger number of ships that eluded the patrol, these records do provide some indication of mortality in the overall slave trade in this period.¹

In order to limit the number of variables, this study is confined to the slave trade from the most important slaving coast in West Africa during the first third of the nineteenth century, the Bight of Biafra, which extends east and south from the mouth of the Niger River to the equator. This coast included the main slaving ports of Bonny, New Calabar, and Brass in the eastern Niger

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Special thanks are due to David Eltis, who most generously shared the bounty of his own research, offered much useful advice and encouragement, and made helpful comments on an earlier draft of this study. Thanks are also due to Stanley L. Engerman and Stephen Baier for valuable suggestions and comments on earlier drafts. Some of the research was made possible by a grant from the Fulbright-Hays Commission of the United States Office of Education.

Delta, Old Calabar on the Cross River, and some less important trading areas further east and south known as the Cameroon and Gabon rivers. The study is based on the records of 100 ships from the Bight of Biafra apprehended by the West African Squadron between 1821 and 1839, the slaves from which were emancipated and registered in Sierra Leone.\(^2\) These 100 ships include all but a few of the ships apprehended with slaves on board from this coast, and these records account for most of the Africans originally loaded on these vessels. Excluded are the small number of captured ships lost at sea en route to Freetown and a few others that were judged to have been stopped illegally and were restored to their owners along with the captive Africans on board. Also excluded are those slave cargoes or portions of cargoes that were deposited on the island of Fernando Po and were not subsequently registered in Sierra Leone. Although these omissions are significant (since their inclusion would raise the total losses connected with the captured ships to over 19 percent) the calculations in this essay are possible only when such anomalies have been removed. Except for indicating a slightly lower number of captures and deaths than in fact occurred, the cases included here accurately mirror the actual circumstances of the trade.\(^3\)

The losses in transit to Sierra Leone are not calculated on exactly the same basis as is customary for those ships completing the middle passage to the Americas. In the first place, the losses and durations of the voyages are counted not from the point of embarkation at a port in the Bight of Biafra, but from the point

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2 The accounts of the capture of these ships and the tallies of the Africans on board are found in the British Foreign Office Slave Trade Series (F.O. 84). The records of the emancipation and registration of liberated Africans are also scattered throughout this series, but are more conveniently consulted in the "Registers of Slaves Emancipated," originally kept in Sierra Leone and now in the Public Record Office (F.O. 315/31–36). Most of the correspondence on this subject can also be found in the British Parliamentary Papers along with useful (but sometimes imperfect) summaries of African liberation figures. The registers of emancipation have not been printed. Most of the information for the sample used here was compiled from the original files. The transit times used here are entirely from the following summaries: Parl. Papers 1830, x (661), appendix 8, "Return of the Number of Vessels which have been adjudicated in the Courts of Mixed Commission" (1819–29); ibid. 1832, xlvi (Correspondence, Class A), enclosure in no. 2, "Cases adjudicated in the year 1830"; ibid. 1842, xlv (385), "Return of Vessels brought before the Courts of Mixed Commission 1830–41."

of their capture by the British patrol. The latter point seems preferable because the number of slaves alive at capture is normally based on an actual count, whereas the total at embarkation must be selected from the often conflicting testimony of the captured ship's officers and crew. In any event, since nearly all of these captures took place in port or within a short time after sailing and before many deaths could occur, this decision introduces only a very slight downward bias in the overall mortality rate, a bias that is more than offset by the fact that the losses in transit and the duration of the voyages are not calculated on the basis of identical time periods. A captured ship's voyage ended upon its arrival at Freetown, but the emancipation and registration of liberated Africans did not take place until the ship had been officially judged and condemned by the appropriate binational Mixed Commission there, a process which generally took only a few days, but when backlogs or disputes occurred could drag on for weeks. Until this process was completed captive Africans generally remained on board the slave ship, where they were given medical care, food, and water. Because these circumstances delayed the registration of Africans, the mortality figures used here represent deaths over a somewhat longer period of time than the actual duration of the voyage to Freetown, and because deaths were more likely to occur at the end of a voyage than at the beginning, this fact produces a small upward bias in the mortality figures when compared to transatlantic losses. At the same time, this manner of accounting does provide a more complete picture of the mortality in the slave trade since deaths occurring after the arrival in Freetown were directly attributable to the rigors of the trade.4

One of the most obvious factors governing the rate of mortality on ships from the Bight of Biafra, as from elsewhere in Africa, was the length of the time at sea. Slave ships were crowded and insanitary to begin with, so the longer the voyage the more likely were shortages and spoilage in food and water supplies and

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4 Using records of the Africans alive upon arrival in Sierra Leone generously supplied by David Eltis, it has been possible to compute the difference between losses at arrival and those at registration for a significant portion of my 100 ships. For the period from 1821 to 1825 there was no significant difference from the losses reported in Table 2, but the losses at registration were higher by 1.1 percentage points for 1826 to 1830, by 2.1 points for 1831 to 1835, and by 3.0 points for 1836 to 1839.
### Table 1 Mortality between the Bight of Biafra and Sierra Leone related to Length of Voyage and compared with Transatlantic Mortality

<table>
<thead>
<tr>
<th></th>
<th>TO SIERRA LEONE, 1821–39</th>
<th>TO AMERICAS, 1817–43&lt;sup&gt;a&lt;/sup&gt;</th>
<th>TO RIO DE JANEIRO, 1825–30&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>DAYS</td>
<td>% OF SHIPS</td>
<td>AVERAGE LOSS (%)</td>
<td>% OF SHIPS</td>
</tr>
<tr>
<td>10–19</td>
<td>11</td>
<td>9.6</td>
<td>&gt;1</td>
</tr>
<tr>
<td>20–29</td>
<td>32</td>
<td>15.5</td>
<td>23</td>
</tr>
<tr>
<td>30–39</td>
<td>31</td>
<td>18.1</td>
<td>40</td>
</tr>
<tr>
<td>40–49</td>
<td>13</td>
<td>23.8</td>
<td>12</td>
</tr>
<tr>
<td>50–59</td>
<td>3</td>
<td>22.6</td>
<td>10</td>
</tr>
<tr>
<td>60–69</td>
<td>7</td>
<td>28.0</td>
<td>—</td>
</tr>
<tr>
<td>70–79</td>
<td>1</td>
<td>31.4</td>
<td>14</td>
</tr>
<tr>
<td>80–89</td>
<td>1</td>
<td></td>
<td>14</td>
</tr>
<tr>
<td>90+</td>
<td>1</td>
<td></td>
<td>&gt;1</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>17.9</td>
<td>100</td>
</tr>
</tbody>
</table>

<sup>a</sup> From Philip D. Curtin, *The Atlantic Slave Trade: A Census* (Madison, 1969), Table 81, based on a sample of 206 ships known to the British Foreign Office listed in Parl. Papers 1845, xlix (73).

<sup>b</sup> From Herbert S. Klein and Stanley L. Engerman, "Shipping Patterns and Mortality in the African Slave Trade to Rio de Janeiro, 1825–30," *Cahiers d'Etudes Africaines*, XV (1975), Table 9, based on the newspaper records of 386 ships.

The more common the spread of infectious diseases. The relationship between the losses suffered and the length of the voyage from the Bight of Biafra is shown in Table 1 along with two transatlantic series which are drawn primarily from other parts of the continent. Although all three series show that mortality increased with the time at sea, deaths on captured ships mounted less rapidly on the longest voyages since shortages of food and water were less likely to occur. More significant is the higher magnitude of loss on short and average length voyages in the series of captured ships. In part this is because neither transatlantic series includes ships from the Bight of Biafra, which other records suggest had an especially high mortality rate in crossing the Atlantic. This and other possible causes of these differences will be considered below.

The distribution of ships over time in Table 1 might be taken to suggest that voyages to Sierra Leone were generally faster than those to the Americas were it not for the fact that both transatlantic tabulations include ships from Mozambique, which had enormously long crossings (and notoriously high mortality rates).
Table 2  Mortality between Capture and Registration by Quinquennia and Decades, 1821-1839

<table>
<thead>
<tr>
<th>NUMBER OF SHIPS</th>
<th>AVERAGE DAYS IN TRANSIT</th>
<th>ALIVE AT CAPTURE</th>
<th>REGISTERED IN SIERRA LEONE</th>
<th>AVERAGE LOSS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1821-25</td>
<td>12</td>
<td>49</td>
<td>2,630</td>
<td>2,103</td>
</tr>
<tr>
<td>1826-30</td>
<td>26</td>
<td>39</td>
<td>6,311</td>
<td>4,850</td>
</tr>
<tr>
<td>1821-30</td>
<td>38</td>
<td>43</td>
<td>8,941</td>
<td>6,953</td>
</tr>
<tr>
<td>1831-35</td>
<td>31</td>
<td>30</td>
<td>10,668</td>
<td>9,020</td>
</tr>
<tr>
<td>1836-39</td>
<td>31</td>
<td>28</td>
<td>9,037</td>
<td>7,536</td>
</tr>
<tr>
<td>1831-39</td>
<td>62</td>
<td>29</td>
<td>19,705</td>
<td>16,556</td>
</tr>
<tr>
<td>1821-39</td>
<td>100</td>
<td>34</td>
<td>28,646</td>
<td>23,509</td>
</tr>
</tbody>
</table>

Source: The sources for Tables 2-6 are indicated in footnote 2.

In fact it appears that the transit time to Sierra Leone was actually about the same as the average transatlantic time. The transit time to Sierra Leone of the sample from the Bight of Biafra captured by the British navy from 1821 to 1839 averaged thirty-four days (Table 2), while twenty-seven ships arriving in Bahia during the first half of 1830 “chiefly from the Ports of Onim [Lagos], Bonny, New Calabar, &c.” took an average of thirty-two days.⁵

The main reason for this long transit time was that Sierra Leone, from a navigational point of view, was a poor choice as the seat of the Mixed Commissions. Situated far from the Bights of Benin and Biafra, the principal slaving centers in West Africa during the nineteenth century, Sierra Leone could be reached only by sailing against the prevailing winds and currents along the coast. The shortcomings of the Colony as the seat of the Commissions were accurately stated by an investigating committee of Parliament in 1830:

...it is the opinion of this Committee, That the situation of the Mixed Commission Court at Sierra Leone, for the adjudication of

⁵ Consul John Parkinson to Earl of Aberdeen, Bahia, Oct. 13, 1830, in Parl. Papers 1831, xix (Correspondence, Class A), no. 62. This is the source, imperfectly copied, for entries 1138-1171 in the summary list entitled “Return of the Number of Slave Vessels arrived in the Transatlantic States since 1814,” Parl. Papers 1845 xlix (73). The extensive work by Eltis on the whole range of British records of the slave trade in this era strongly suggests that, despite the Consul’s statement, nearly all of these twenty-seven ships to Bahia were from Lagos. Nevertheless, on the basis of presently available evidence this is as close as one can come to calculating sailing times from the Bight of Biafra to the New World in this era.
captured Slaves, is highly inconvenient for that purpose, consider-
ing that the Slaves are captured chiefly at the distance of 800 or 1,200 miles to the Eastward; and that as a current constantly sets from West to East, the captured ships are sometimes eight or nine weeks, and on the average, upwards of five weeks, on their passage from the place of capture to Sierra Leone; occasioning a loss of the captured Slaves, amounting to from one-sixth to one-half of the whole number, whilst the survivors are generally landed in a mis-
erable state of weakness and disease.  

The solution to this regrettable situation, in the Committee’s view, was to relocate the Mixed Commissions on Fernando Po, an island in the Bight of Biafra within easy sailing distance from the major slaving ports of West Africa. Some slaves were landed on the island about this time and it was later used as a residence for the Consul to the Bights of Benin and Biafra, but this sensible recommendation was never carried out because Spain, which claimed Fernando Po but had never occupied it, refused to sell the island to Britain. Without sovereignty Britain could not le-
gally guarantee the freedom of liberated Africans and so the proj-
ect was abandoned.  

Despite this major setback some reduction in losses en route to Sierra Leone did take place after 1830 as the result of other efforts begun in the previous decade. Changes made in the British patrol in the late 1820s had increased the number of captures and reduced the transit time to the Colony by an average of ten days between the period 1821 to 1825 and 1826 to 1830, although there was no corresponding reduction in mortality, which in fact increased (Table 2). One of the major problems with the patrol had been the poor quality of its ships. Most patrol vessels were either large, slow frigates, the tall masts of which rounding the horizon gave slave ships enough advance warning to escape, or “smaller ships, . . . mostly Seppings brigs, which everyone agreed sailed like haystacks, compared with the clean lines of the slaving schooners.” However, encouraged by the practice of granting “prize-money” to the captors, enterprising naval officers had be-
gun buying condemned slave vessels to serve as tenders to their official ships. The incorporation of the Henriqueta (renamed Black Joke) into the patrol in this manner in 1828 had greatly increased captures and reduced sailing times. Other measures aimed specifically at saving African lives, when combined with better sailing times, had cut losses so that by the 1830s the transit time to Sierra Leone had been reduced from an average of six weeks in the previous decade to an average of four weeks.\(^8\)

As Table 3 shows, this faster sailing time was accompanied by a reduction in losses from 22.2 percent to 16.0 percent. Not all of this reduction can be attributed to the shorter sailing time since changes in the condition of slaves at the point of supply, in carrying techniques, and in the care provided by the British patrol also would have affected this figure. It is possible to isolate the reduction in losses attributable to the shorter sailing time from these other factors by redistributing the slaves captured from 1821 to 1830 according to the faster sailing times of the 1831 to 1839 ships.\(^9\) Such a projection gives a mortality rate of 20.2 percent, indicating that the faster sailings of the 1831 to 1839 period would have reduced the 1821 to 1830 losses by 2 percentage points. Since

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\(^8\) Lloyd, *Nav\y*, 71; Ward, *Royal Navy*, 128. From 1830 patrol ships were required to place a medical officer on captured vessels to treat those who were ill; see *Parl. Papers* 1831–32, xlvii (Correspondence, Class A), no. 4.

\(^9\) This is accomplished by multiplying the percentage of slaves in each sailing category from 1831 to 1839 by the average mortality rate for the same category from 1821 to 1830. Thus for 10–19 day voyages: .138 \times .084 = .0116. Similarly for 20–29 days we get .1010, for 30–39 days, .0618, for 40–49 days .0280, giving a total loss of 20.2% in this projection versus actual losses of 22.2%.
Table 4  Mortality between Capture and Registration by Month of Capture and by Principal Month en route to Sierra Leone, 1821–39

<table>
<thead>
<tr>
<th>MONTH OF CAPTURE</th>
<th>NUMBER OF SHIPS</th>
<th>AVERAGE DAYS IN TRANSIT</th>
<th>AVERAGE LOSS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>7</td>
<td>45</td>
<td>18</td>
</tr>
<tr>
<td>February</td>
<td>9</td>
<td>42</td>
<td>16</td>
</tr>
<tr>
<td>March</td>
<td>8</td>
<td>39</td>
<td>21</td>
</tr>
<tr>
<td>April</td>
<td>13</td>
<td>31</td>
<td>17</td>
</tr>
<tr>
<td>May</td>
<td>5</td>
<td>29</td>
<td>28</td>
</tr>
<tr>
<td>June</td>
<td>3</td>
<td>25</td>
<td>16</td>
</tr>
<tr>
<td>July</td>
<td>4</td>
<td>28</td>
<td>11</td>
</tr>
<tr>
<td>August</td>
<td>10</td>
<td>39</td>
<td>25</td>
</tr>
<tr>
<td>September</td>
<td>10</td>
<td>31</td>
<td>16</td>
</tr>
<tr>
<td>October</td>
<td>13</td>
<td>30</td>
<td>14</td>
</tr>
<tr>
<td>November</td>
<td>8</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>December</td>
<td>10</td>
<td>34</td>
<td>24</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>34</td>
<td>18</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>MONTH EN ROUTE</th>
<th>NUMBER OF SHIPS</th>
<th>AVERAGE DAYS IN TRANSIT</th>
<th>AVERAGE LOSS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>10</td>
<td>38</td>
<td>23</td>
</tr>
<tr>
<td>February</td>
<td>8</td>
<td>41</td>
<td>19</td>
</tr>
<tr>
<td>March</td>
<td>8</td>
<td>40</td>
<td>21</td>
</tr>
<tr>
<td>April</td>
<td>12</td>
<td>41</td>
<td>19</td>
</tr>
<tr>
<td>May</td>
<td>12</td>
<td>33</td>
<td>18</td>
</tr>
<tr>
<td>June</td>
<td>4</td>
<td>22</td>
<td>30</td>
</tr>
<tr>
<td>July</td>
<td>4</td>
<td>20</td>
<td>12</td>
</tr>
<tr>
<td>August</td>
<td>9</td>
<td>32</td>
<td>22</td>
</tr>
<tr>
<td>September</td>
<td>7</td>
<td>38</td>
<td>14</td>
</tr>
<tr>
<td>October</td>
<td>11</td>
<td>29</td>
<td>14</td>
</tr>
<tr>
<td>November</td>
<td>14</td>
<td>33</td>
<td>14</td>
</tr>
<tr>
<td>December</td>
<td>5</td>
<td>26</td>
<td>11</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>34</td>
<td>18</td>
</tr>
</tbody>
</table>

The actual reduction in mortality from the earlier period to the later one was 6.2 percentage points, it may be concluded that the various other factors influencing mortality rates were twice as important for this reduction as was the shortened sailing time.

Another factor linked with the mortality rate was the time of year in which the sailing took place. In order to explore possible links between weather and mortality two series were constructed, both shown in Table 4. The first distributes ships and mortality by the month of capture; the second by the month during which they spent the greatest number of days en route to Sierra Leone after capture. Neither arrangement shows any consistent linkage between the time of year and the rate of mortality. What does show up is a remarkably consistent reverse congruence between the average rainfall per month in the Bight of Benin and the average number of days required to reach Sierra Leone. This trend exists in both series but is stronger in the arrangement by month of capture, indicating a strong influence of the weather during the early part of the voyage on the length of its overall duration. It was not the rain itself which sped the ships on their way but the
accompanying winds, the amount of rainfall being a convenient quantifiable measure of the seasons along the West African coast. During the long dry season in November-February and the short dry season in August winds were lighter and consequently the voyages took much longer.  

The health of the slaves might be expected to have varied more directly with the amount of rain since many travelled exposed on the main deck and the others had to endure the stifling heat and foul air below decks when rain and rough seas necessitated closing the ventilators. However, the ill effects of the storms were partly offset by the shorter duration of the voyage in the rainy season except when either factor was acute. Then the mortality rate swung upward. This is illustrated most clearly by using the distribution of ships by the principal month of voyaging (rather than by the month of capture), indicating the cumulative nature of these effects. Thus the three peaks in mortality (the dotted line in Fig. 1) in January, June, and August correspond to the extremes either in the amount of rainfall or in the duration of the voyages. The January and August extremes correspond with the peaks of the dry seasons (and longest sailing times), while the apex of mortality occurred in June at the peak of the rains (despite the shortest average sailing times). At other times of the year the mortality rate was pulled in opposite directions by these two factors and occupied an intermediate position.

A cause of mortality on slave ships that has not been measured here is overcrowding. Although it was once common to attribute large numbers of deaths to cruel and greedy captains who maltreated their slaves, especially by packing excessive numbers of them into their ships, several recent studies have dis-

10 In studying the Congo-Angola slave trade, Herbert S. Klein and Stanley L. Engerman found a strong correlation between high mortality and sailing times made during the stormy season: “Shipping Patterns and Mortality in the African Slave Trade to Rio de Janeiro, 1825–30,” Cahiers d’Etudes Africaines, XV (1973), 394. Whether the slave traders were aware of these seasonal variations and were influenced by them is a question that cannot be answered on the basis of the present sample, which may reflect the sailing preferences of the British patrol as likely as those of the slavers. A study by W. E. Minchinton suggests that reaching the Americas during the summer months was the prime concern for slavers to South Carolina and Virginia. See his “The Slave Trade of Bristol with the British Mainland Colonies in North America 1699–1770” in Roger Anstey and P. E. H. Hair (eds.), Liverpool, the African Slave Trade, and Abolition (Liverpool, 1976), 47–49.
Fig. 1 Monthly Variations in Rainfall, Mortality, and Transit Time to Sierra Leone, 1821–1839.

source: The average rainfall in the Bight of Benin measured in inches at Lagos-Ikeja is from William A. Hance, *The Geography of Modern Africa* (New York, 1975; 2nd ed.), 48. The average number of days in transit (by month of capture) from the Bight of Biafra to Sierra Leone and the average loss of life among slaves (by principal month en route) are from Table 4.

counted the importance of overcrowding as a factor directly linked to high transatlantic mortality. In the case of the ships captured in the Bight of Biafra the possibility of more than ordinary mistreatment was reduced through supervision by the British captors, although overcrowding was sometimes relieved by transferring some Africans to other captured ships or by disembarking some on the island of Fernando Po. Because of these and other anomalies in the sample used here, which made conditions on captured ships significantly different from those which existed during the middle passage to the Americas, and because of the difficulties of accurately converting different measures of capacity to a single system, no attempt has been made to produce slave/capacity ratios. However, the records of individual ships


12 Some ships were taken before they had completed their cargoes; others after partly unloading their slaves as the patrol ship approached, since until “equipment” treaties were signed in the 1830s and 1840s only ships actually carrying slaves could be captured.
do support the subjective impression that little correlation existed between mortality rates and the conditions on the captured ships.

In the case of the ship *Invincival*, which was captured in the Cameroon estuary just before Christmas in 1826, only 250 of 440 slaves on board at capture survived the journey to Sierra Leone, a loss of 43 percent. The British captors reported that the ship carried food and water “in abundance and of the best quality,” but that it was “terribly overcrowded” with 200 African boys being kept on deck and in the lifeboats sheltered only by spare sails. Despite these crowded conditions, the ship was actually carrying sixty slaves less than the 500 a ship of its size was permitted under Brazilian law. In part, the captors attributed this high mortality to the fact that the captain and crew had fallen sick while trading in Cameroon and had also become embroiled in a dispute with the African slave traders there. As a consequence the slaves had been loaded with unusual haste into an ill-prepared and dirty ship, a fact which promoted the rapid spread of disease among the slaves. As was usual on Brazilian ships there was no doctor to minister to the sick. Moreover, during the voyage to Sierra Leone the ship had the extreme ill-luck to the struck by lightning twice and to encounter twenty-seven consecutive days of heavy rains. Temporary repairs to the damaged masts and these storms delayed the ship’s progress so that it took two months to reach Freetown. Another two weeks elapsed before the adjudication was completed.13

Although the lives of 190 African captives were lost on this voyage, there seems little in the circumstances of it to warrant placing unusual blame on the Brazilian captain and crew, who could not have anticipated their own illness, the dispute, the capture, or the necessity of navigating against foul weather en route to Sierra Leone. This does not mean that they were blameless, but in carrying no medical officer and in filling the decks with slaves they were doing no differently than other slave traders who did not suffer such enormous losses.

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13 *Parl. Papers* 1828, xxvi (Correspondence, Class A) no. 53: Commissioners to Canning, Sierra Leone, Mar. 31, 1827; *Parl. Papers* 1830, x (661). “Report from the Select Committee,” evidence of Captain William Jardin Purchase, 24–25. Brazilian law limited the number of slaves to 5 per 2 tons of registered burden, while British law from 1788 to 1799 limited its slavers to 5 slaves for every 3 tons up to 200 registered tons and 1 slave per ton thereafter. See Klein and Engerman, “Slave Mortality,” 119–121.
Two other Brazilian cases will underline the vagaries of the trade. The *Silveirinha* was captured at Old Calabar in March 1827 with 266 slaves on board, sixty-one more than its small size (eighty-two tons) permitted even by the lax Brazilian law, making it grossly overcrowded. Since the small ship sailed poorly against the wind and currents, it took over eleven weeks to reach Freetown, but its losses, although substantial enough, were still not far from average for this period: 21.4 percent. A month later the *Creola* was taken with 308 slaves from Old Calabar, and the record of the captor is vehement with denunciations of her captain:

Although she was limited to carry only 214 slaves, the rapacity of her inhuman master induced him to cram nearly 100 more into her, making upwards of seven for every two tons, aggravating in a most cruel degree the horrid misery of his victims by such studied barbarity; putting the law of his own government at defiance, as well as the treaty and the convention [with Britain]. On her arrival here she presented the shocking spectacle of a living mass, etc.

Yet, taken in tow by her captor, she arrived in Sierra Leone after a remarkably short voyage of only two weeks having lost twenty of her slaves for a mortality rate of only 6.5 percent. Compared to the captain of the *Invincival*, the masters of these other two ships were surely less mindful of the safety of their slaves, yet their cruelty is not reflected in the actual rates of mortality aboard their ships, leading one to doubt that overcrowding was directly linked to loss of life in a significant way.

The causes of mortality considered so far refer primarily to the conditions on captured ships; the remainder of this essay examines factors that should apply equally to captured ships and to those successfully reaching the Americas. Because of poor records for the transatlantic slave trade in this period calculations of mortality have varied. James Bandinel, long time chief clerk and superintendent of the British Foreign Office’s Slave Trade Department, advised Parliament in 1848 that the transatlantic mortality during the previous two decades had been about 24

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14 *Parl. Papers* 1828, xxvi (Correspondence, Class A), no. 69: Commissioners to Canning, Sierra Leone, June 20, 1827.
15 *Ibid.*, no. 68: Commissioners to Canning, Sierra Leone, June 18, 1827.
percent, a figure he arrived at by calculating losses on captured ships taken to Freetown at 18 percent and adding another 6 percentage points to account for the longer distance to the Americas. This figure can no longer be accepted, not least because, as was suggested above, sailing times to the Americas from key slaving points such as the Bight of Biafra were generally not longer than those to Sierra Leone. In contrast, a study by Curtin of British records from 1817 to 1843 has arrived at a mortality rate of only 9 percent, and a more limited study by Klein and Engerman of the slave trade to Rio de Janeiro based on Brazilian records for the period from 1825 to 1830 has arrived at a mortality rate of 7 percent (See Table 1). However, new work by Eltis making more critical use of a broader range of sources, strongly suggests that these figures are much too low, both because the samples used include few ships from high loss areas such as the Bight of Biafra and because the records themselves probably underreport losses for the regions that they do consider. Eltis estimates that transatlantic mortality from 1821 to 1843 ranged from 7 to 25 percent depending on the African region of origin and the American region of importation.16

Significantly, Eltis postulates a mortality rate of 17.2 percent for ships from the Bights of Benin and Biafra to the Spanish and French Caribbean, the primary destination of slaves from these coasts in this era. He also maintains that ships from the Bight of Biafra had the highest losses on the Atlantic side of Africa. This reinforces the conclusion of another study by Klein and Engerman of British ships trading in slaves to the West Indies in the 1790s which reports that ships from the Bight of Biafra suffered losses double the average for this group. There are several reasons why losses from the Bight of Biafra ought to have been higher than those from elsewhere on the Atlantic side of Africa, but the full explanation may still be obscure. Sailing times and sailing conditions on this coast were certainly one factor. Because the winds and currents ran strongly from the west, ships leaving the Bight commonly sailed south and east before swinging round near the

equator to head for the Americas, a detour that added one or more weeks to their voyages. Klein and Engerman’s figures show, for example, that ships from the Bight of Biafra took longer to reach their destinations than those from any other African coast (except the Bight of Benin, which is represented in their study by only three ships). Moreover, as was shown above, the weather conditions encountered in the Gulf of Guinea during the early days of the voyage markedly affected the losses on captured ships and presumably (though the routes were different) on those that escaped detection as well. However, Klein and Engerman found too little correlation in their sample from the 1790s to warrant attributing the losses to these conditions of shipment, arguing instead that the high losses from the Bight of Biafra “must relate to the nature of the African supply source,” that is, to the conditions under which the Africans were enslaved, taken to the coast, and readied for shipment.17

Since the identification of African supply conditions as most responsible for these high losses was arrived at by eliminating other possibilities rather than by an analysis of the actual African supply conditions, this conclusion must remain open to some doubt. Many conditions could have been significant and most are known imperfectly if at all. Certainly what is known of the general conditions of supply to this coast suggests no obvious circumstance that can be used to explain the high mortality rates encountered. The hinterland of the Bight of Biafra was densely populated and may have been experiencing some shortages of food at that time, but there is no reason to think that this produced conditions notably worse than elsewhere on the continent. Rather the density of the population meant that the slaves exported were drawn principally from among the Igbo (Ibo) and Ibibio peoples just behind the coast and thus were not subjected to the long marches that have been suggested as causes of high mortality elsewhere.18 Moreover, the absence of large-scale states among

18 The operation of the slave trade in this region is discussed in considerable detail in Northrup, Trade Without Rulers, esp. chs. 3–6. The main lines of this region’s economy in the period under consideration are described in Northrup, “The Compatibility of the Slave and Palm Oil Trades in the Bight of Biafra,” Journal of African History, XVII (1976), 353–364.

Faced with a discrepancy between the losses in transit on slave ships from West Africa
these peoples meant an absence of the major conflicts and disruptions which sent so many prisoners of war and refugees into the overseas slave trade elsewhere. Instead, most of those enslaved seem to have been the victims of kidnapping or of sales for various reasons by fellow villagers. Perhaps because of these circumstances the Bight of Biafra supplied a greater proportion of female slaves than was usual elsewhere in Africa as is attested by a small sample of cargoes near the end of the seventeenth century and by a larger sample near the end of the eighteenth century. However, there is no known connection between sex ratios in the slave trade and mortality. In any event, by the 1820s and 1830s the proportion of males (62 percent) was more typical of the Atlantic trade overall.19

Although the proportion of females had fallen, the proportion of children had increased from one-eighth in the late seventeenth century to two-fifths of the total in the first half of the nineteenth century. Table 5 suggests a strong link between the proportion of children in a cargo and the mortality rate incurred. Because the age ratios at capture are unknown, it is impossible to

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19 The earlier sample (5 ships) contained 56% males and 13% children; see Northrup, *Trade Without Rulers*, Table 1. The latter sample (over 100 ships) contained 56.6% males and about 13% children; see Klein and Engerman, “Slave Mortality,” 119 and Table 3.
calculate the actual mortality ratios of adults to children on these voyages and thus the precise nature of this link is unknown. Children may simply have died more readily or there may have been some more complex contagion effect which also raised adult mortality.\textsuperscript{20}

Another approach to the African supply situation is to consider the variations in mortality among the ports of call in the Bight of Biafra. Table 6 shows that mortality and transit times rose with the distance of the trading sites from Freetown, being lowest at the Nun branch of the Niger and progressively higher at points to the east. The direction of this trend is not surprising, but the rate at which mortality rose appears out of proportion to the additional time and distance involved. The losses are particularly out of line in the eastern part of the Bight at Old Calabar and even more in the Cameroon estuary.

Part of the explanation of the higher losses in the eastern Bight seems to be the higher proportion of children shipped from these ports than from the Rio Real (Table 6, column 6).\textsuperscript{21} It is possible that a higher mortality on the arduous voyage to Freetown also resulted from slaves from Old Calabar and the Cameroon estuary being in a weaker physical condition than those shipped from elsewhere in the Bight. Although the information on this point is short on details, oral traditions and other sources suggest the strong possibility that in the 1820s and early 1830s a large proportion of the slaves shipped from the Cameroon estuary were in a weakened condition. During this period the highlands of central Cameroon were rocked by a succession of raids and wars separately initiated by the Chamba-led Bali (Ba’ni), the Fulani of the Adamawa Emirate, the Bamum, and others. These conflicts created widespread disruption and dislocation that resulted in many persons being sold into the overseas slave trade.\textsuperscript{22}

\textsuperscript{20} Whether children formed a significantly larger share of the slaves from the Bight of Biafra than from elsewhere in Africa in this period is not known.
\textsuperscript{21} Children appear to have been particularly vulnerable to long sailing times. Table 6 shows that ships from Brass, which carried slightly more than the average proportion of children but had much shorter than average sailing times, suffered losses averaging only 9\%, although ships from the Cameroon estuary, which had much longer sailing times as well as a much higher proportion of children than was average, had losses over three times as severe. The Gabon figures do not agree with this pattern, a fact that is probably to be explained by the statistically inadequate number of ships from that port.
\textsuperscript{22} There is no single reliable account of this area in that era, but aspects of its history are treated in Merran McCulloch, Margaret Littlewood, and I. Dugast, \textit{Peoples of the
Table 6  Mortality and Proportion of Children in Cargo by Area of Origin, 1821–39

<table>
<thead>
<tr>
<th>Area of Origin</th>
<th>Number of Ships</th>
<th>Average Days in Transit</th>
<th>Alive at Capture</th>
<th>Registered in Sierra Leone</th>
<th>Average Loss %</th>
<th>Children %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nun River (Brass)</td>
<td>9</td>
<td>26</td>
<td>2,611</td>
<td>2,376</td>
<td>9.0</td>
<td>41.0</td>
</tr>
<tr>
<td>Rio Real (Bonny and New Calabar)</td>
<td>51</td>
<td>32</td>
<td>15,180</td>
<td>12,847</td>
<td>15.4</td>
<td>34.6</td>
</tr>
<tr>
<td>Cross River (Old Calabar)</td>
<td>26</td>
<td>37</td>
<td>7,377</td>
<td>5,803</td>
<td>21.3</td>
<td>43.8</td>
</tr>
<tr>
<td>Cameroon River (Bimbia, etc.)</td>
<td>12</td>
<td>35</td>
<td>2,943</td>
<td>2,039</td>
<td>30.7</td>
<td>53.0</td>
</tr>
<tr>
<td>Gabon River</td>
<td>2</td>
<td>40</td>
<td>535</td>
<td>444</td>
<td>17.0</td>
<td>51.4</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>34</td>
<td>28,464</td>
<td>23,509</td>
<td>17.9</td>
<td>39.4</td>
</tr>
</tbody>
</table>

Koelle’s biographies of liberated Africans in Sierra Leone from central Cameroon show that many victims of these disruptions were sold from the Cameroon estuary and Old Calabar, a fact that may have raised losses from these ports. However, the effects would have been less pronounced in Old Calabar, which drew many more slaves from other areas, than in the Cameroon estuary, which was more dependent on central Cameroon supplies.23

In conclusion, it may be said that the factors governing the mortality on slave ships from the Bight of Biafra captured and condemned in Sierra Leone in the period from 1821 to 1839 fall into two broad categories. First are those general circumstances which determined that the overall mortality would be high. These include the normally high proportion of children supplied along this coast, the distance to be covered to Sierra Leone, and the difficulties of navigating along that route, particularly in the Gulf of Guinea. The second category includes those factors which governed the sizeable variations in mortality from ship to ship. The most significant of these were the time of the year in which the sailing took place (because of variations in winds and rains) and the port at which the slaves were purchased (because of

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variations in the physical condition of the slaves, the proportion of children offered for sale, and the distance to be covered to Sierra Leone).

Some of these factors would also have affected mortality rates on transatlantic voyages, notably the condition of the slaves at loading and the proportion of children. Problems of navigation were common to all ships, but the actual circumstances were different enough that the normal transatlantic crossing might well have been less costly of life than the hard passage to Sierra Leone. However, any lessening of the relative mortality thus achieved would probably have been offset by the measures that the patrol took to relieve congestion and shortages of supplies on captured ships. Finally, it is possible that ships captured by the patrol were not a random sample of the total trade, since faster ships and more alert crews may have been more successful in eluding capture. Measurement of some of these variations may come from future research, but it seems reasonable to conclude that the mortality in the transatlantic slave trade from the Bight of Biafra to the Americas in this era (when measured to include passage-related deaths occurring within a reasonable period after the actual end of the voyage) was of a comparable order of magnitude to that reported here for captured ships.
Kenneth F. Kiple and Virginia H. Kiple

Deficiency Diseases in the Caribbean  The historiography of slavery in the Americas has recently taken on a new biological dimension as historians have begun to appreciate the importance of pathogenic agents in any holistic understanding of their subject. Of special interest is the impact of these agents on slave mortality and, more specifically, the extent of the role that they played in preventing most Caribbean slave populations from sustaining a natural rate of growth.

Historians are also looking beyond pathogens to the nutritional factor, which may have figured prominently in the etiologies of slave diseases. Seldom does a new work appear which does not allege that malnutrition was a serious problem of Caribbean slave health. However, no attempt has been made to single out specific nutritional deficiencies in the Caribbean slave diet and in the process prove that Caribbean bondsmen were malnourished. Nor for that matter has any effort been made to link suspected nutritional deficiencies with some of the more important West Indian slave diseases.

This study makes such an attempt by investigating the West African nutritional heritage of Caribbean slaves, by analyzing the Caribbean slave diet, and by matching nutritional deficiencies revealed by this analysis with the symptoms of the diseases which plagued slaves exclusively.

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This study is an elaboration of the Caribbean portion of the paper “Slave Nutrition and Disease during the Nineteenth Century: The United States and the Caribbean,” which was delivered at the annual meeting of the Organization of American Historians (1979). The authors wish to thank the Joint Committee on Latin American Studies of the Social Science Research Council and the American Council of Learned Societies for an award to support the project out of which this research was generated. They are grateful also to the Bowling Green Faculty Research Committee for assistance with supplementary travel funds.

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© 1980 by The Massachusetts Institute of Technology and the editors of The Journal of Interdisciplinary History.
THE AFRICAN NUTRITIONAL HERITAGE Although there were some exceptions, West African diets were poor. Prior to the sixteenth century, they consisted of bananas, taro, the small African yam, millet, and rice crops, which researchers believe did little more than sustain life. In the sixteenth century, American cassava and maize were imported, and are credited with stimulating the growth of West Africa's population—a growth that kept pace, or even exceeded, the drain of the slave trade.1

Although the introduction of these two starchy plants may have resolved problems of quantity, the quality of West African diets remained deficient. Animal protein has never played a major role in West African nutrition, with much of the blame belonging to the tsetse fly—a bloodsucking insect which imparts African sleeping sickness to animals as well as to man. In much of West Africa the tsetse fly was so prevalent that it made the raising of cattle and other large animals, if not in all cases impossible, at least unprofitable. Thus many West Africans were limited to keeping a few goats, chickens, and dogs and sometimes a pig—animals so scarce and highly prized that they were slaughtered only on special occasions.2

Bovine milk was thus excluded from West African diets, which may be a reason for the high frequency of lactose intolerance today among blacks of West African origin. Such intolerance occurs among people with a history of low milk consumption. Also excluded in many places were eggs, for some because of taboos against their consumption, and for others because it seemed wasteful to eat the egg rather than wait for the chicken. Finally, because of cultural beliefs, fruit consumption was frequently frowned upon as was the use of most vegetables, except the yam, taro, cassava, and maize.3

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3 Although West Indian figures are not available, about three-quarters of the Afro-Americans living in the United States are lactose intolerant, meaning that, because they have low levels of the lactase enzyme which breaks down milk sugars, they cannot drink
One consequence of this background was that many West Africans must have reached the New World with a history of malnutrition. Empirical data suggest that this was the case. Research conducted by Fogel, Engerman, and Higman concerning the height of some 25,000 Trinidadian slaves indicates that newly imported Africans were significantly shorter on average than Creole-born slaves. Fraguinals has found the same to be true for Cuba. First-generation Creole slaves were significantly taller than freshly imported Africans.  

Research has demonstrated that a radical change in dietary habits produces a dramatic increase in height. The rapid growth of New World slaves over the course of a generation or so implies that West Indian diets were at least more protein laden than those of West Africa and were probably also of better overall quality. We discuss West Africa in order to emphasize the poor nutritional status of a sizable portion of slaves in the Caribbean.  

West Indian slave populations were seldom self-sustaining so long as the slave trade endured. Rather, they received massive injections of fresh imports from that traffic until the beginning of the nineteenth century and, in the case of Cuba, through the middle of that century. Hence Caribbean slave populations always had many badly nourished newcomers crowding their ranks—a condition which the circumstances of the Middle Passage could only have aggravated. The standard menu for slaves making the passage was a boiled cereal (usually rice) with a “sauce” made by boiling salted fish—a diet lacking in many important nutrients.


Moreover, the dysentery and diarrhea, always rise aboard a slaver, would have leached away many of those nutrients which such a diet could have provided.\(^6\)

Thus many West Africans, badly nourished to begin with and then subjected to the disastrous nutritional circumstances of the Middle Passage, reached the New World in a malnourished condition. Doubtless some never recovered, which must count as an important reason for the high incidence of "seasoning" mortality.

Malnutrition, in addition to its own inherent destructiveness, also renders the body more susceptible to pathogenic invasion and, in the case of slaves moving into a new disease environment, to pathogens and strains of pathogens against which they had inefficient defenses. Assuming, however, that the newly imported slaves did survive, they did so on a diet which, although in many ways superior to their accustomed diets in West Africa, may nonetheless have been seriously deficient in some nutrients.

**DIET**

Our technique for constructing the Caribbean slave diet reflected in Table 1 has been to assign to slaves the amount of meat and cereal that planters claimed that they issued—the kind of standardized *ideal* allotment which is mentioned over and over again in everything from instructions to overseers, to travel accounts, to tracts on slave care—and then "build up" that basic allotment with the most commonly mentioned and readily available or easy to store supplements to reach a caloric intake of about 3,000 calories daily. Three thousand calories would be too low for a young male laboring during a sugar harvest; 3,200 to 4,000 calories would be closer to his requirement for this fraction of the year. However, not all islands employed a majority of their slaves in sugar cultivation, and female requirements run on the average about 1,200 calories fewer than males. Thus 3,000 calories seems a reasonable intake for the average adult slave for at least most of the year.\(^7\)

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By employing this technique, we may assume that, ideally, Caribbean slaves received an allotment of a little less than a half pound of animal protein daily, either as dried beef or salted fish, and about a pint of cereal in the form of either cornmeal or rice. This core allotment would have provided in the neighborhood of a third of the daily calorie requirements but about twice as much protein as today’s recommendations suggest.\(^8\)

Because of a lack of calories provided by the core, Caribbean slaves were dependent upon supplements to that core, which explains the importance of their provision grounds and vegetable gardens often mentioned in the literature on Caribbean plantations. Although West Indian plants and dietary preferences varied from place to place, yams, taro, plantains, and bananas are the most frequently cited supplements. The usual practice was to boil all of these foods (save the bananas) with the ration of animal protein and cereal; and, assuming that out of the cooking pot the average slave plucked a half pound of yam, another of taro root, and perhaps a pound of plantains, he would have satisfied his caloric needs for the day.\(^9\)

Surprisingly, despite the usual description of these basic diets as “starchy” and “protein poor,” they seem to have supplied most of the basic nutrients. The diets were poor in calcium, and with milk—the one food which might have remedied the problem—in

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\(^8\) The fish or meat ration in particular varied erratically from the ideal. For example, slave laws in the Leeward Islands required planters to issue a slave 1¼ lbs. of salt fish weekly. Elsa V. Goveia, *Slave Society in the British Leeward Islands at the End of the Eighteenth Century* (New Haven, 1965), 193. Barbadian planters, however, claimed that they issued a pound of fish daily. A Report on a Committee of the Council of Barbados, Appointed to Inquire into the Actual Condition of the Slaves (London, 1824), 106, 113. Yet Jerome S. Handler and Frederick W. Lange in *Plantation Slavery in Barbados* (Cambridge, 1977), 87, an investigation on the Newton plantation, found that its slaves received only one-half pound of salt fish every two weeks, although for the island as a whole the norm was about one pound weekly. Richard N. Bean, “Food Imports into the British West Indies: 1680–1845,” *Journal of the New York Academy of Sciences*, CCXCVII (1977), 581–590, also found the average to be “just a bit over one pound of preserved fish per slave per week” (587). For Cuba, Fraginals, “Africa in Cuba,” 198, states that “the daily norm was some 200 grams [about one-half pound] of jerked beef.” The point is that by accepting planters’ claims of about one-half pound of fish or meat daily, we are doubtless erring on the high side.

short supply and most slaves lactose intolerant, a widespread calcium deficiency must have been a nutritional fact of life. Caribbean slaves seem also to have been slightly deficient in vitamin A, but, superficially at least, the slave diets of the Caribbean might be characterized as not that poor by eighteenth and nineteenth-century standards.¹⁰

A closer look, however, reveals some serious problems, in part because of the chemical composition of some of the foods in question and in part because of the peculiar relationships among some of the nutrients. For example, an enormously complicating factor is the absence of sufficient fats. In both the beef and fish slave diets the animal protein and cereal were the only items to supply any amount of fat. However, because of the low fat content of dried fish and jerked beef, this meant only 20 grams of fat daily for West Indian slaves on a beef-corn core, and a measly 3 grams for those on a fish-rice core. Yet the established world standard for fat intake suggests 80–125 grams as a safe minimum.¹¹

Although the low fat intake of slaves may have been good for cholesterol levels, such a diet means that the amount of vitamin A that Table 1 shows West Indian bondsmen as receiving is considerably overstated. Vitamin A is fat-soluble; hence a low fat diet impairs the ability of the body to absorb that vitamin. Moreover, the fish or meat allotment to slaves was usually reported as rancid; indeed, slaves allegedly preferred it that way. Unfortunately rancidity has a destructive effect on fat-soluble vitamins. Thus, instead of being only mildly vitamin A deficient, many West Indian slaves, because of an absence of dietary fat and the rancidity of much of the fat that they did ingest, must have been severely vitamin A deficient.¹²

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Less well known is the relationship between fat and thiamine or vitamin B₁. Because thiamine is part of the water soluble vitamin B complex, fat has little to do with its absorption. But in the case of low fat diets, such as those of Caribbean slaves, carbohydrates replace fat as the major energy source, and carbohydrates require thiamine for metabolism. Thus the low fat/high carbohydrate content of the Caribbean slave diets would have greatly accelerated thiamine requirements. For West Indian slaves thiamine requirements would have been higher than for the whites on the islands whose diets were heavily fat-laden. But even more to the point, it is highly doubtful that Caribbean bondsmen actually received as much thiamine as is suggested by Table 1.¹³

The process of pickling, salting, and drying beef or fish treats thiamine poorly. Both alkaline solutions and prolonged dehydration have a destructive effect on thiamine, the least stable of the B complex vitamins. This lack of stability also means that heat is more destructive to thiamine than to riboflavin or niacin. Thus data on the thiamine content of foods are usually given, as they are in Table 1, before cooking because of the losses which occur in the process. The loss from cornmeal, for example, runs between 15 and 25 percent. In the case of meat, however, that loss can be as high as 85 percent.¹⁴

Moreover, because thiamine is so highly soluble in water, it is readily leached out of the food during boiling, the standard method of cooking on most West Indian plantations. Fish or beef was tossed into the family or communal pot where it simmered all day along with yams, plantains, taro root, and, quite possibly, the cereal ration as well. Because of such factors as dehydration


and cooking losses, the thiamine delivered by the West Indian slave diet is probably overstated by at least 50 percent. Those slaves especially whose cereal allotment was rice were bound to be seriously $B_1$ deficient because, when rice is subjected to a polishing process to retard spoilage, the process also strips away the thiamine-rich husk of the grain.  

Finally, it bears repeating that the low fat diet of Caribbean slaves would have severely exacerbated a condition of thiamine deficiency by elevating thiamine requirements. To this factor should be added two more exacerbating difficulties: a diet high in carbohydrates also accelerates thiamine requirements; and thiamine is the most poorly stored of all of the $B$ vitamins.

Not all Caribbean bondsmen suffered from deficiencies of calcium, vitamin A and thiamine. Red peppers provided much in the way of vitamin A, as did mangoes and ackee, both of which came into widespread West Indian use at about the turn of the nineteenth century. Milk was not totally unavailable to Caribbean slaves and, although most blacks were lactose intolerant, a few ounces in cornbread or coffee would have made a nutritional contribution—especially in the areas of sorely needed tryptophan and calcium—without necessarily producing symptoms of lactose intolerance.

DISEASE Not all slaves had access to a variety of supplementary comestibles, and many had access to very few. Even assuming that slave diets were marginally, rather than severely, deficient in one or another vitamin, economic, political, and climatic circumstances could quickly have changed those diets for the worse. The English-French global struggle frequently deprived West Indian slaves of their meat and cereal core diet, as did the end of North American imports to the British West Indies after the American Revolution. Hurricanes were always destructive to slave provision


16 Lloyd, McDonald, and Crampton, *Fundamentals of Nutrition*, 163.

grounds, particularly to the fragile plantain and banana trees, and high prices for meat and cereal or low prices for sugar reduced supplies for slave rations.¹⁸

Even during good times Caribbean slave diets were badly out of balance, and times were often far from good. Yet all that a nutritional analysis of the basic foodstuffs in the slave regimen has done is to create the suspicion of widespread deficiencies of vitamins A and B₁. That suspicion can only be transformed into something more concrete if the diseases triggered by these deficiencies can be found to have afflicted slaves in large numbers. A correlation could then be established between a nutritional deficiency and a deficiency disease.

In any search for a deficiency disease one is quickly confronted with the phenomenon of so-called “Negro diseases,” given much prominence by West Indian physicians of the period. These were diseases from which blacks were far more likely to suffer than whites. Yaws, for example, was reportedly an affliction with an enormous color prejudice. The prejudice, however, is easily explicable in terms of a slave trade which constantly introduced persons infected in West Africa. Because of skin-to-skin transmission, the disease flourished among a people who lived in close contact and wore few clothes. Another such disease—a major killer of infants—was the “jawfull,” or neonatal tetanus. The affliction singled out black infants for special grim attention because of the frequency of umbilical stump infections. The unsanitary condition of many slave quarters, on the one hand, and West African practices such as packing the stump with mud, on the other, guaranteed a high black as opposed to white death rate from the disease. There are, however, other Negro diseases the etiologies of which are not so easily understood in terms of pathogenic discrimination, but the symptoms of which do suggest problems of nutrition.¹⁹


Nutritional analysis has suggested that many Caribbean slaves would have been vitamin A and thiamine deficient, both because the diet itself was low in these vitamins and because a low fat diet meant that much vitamin A was not absorbed and that thiamine requirements were elevated. Considering vitamin A first, one discovers among those peculiarly "Negro afflictions" which occupied the attention of Caribbean slave physicians the malady "sore eyes." Eye afflictions were so widespread among the slaves that whole chapters in books on slave medicine were devoted to the problem, which is characteristic of several nutrient deficiencies. Night blindness, described as a "disease which is so frequently seen among Negroes," is prominently mentioned in those chapters, and is a classic sign of vitamin A deficiency. Its prevalence among the slaves, along with the high incidence of sore eyes, does much to strengthen the suspicion born of nutritional analysis that many Caribbean bondsmen were vitamin A deficient.\footnote{See William Hillary, \textit{Observations on the Changes of the Air and the Concomitant Epidemical Diseases, in the Island of Barbados} (London, 1811; 2nd ed.), 297–304, for a discussion of nyctalopia. See also Collins, \textit{Practical Rules}, 287; James Grainger, \textit{Essay on the More Common West Indian Diseases} (London, 1807; 2nd ed.), 60.}

Another sizable body of literature amassed by Caribbean physicians concerns one of the worst curses to befall a West Indian slave—the \textit{mal d'estomach}. Also called \textit{mal de estomágo}, \textit{hati-weri}, \textit{cachexia africana}, and just plain dirt-eating, the disease afflicted the black population of the West Indies exclusively. In Puerto Rico it was reportedly one of the two worst diseases of the slaves. In Jamaica it was portrayed as "common upon almost every plantation" and on some estates the cause of about half of the deaths. At first, doctors were powerless against it, and iron masks were used to break the pica habit. However, by the last decades of the eighteenth century, physicians were nearly unanimous in their prescribed cure—a better, more balanced diet.\footnote{Bengt Anell and Sture Lagercrantz, \textit{Geographical Customs} (Uppsala, 1958), 60; John Stewart, \textit{An Account of Jamaica} (London, 1808), 273; Abbad y Lasierra, \textit{Historia de Puerto Rico}, 207; John Imray, "Observations on the Mal d'estomach or Cachexia Africana, as it Takes place among the Negroes of Dominica," \textit{Edinburgh Medical and Surgical Journal}, CIX (1843), 314; John Williamson, \textit{Medical and Miscellaneous Observations Relative to the West India Islands} (Edinburgh, 1817), I, 177–182; II, 267; Edwards, \textit{History of British in West Indies}, II, 167; Collins, \textit{Practical Rules}, 274.}

This mysterious malady, as described by eighteenth-century
physicians, made its victims “languid and listless,” “short breathed,” and “giddy,” and afflicted them with “palpitations of the heart” and “loss of appetite.” With the progression of the sickness, legs swelled, the countenance became bloated and “dropsy ensued.” These symptoms are a classic portrayal of beriberi advancing from the dry to wet stage—and beriberi is caused by thiamine deficiency.22

In addition to these outstanding symptoms of thiamine deficiency, many other reasons exist for suspecting that mal d’estomach was in fact beriberi. Pregnant and lactating females were reported as the most susceptible to the disease, and pregnant and lactating females have historically proven the most vulnerable to beriberi because of accelerated requirements for most nutrients, including the B complex. Also vulnerable were young girls who suffered a disproportionately heavy incidence of the disease “at a certain time in their life . . . [just] before their periodical evacuations appear.” The females in question, who probably consumed less food than their brothers of the same age, would have been experiencing the period of growth when requirements for all the B vitamins accelerate. The children susceptible to mal d’estomach were depicted as nutritionally deprived and often rickety in appearance. Whether the disease struck at the young or old, male or female, the remedy that physicians prescribed was “wholesome food”; that the cure worked does nothing to weaken the hypothesis that the disease was indeed beriberi.23

There are other explanations for the symptoms. Slaves who manifested dirt-eating symptoms were sometimes thought to be attempting suicide, and slaves who developed mal d’estomach often personally diagnosed their problem as the result of having been poisoned or cursed by an “obeah man.” More recently mal d’estomach has been pronounced to be the result of ankylostomiasis or hookworm infection, and indeed dirt-eating among slaves in the United States has also been attributed to hookworm. Yet this


explanation ignores the relative immunity that blacks have to hookworm "expressed as a resistance both to invasion by the parasite and to the injurious effects after invasion." Another problem with hookworm infection as the culprit is that the disease was not unknown to colonial physicians; rather it was commonly diagnosed in British troops and thus presumably would have been recognized in blacks had they manifested similar symptoms.\(^{24}\)

Others have confused the *mal d'estomach* with the dry belly ache, which was also frequently fatal. This affliction, however, was accompanied by enormous intestinal pains that often had individuals begging to be shot or otherwise put out of their misery and which terminated in convulsions or epileptic seizures. Benjamin Franklin became interested in the problem and speculated in a letter to a West Indian physician that the dry belly ache was the result of drinking rum distilled in apparatuses using lead fastenings and pipes. Franklin's speculation may have been correct, for other physicians observed that the dry belly ache often caused lead poisoning. *Mal d'estomach* and dry belly ache were clearly two separate afflictions, with only the former, according to contemporary doctors, having a nutritional etiology.\(^{25}\)

Evidence of a general B vitamin deficiency in the West Indies is contained in a seasonal phenomenon which occurred at crop time on sugar plantations; despite the long hours of extraordinarily hard labor, blacks paradoxically enjoyed better health throughout the harvest. Physicians commented on the "peculiar glossiness of the skin, so indicative of health [which] is never seen to the same extent at any other season." Moreover, it was accepted wisdom that slaves purchased during crop time would do better in terms of health than slaves purchased at any other time of the year. Physicians and planters attributed this condition to the drinking of sugar cane juice. Custom allowed slaves to drink as much as they wished of the "hot liquor" from the "last copper," which contained a mixture of brown sugar and molasses. Thus

\(^{24}\) This "very pronounced" black resistance to hookworm infection was discovered by investigators in the American South during the early decades of this century. See for example, A. E. Keller, W. S. Leathers, and H. C. Ricks, "An Investigation of the Incidence and Intensity of Infestation of Hookworm in Mississippi," *American Journal of Hygiene*, XIX (1934), 629–656.

slaves who drank from the “last copper” were imbibing a liquid rich in iron and the B vitamins. If island physicians were not certain why, they nonetheless knew that this annual infusion of minerals and vitamins was beneficial for slaves.26

Physicians also (significantly in terms of thiamine deficiency) periodically suspected rice of producing bad health. A Jamaican physician reported early in the nineteenth century that rice had lately “fallen into disuse” because it caused “dropsical swellings.” Edema can be symptomatic of many disorders but, because they were linked to rice consumption, the “dropsical swellings” do suggest beriberi, and dropsy had the reputation of being a major killer of adult Caribbean slaves.27

Mortality records bear out dropsy’s deadliness; in those consulted for Barbados, dropsy dominated as a cause of plantation deaths. In Jamaica, on the Worthy Park estate between 1811 and 1834, dropsy accounted for fully 10 percent of the 222 deaths, ranking only behind deaths from old age (53) and fever (23); of 357 slave deaths in St. James Parish, Jamaica, between 1817 and 1820, dropsy accounted for 11 percent of the deaths, second only to old age. If dirt-eating and mal d’estomach deaths are added to those from dropsy, the three accounted for 16 percent of the deaths. Dropsy alone claimed 11 percent of the 288 deaths on three other Jamaican estates from 1817 to 1829. If other diseases with symptoms suggestive of beriberi, such as “fits,” “convulsions,” and “bloating,” are included with dropsy, the diseases in question accounted for 22 percent of the deaths registered.28

On the Newton plantation in Barbados from 1796 to 1801 and from 1811 to 1825, dropsy accounted for 9 percent of the 153 deaths (14), tying with consumption for third place behind old


28 Craton and Walvin, A Jamaica Plantation, 113, 197–198; Higman, Slave Population, 112. Deaths were substantially understated because of a failure to report infant deaths.
age (22) and “no cause given” (19). By race the death records for the dioceses of Havana for 1843 recorded a black death rate from anaemia (general dropsy) of about three times that for whites. Finally, slave death statistics compiled by a West Indian physician during the 1820s for a Jamaican parish credits dropsy, plus other beriberi-like afflictions, with fully 20 percent of the victims aged over one year of age in the district in question.29

As with mal d’estomach and “cachexias,” there was a cure for dropsy. In the words of Long, “Sometimes they [the slaves] fall into dropsies, which generally prove mortal; for this disorder requires a very nutritious diet.” In the minds of white planters good nutrition was equated with fresh meat, and fresh meat contains that thiamine which could have cured the “dropsies.”30

FERTILITY AND INFANT MORTALITY Beriberi is one of those nutritional diseases, like pellagra, which escaped identification as a disease sui generis for many years, largely because its protean symptoms misled physicians into thinking that they were confronting a number of diseases. Because it was in the Far East that beriberi was finally identified, it subsequently was associated with rice-eating cultures; hence, beriberi is not usually thought of in a Caribbean context. Yet in 1865, early in the effort to conquer beriberi, Hava reported that the disease was epidemic among blacks on Cuban plantations and described all of the symptoms of wet beriberi. In 1871 a French physician observed the disease on Cuban ingenios (plantations) and attempted, not very successfully, to treat it with arsenic. Finally in 1873 the disease was reported as raging with virulence on some Cuban plantations, causing a mortality rate between 60 and 75 percent. Because the slave diet in Cuba could and did produce beriberi, there are grounds for the suspicion that the disease was fairly widespread among West Indian slaves and that many of those who died from mal d’estomach, dropsy, or convulsions were actually dying of beriberi.31

31 Juan G. Hava, “Communicacion Dirigida a la Academia sobre una Epidemia de Beriberi,” Academia de Ciencias Medical de la Habana. Anales, II (1865), 160–161; J. Min-
If these deaths were caused by beriberi, then the dietary deficiency which produced this disease may have significantly altered the demographic history of West Indian slave populations—not so much because of the adult deaths, but because of infant mortality. For all the major nutritional diseases, only beriberi is a killer of otherwise normal infants receiving an adequate supply of breast milk. Adult slaves in the United States, for example, suffered from pellagra caused by niacin deficiency. Yet it is almost impossible for infants to be niacin deficient, because human milk supplies an adequate amount of both niacin and tryptophan (niacin's precursor), even if the mother is niacin deficient. However, a mother deficient in thiamine will invariably have milk deficient in that vitamin. To complicate matters for medical personnel, she may show few or even no signs of thiamine deficiency herself; in other words, a mother whose child develops infantile beriberi may not display overt signs of the malady.32

Infantile beriberi symptoms are very different from adult symptoms, and edema is only occasionally seen. The disease begins with vomiting, pallor, restlessness, loss of appetite, and insomnia and terminates life with convulsions and/or cardiac failure. Clearly the variety of symptoms makes it difficult to pin down the disease on West Indian plantations of yesterday. But if beriberi were fairly widespread, then infantile beriberi had unquestionably to be a major destroyer of West Indian slave infants. How major a destroyer may be gauged by looking at the Philippines, where beriberi has been and still is a chronic problem; in the late 1950s between 75 and 85 percent of the 25,000 beriberi deaths reported there annually were infants. By the turn-of-the-century, nearly half of all infants born alive in Manila failed to reach one year of age; infantile beriberi bears much of the blame for this mortality.33

How major a killer beriberi was in the West Indies can only

be speculated upon. The disease usually strikes infants between the first and sixth months of life, before supplements have been added to their diets. If the attack is acute, the infant has difficulty in breathing, becomes cyanosed, and dies of cardiac failure with "unnerving rapidity." If it is chronic, the infant grows thin and wasted, edema occasionally occurs, and convulsions are frequently seen in the terminal stages.34

To be sure, the infant slave had to live long enough to contract infantile beriberi by first escaping that primary destroyer of the newborn in the Caribbean—neonatal tetanus. The fearsome reputation of this affliction was well deserved; indeed, the ailment has been credited with carrying off about one quarter of all slave infants within their first two weeks of life. However, as pointed out by students of Caribbean slave mortality, "it is evident that the majority of fatalities among slave children born at Worthy Park occurred not at birth but in the children's early years."35

A consultation of the literature produced by West Indian physicians and of the mortality data suggests that the latter statement applies not only to Worthy Park but to the Caribbean slave population as a whole. Rivaling neonatal tetanus as major causes of infant deaths were marasmus, convulsions, and tetanic convulsions. Researchers today looking for evidence of beriberi's presence in a region scrutinize any death that is classified under the rubric of convulsions or marasmus. Convulsions were also associated with teething difficulties, yet infants do not convulse simply because of teething. However, the months of teething are also those months during which infants would be most likely to succumb to beriberi.36

Another signal to researchers is the phenomenon of mothers with a history of losing one baby after another within the first few months of life. The West Indian literature offers many ex-

34 Davidson et al., *Human Nutrition*, 415; Latham et al., *Scope on Nutrition*, 39.
35 Craton and Walvin, *A Jamaican Plantation*, 134. See also the parochial records for Barbados: the parish of St. Thomas, for example, buried 168 slave children aged ten and under for the period 1816-1834. Only 36 of the deaths were one year of age or less. In St. Phillips parish, 33 of their 106 burials were infants. The records for other parishes are incomplete, yet what data are available continue to suggest that only about one third of those aged ten and under of those slave children who died were infants aged one or less.
amples of mothers who had produced fifteen children and lost all but two, or who "had borne ten children, and yet has now but one alive," or "the instances of those who have had four, five, six children, without succeeding in bringing up one in spite of the utmost attention and indulgence . . . ." 37

Further analysis of mortality data and plantation records is needed. But this study does show that there is a correlation between a thiamine deficiency in the West Indian slave diet and diseases with beriberi-like symptoms, which ranked among the most important causes of slave mortality. If beriberi were as widespread as the evidence gathered thus far suggests, then thiamine deficiency must join with other factors produced by the slave trade and sugar monoculture to explain why Caribbean slave populations did not sustain themselves by natural means as did the slave population of the United States. It has often been urged that low fertility was at fault. Yet none of the islands have adequate birth records either to confirm or refute this suggestion. A possible measure of fertility relates the number of children under one year to the number of women able to bear those children. But if something is killing those infants at a brisk rate, then this kind of fertility ratio is a very misleading statistic.

Finally, it has been suggested that low fertility may have been partially the result of the practice of West African mothers to nurse their children for periods as long as three years. West Indian planters tried to discourage prolonged nursing but were not so successful in combating West African cultural practices as were United States' planters, who were dealing with a people much further removed in time from their homeland. United States' slaves tended to nurse their babies for a year or less. Although the ability of lactation to prevent pregnancy after a few months of nursing is in doubt, there seems no question that prolonged lactation substantially increased the risk of death by thiamine deficiency for many West Indian slave infants. 38

37 M. G. Lewis, Journal of a West Indian Proprietor, 1815–1817 (London, 1929), 97, 111.
Although the slave diets of both the United States and the West Indies contained the potential for precipitating a B vitamin deficiency disease, pellagra, the deficiency disease of United States slaves would not have killed the very young. But beriberi, the disease of Caribbean slaves, most certainly would have. The result may well help to account for the "astounding fact, that while the blacks in the United States have increased tenfold, those of the British West Indies [and the West Indies] generally have decreased in the proportion of five to two." 39

Fertility, Nutrition, and Pellagra: Italy during the Vital Revolution

During the last decades of the nineteenth century, fertility began its secular decline in several regions of the north and center of Italy, at a faster rate in the urban and semi-urban areas and more slowly elsewhere. Fertility control spread to other regions in the area and, with the return to normalcy after World War I, the process became firmly rooted. The story has been written elsewhere and does not need to be told here.

There was, however, an interesting exception to this classical development: Veneto, a large region with some populous adjoining areas, did not follow the course of the other northern regions. At the beginning of this century it saw a significant increase in marital fertility despite the contemporary diffusion of contraception in the urban centers and, quite probably, in some non-urban social strata. In this same area, in the second half of the nineteenth century, the gradual deterioration of the peasants’ diet had brought to its height the incidence of pellagra, a chronic disease with seasonal relapses caused by a dietary deficiency of niacin, called also PP vitamin (Pellagra-Preventive) of the B group. By the beginning of this century, the general improvement in the economic condition of the rural population and in its nutritional standard rapidly reduced the incidence of the disease while, at the same time, fertility rose.¹

Before proceeding further, something more must be said about the etiology of pellagra, with its symptoms of weakness, apathy, loss of appetite, and neurasthenia. In later stages of the disease appear the classic three D’s: dermatitis, diarrhea, and dementia, frequently followed by a fourth one: death. Dermatitis

¹ For an analysis of the history of fertility in Italy, see Livi-Bacci, A History of Italian Fertility during the Last Two Centuries (Princeton, 1977).
begins with an erythema as in a sunburn; lesions then appear on various parts of the body—the hands, wrists, elbows, under the breasts, and on the knees and feet—and particularly in parts of the body exposed to the sun. The digestive system is affected with anorexia, dyspepsia, vomiting, and, in the acute stage, watery diarrhea. Psychic symptoms are present at all stages of the sickness, and they become acute in the final stages: insomnia, dizziness, and irritability at first; neurasthenia, confusion, and hallucinations in the final stage. Death inevitably follows unless intensive treatment is administered.  

As said above, a niacin deficient diet is a root cause of pellagra. Niacin is a common B group vitamin present in some foods either in low quantities or in quantities that cannot be absorbed. In general, a diet based on maize—polenta and its varieties in Italy—is poor in niacin. With little or no supplementation of milk, meat, and vegetables, it was at the base of the disease. The diffusion of pellagra in Europe followed the diffusion of the cultivation of maize and the popularization of its consumption.

The hypothesis that changes in fertility around the turn of the century were associated with the rise and fall of pellagra, itself a consequence of the changing nutritional and dietary patterns of the population, is advanced in this article, raising again the much-debated nutrition fertility issue. That malnutrition can impair the functions of the human reproductive process, particularly during famine and starvation, is an accepted fact; opinions diverge much more on the evaluation of the dimensions of the fertility depressing effects of chronic malnutrition.

Bongaarts, in a review of the evidence drawn from contemporary populations, concludes that "moderate chronic malnutrition has only a minor effect on fecundity, and the resulting decrease in fertility is very small." Among the components of fecundity, menarche and the duration of postpartum amenorrhea appear to be most affected, whereas the evidence concerning other components, such as age at menopause, permanent sterility, regularity of ovulation, and intrauterine death, appear insufficient or contradictory. Menken, Trussell, and Watkins affirm that "the

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Table 1  Demographic Indicators for Veneto, 1871–1931

<table>
<thead>
<tr>
<th>YEAR</th>
<th>POPULATION</th>
<th>BIRTH RATE</th>
<th>DEATH RATE</th>
<th>RATE OF NATURAL INCREASE</th>
<th>INFANT MORTALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1871</td>
<td>2643</td>
<td>37.6</td>
<td>28.3</td>
<td>9.3</td>
<td>237.0</td>
</tr>
<tr>
<td>1881</td>
<td>2814</td>
<td>34.4</td>
<td>26.0</td>
<td>8.4</td>
<td>200.7</td>
</tr>
<tr>
<td>1891</td>
<td>2970</td>
<td>35.1</td>
<td>22.4</td>
<td>12.7</td>
<td>180.8</td>
</tr>
<tr>
<td>1901</td>
<td>3134</td>
<td>36.9</td>
<td>20.0</td>
<td>16.9</td>
<td>151.6</td>
</tr>
<tr>
<td>1911</td>
<td>3527</td>
<td>37.0</td>
<td>18.3</td>
<td>18.7</td>
<td>133.8</td>
</tr>
<tr>
<td>1921</td>
<td>3957</td>
<td>33.3</td>
<td>15.9</td>
<td>17.4</td>
<td>116.0</td>
</tr>
<tr>
<td>1931</td>
<td>4123</td>
<td>26.2</td>
<td>12.5</td>
<td>13.7</td>
<td>87.5</td>
</tr>
</tbody>
</table>

Note: Rates are computed on the basis of three-year averages of events centered on the census date.

Infant mortality. The trend of the birth rate, showing a slight increase at the beginning of the century, is worth investigating more closely. Unfortunately, birth statistics by age of mother and birth order are not available before 1929 and therefore the analysis of fertility must rely on aggregate indicators. However, the standardized indices of general, marital, and non-marital fertility are, at this stage, adequate to investigate the trends of fertility; these measures are supplemented by a few indicators of nuptiality (Table 2).

$I_f$, $I_g$, $I_h$, and $I_m$ are the standardized indices used throughout the Princeton European Fertility Project and do not need extensive description here. $I_g$ is the corrected index of marital fertility used throughout the Italian study. It is defined as $I_g$ multiplied by the ratio of married females to married males, in order to eliminate the effects of an unbalanced sex ratio in the reproductive ages due to male emigration. Both $I_g$ and $I_f$ show evidence of an increase in marital fertility before World War I: the rise of $I_g$ from the minimum (1881) to the maximum (1911) is 18.4 percent; the increase of the mean level of $I_f$ between 1871 and 1881 and between 1901 and 1911 is less but still very significant at 14.4 percent. Even after the end of the war, Veneto’s marital fertility was higher than in the latter part of the nineteenth century, and this in spite of birth control being well established in the large urban centers. The contrast with the change of $I_g$ in the other Italian regions is noteworthy: in Lombardia and Emilia, which
<table>
<thead>
<tr>
<th>Year</th>
<th>Index of General Fertility</th>
<th>Index of Marital Fertility</th>
<th>Index of the Proportion Married (lm)</th>
<th>Percent Females Single, Age 50–54</th>
<th>Mean Age at First Marriage (Females)</th>
<th>Index of Illegitimate Fertility</th>
</tr>
</thead>
<tbody>
<tr>
<td>1871</td>
<td>.412</td>
<td>.682</td>
<td>.586</td>
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<td>24.7</td>
<td>.028</td>
</tr>
<tr>
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<td>.379</td>
<td>.651</td>
<td>.546</td>
<td>10.0</td>
<td>24.4</td>
<td>.051</td>
</tr>
<tr>
<td>1891</td>
<td>.400</td>
<td>.698</td>
<td>.540</td>
<td>..</td>
<td>..</td>
<td>..</td>
</tr>
<tr>
<td>1901</td>
<td>.419</td>
<td>.743</td>
<td>.534</td>
<td>10.2</td>
<td>23.8</td>
<td>..</td>
</tr>
<tr>
<td>1911</td>
<td>.400</td>
<td>.738</td>
<td>.512</td>
<td>10.5</td>
<td>23.6</td>
<td>.049</td>
</tr>
<tr>
<td>1921</td>
<td>.348</td>
<td>.704</td>
<td>.457</td>
<td>11.3</td>
<td>24.3</td>
<td>.046</td>
</tr>
<tr>
<td>1931</td>
<td>.265</td>
<td>.527</td>
<td>.470</td>
<td>11.8</td>
<td>24.2</td>
<td>.033</td>
</tr>
</tbody>
</table>

Note: Indexes of fertility and nuptiality have been computed on the basis of three-year averages of events centered on the census date. For 1921 the average of 1921–26 has been considered and the census population has been reported at the end of 1923.
shared some of the nutritional patterns of Veneto, there was a slight increase of 2 to 3 percent between 1871 and 1881 and between 1901 and 1911; in the other regions of the center and of the north there was a fall of 11 percent; in the south there was no change at all. It is only at the end of the 1920s that natural fertility patterns were rapidly abandoned by the population of Veneto, some half a century after Liguria and Toscana and considerably later than in other areas of northern Italy.6

The other indicators of fertility, $I_f$ and $I_h$, show a remarkable stability, whereas the measures of nuptiality indicate that no major revolution took place during the period under consideration. Nuptiality is not independent of marital fertility, particularly when its level is high and contraception is absent. However, there are no changes in nuptiality that can account, even partially, for the changes in marital fertility; the mean age at first marriage for women fluctuated around twenty-four years and the proportion remaining single between the ages of fifty and fifty-four among women experienced only a slight increase. The 13 percent decline of $I_m$ between 1871 and 1911 was largely due to the numerical imbalance between males and females caused by selective migration of men; a decline of a similar order of magnitude (10 percent) can be observed in the other regions of the north.

The rise in marital fertility is real and not a statistical artifact. Birth registration was virtually complete. Census coverage and quality improved over time but certainly cannot be held responsible for false indications of an increase in fertility rates. Emigration certainly had a depressive effect on fertility, an effect that is in part eliminated by the calculation of $I_g$; on no account should it be invoked to explain the rise in fertility. Indeed both seasonal and, particularly, permanent emigration increased at the end of the nineteenth and at the beginning of the twentieth century, causing an alteration in the demographic and social structure before World War I with a negative impact on fertility. Yet $I_g$ and $I_g$ reached their maximum during those years. Other standard demographic factors cannot be invoked to explain the rise in

6 Livi-Bacci, *Italian Fertility*, 56, 81–83. The general marital fertility rate in Veneto was 284.5 per 1,000 in 1901 for the small *comuni* with less than 30,000 inhabitants; 222.5 per 1,000 for the *comuni* with 30,000 to 100,000 inhabitants; 186.5 per 1,000 for Venezia, the only *comune* with more than 100,000 inhabitants. *Ibid.*, 122. (The *comune* is the smallest administrative unit.)
ments of France...it was also apparent in Denmark, England and Wales, Germany and some regions of Italy.”

There are many reasons why the rise in fertility has passed unnoticed. Since this rise occurred mainly during the nineteenth century, observers and scholars have been interested, above all, in detecting a pattern in the fall of marital fertility as an indicator of spreading birth control and of demographic modernization. The rise in fertility was often attributed to the poor quality of the data; to an improved completeness of registration; and to random fluctuations in the factors of natural fertility. Seldom has such a rise been recognized as a real change in one or more of the components of natural fertility.

Of great interest is a recent article by Knodel and Wilson. They examined the reproductive histories of couples in fourteen German villages married between 1750 and 1899. The absence of change in overall indicators of natural fertility (such as $I_g$) is deceptive because the decline in marital fertility rates at older ages is compensated for by an increase at the younger ages and therefore “the earlier onset of voluntary fertility control was masked in the measures of observed fertility by a substantial and concurrent rise in the underlying level of fertility.” The underlying level of marital fertility is measured by the parameter $M$ of the Coale and Trussel equation. When the separate components of fecundity are examined, the authors find firm evidence of a rise in fecundability—the probability of conceiving; less conclusive evidence of a decline in the non-susceptible period following birth; and little or no change in primary sterility. As to the causes of the rise in fecundity, the authors advance a few tentative hypotheses: a decline in intrauterine mortality; improved nutrition or changes in infant feeding practices; or a combination of these factors.

POPULARIZATION OF MAIZE AND THE APPEARANCE OF PELLAGRA
The case of northeastern Italy, unfortunately, cannot be investi-
gated with the same richness of detail available for the fourteen German villages; it is possible, however, to gather some interesting circumstantial evidence on the possible underlying causes for the rise in marital fertility at the beginning of this century. My intention is to prove that there is a connection between fertility and nutrition, and that the history of pellagra is the symptom, if not the proof, of that connection.

The history of pellagra is closely related to the history of maize in Spain, France, Italy, and in several areas of Eastern Europe. Maize was already known in Veneto in the sixteenth century, and its diffusion increased in the seventeenth, spreading to Lombardia. The popularization of corn in Romagna (part of the Emilia region) took place in the latter half of the eighteenth century, when it was still relatively rare in Toscana. From the end of the eighteenth century the cultivation and consumption of corn rapidly increased in the north and in the center of the country. The worsened living conditions of the rural populations at the end of the eighteenth century, the consequences of the Napoleonic Wars, and the profound subsistence crisis of 1816/17 were all powerful incentives to the cultivation of maize. Its high productivity and nutritional value and its relatively low price made it particularly attractive to the impoverished peasants. Maize became increasingly popular in the form of bread and, particularly, polenta, becoming practically the exclusive food of the very poor. In the years of poverty, when the price of wheat rose, maize became its universal substitute; diets already poor in nutritional value were further impoverished and imbalanced, and pellagra increased. Many writers have emphasized the fact that the modernization of agriculture in the lowlands of northern Italy was responsible for the proletarization of a large part of the rural population and for a downgrading of their nutritional patterns. Maize was grown on the farm mainly for direct consumption, whereas the other products were sold on the market.\(^{12}\)

\(^{12}\) On the history of the cultivation of maize in Italy and of its role in nutrition, see L. Messedaglia, *Il mais e la vita rurale in Italia* (Piacenza, 1927). See also, *idem, Per la storia dell’agricoltura e dell’alimentazione* (Piacenza, 1932). One of the many examples of fluctuations of pellagra is its increase in Veneto in 1880 after the scarce harvest of 1879. See Emilio Morpurgo, “Relazione sulla XI Circoscrizione,” in *Atti della Giunta per l’inchiesta agraria* (Rome, 1882), IV. On the living conditions of the rural population, see Giorgio Porisini, “Agricoltura, alimentazione e condizioni sanitarie. Prime ricerche sulla pellagra in Italia dal 1880 al 1940,” unpub. ms. (Bologna, n.d.).
The increasing presence of corn in the daily diet was accompanied, in the area of investigation, by general deterioration in real income and in nutritional patterns in the 1860s and 1870s; the monumental parliamentary *Inchiesta Agraria* carried out in the early 1880s leaves no doubt about this decline. The agrarian crisis of the 1880s, triggered by the devastating competition from North American products, added new difficulties to a situation of grave poverty and widespread destitution. Only at the end of the century was there a general improvement in the living conditions of the rural population, with a sustained trend until World War I. Official surveys and private investigations confirm the corresponding improvement in diet. Official estimates of the per capita daily caloric intake for the entire population show a decrease from 2,647 between 1871 and 1880 to 2,197 from 1881 to 1890 and 2,119 from 1891 to 1900 and a rise to 2,617 from 1901 to 1910; unfortunately the statistics on which these estimates are based leave much to be desired.13

Since maize was mainly consumed where it was grown, and only a small part of the total production was traded even domestically, there was a correspondence between the geography of production and the geography of consumption. In the latter part of last century over half of the total production came from Lombardia, Veneto, and Emilia, and about 80 percent from the seven regions where pellagra was present. With the beginning of the new century an increase in the use of maize for cattle feed and in the growth of internal and international trade lessened the correspondence between area of production and area of consumption.14

Pellagra follows the track of maize; it appears in Spain, probably at the end of the seventeenth century, where it was systematically described by Gaspar Casal, an Asturian physician. The

13 The *Giunta per l'Inchiesta Agraria* was a parliamentary commission created to inquire into the conditions of the rural population; it was chaired by Stefano Jacini, an economist and a senator. See *Atti della Giunta per l'Inchiesta Agraria* (Rome, 1881–1884), 15 v. The material on the living conditions of the rural population in the pellagra area is very rich. See particularly *ibid.* (1882), VI, pts. 1 and 2, concerning Lombardia and the general report by Jacini himself; see also *ibid.*, IV, on Veneto. On the agrarian crisis of the 1880s, see Gino Luzzatto, *L'economia italiana dal 1861 al 1914* (Milan, 1963), 218 ff; Mario Romani, *Un secolo di vita agraria in Lombardia* (Milan, 1963), 50. On the improvement of the living conditions at the turn of the century, see Riccardo Bachì, *L'alimentazione e la politica annonzaria in Italia* (Bari, 1926), 18 ff; Romani, *Un secolo*. Statistics on caloric intakes can be found in ISTAT, *Sommaio di statistiche storiche dell'Italia*, 1861–1975 (Rome, 1976), 161.

in small rural communities, several health boards considered the results to be largely an underestimation of reality. The reasons were various: in some areas there were no medical personnel; pellagra was difficult to detect in the early stages; and there was a widespread reticence to make manifest a disease which was known to undermine the working efficiency of the individual. In short, the surveys underestimated the number of pellagrins on the one hand while, on the other, the criteria followed in estimating their number varied considerably from area to area. In some extreme cases, comuni notoriously afflicted by pellagra were declared immune.\(^\text{17}\)

The second source consists of the cause of death statistics which were initiated in Italy in 1881 in a number of large comuni, comprising one third of the total population, and were extended to the entire country in 1887. Cause of death statistics, particularly in the first years, were subject to criticism and they illustrate only the tip of the iceberg as far as the disease is concerned.\(^\text{18}\)

The first surveys—with their biases and distortions—were taken at the zenith of the epidemic. Indeed, some early surveys taken in Lombardia claimed 20,282 pellagrins in 1839 and 38,777 in 1856, numbers that grew to 40,838 in 1879. In 1870, Balardini gave a total count of 14,502 pellagrins in the province of Brescia (which had the highest prevalence in Lombardia), compared with 10,924 in 1856, and 6,939 in 1839. This quantitative evidence, together with abundant documentary evidence of other kinds, indicates that the gravity of the disease in Lombardia reached a maximum in the 1870s. In Veneto the maximum probably occurred in the 1880s: earlier surveys in the mid-1850s claimed some 15,000 to 16,000 pellagrins, little more than one fourth the number in 1881. Between 1881 and 1899, the prevalence of pellagra declined rapidly in Lombardia and Emilia and less rapidly in Veneto; the rise in other regions, where the prevalence always remained relatively low, reflected a further geographical spread

17 The results of the first survey are published in MAIC, “La pellagra.” The results of the second and more careful survey of 1881 can be found in MAIC, “La pellagra in Italia. Provvedimenti e statistica. Pt. II: Statistica dei pellagrosi esistenti in Italia nel biennio 1880–81,” Annali di Agricoltura, XLIV (1885). On the underestimation of pellagrins, see the explicit comments made by the Health Boards of Pavia, Como, Cremona, Rovigo, and Modena in ibid.

18 Direzione Generale di Statistica (DIRSTAT), Statistica delle cause di morte, yearly volumes since 1881.
of the disease (Table 3). In 1910 the prevalence of pellagrins was reduced to little more than one fourth the level of 1881. Later surveys, in the 1920s, show that pellagra had practically disappeared; only 1,466 cases, mainly chronic elderly cases, were enumerated in 1926.19

Death statistics confirm the trend which, because of the late start of the statistics, can be followed only after 1887, when the zenith of the epidemic was over (Table 4). Between 1887 to 1891 and 1912 to 1916, the mortality rate from pellagra declined from 152 per 100,000 population to 23 in Lombardia; from 228 to 60 in Veneto; from 118 to 12 in Emilia; and from 40 to 8 in the other four regions stricken by the disease. During the same period there was a rapid aging of the distribution of deaths from pellagra, as is to be expected from a chronic disease with declining incidence. The partial statistics from 1881 to 1886 show that mortality from

Table 3  Pellagrins according to Various Official Surveys, 1879, 1881, 1899, and 1910

<table>
<thead>
<tr>
<th>AREA</th>
<th>1879</th>
<th>1881</th>
<th>1899</th>
<th>1910</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lombardia</td>
<td>40,838</td>
<td>36,630</td>
<td>19,547</td>
<td>8,231</td>
</tr>
<tr>
<td>Veneto</td>
<td>29,836</td>
<td>55,881</td>
<td>39,892</td>
<td>20,303</td>
</tr>
<tr>
<td>Emilia</td>
<td>18,728</td>
<td>7,891</td>
<td>4,617</td>
<td>1,808</td>
</tr>
<tr>
<td>4 Regions</td>
<td>8,229</td>
<td>3,467</td>
<td>8,267</td>
<td>3,319</td>
</tr>
<tr>
<td>Other Regions</td>
<td>32</td>
<td>146</td>
<td>208</td>
<td></td>
</tr>
<tr>
<td>ITALY</td>
<td>97,855</td>
<td>104,067</td>
<td>72,603</td>
<td>33,869</td>
</tr>
</tbody>
</table>

Pellagrins per 100,000 population

<table>
<thead>
<tr>
<th>AREA</th>
<th>1879</th>
<th>1881</th>
<th>1899</th>
<th>1910</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lombardia</td>
<td>1,109</td>
<td>995</td>
<td>465</td>
<td>172</td>
</tr>
<tr>
<td>Veneto</td>
<td>1,060</td>
<td>1,986</td>
<td>1,869</td>
<td>576</td>
</tr>
<tr>
<td>Emilia</td>
<td>858</td>
<td>361</td>
<td>189</td>
<td>67</td>
</tr>
<tr>
<td>4 Regions</td>
<td>121</td>
<td>51</td>
<td>109</td>
<td>42</td>
</tr>
<tr>
<td>Other Regions</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>ITALY</td>
<td>344</td>
<td>366</td>
<td>224</td>
<td>98</td>
</tr>
</tbody>
</table>

a The 4 regions are Piemonte, Toscana, Marche, and Umbria.


19 Data for 1839 are quoted by Jacini, “La proprietà fondiaria e le popolazioni agricole in Lombardia,” Biblioteca dell’Economista, II (1861), 361. For the survey of 1856 and for Brescia and Veneto, MAIC, “La pellagra” (1879). For 1926, Ministero dell’Interno, Relazione al Consiglio Superiore di Sanità, 1 Luglio 1926–30 Giugno 1927 (Rome, 1928), I.
Table 4  Deaths and Death Rates by Pellagra, by Region, 1887–91 to 1927–31

<table>
<thead>
<tr>
<th>PERIOD</th>
<th>LOMBARDIA</th>
<th>VENETO</th>
<th>EMILIA</th>
<th>4 REGIONS(^a)</th>
<th>TOTAL 7 REGIONS(^b)</th>
<th>LOMBARDIA</th>
<th>VENETO</th>
<th>EMILIA</th>
<th>4 REGIONS(^a)</th>
<th>TOTAL 7 REGIONS(^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1887–91</td>
<td>5,938</td>
<td>6,679</td>
<td>2,686</td>
<td>2,862</td>
<td>18,263</td>
<td>152</td>
<td>228</td>
<td>118</td>
<td>40</td>
<td>113</td>
</tr>
<tr>
<td>1892–96</td>
<td>5,705</td>
<td>5,528</td>
<td>2,690</td>
<td>2,834</td>
<td>16,917</td>
<td>141</td>
<td>184</td>
<td>115</td>
<td>39</td>
<td>101</td>
</tr>
<tr>
<td>1897–01</td>
<td>4,645</td>
<td>5,547</td>
<td>3,413</td>
<td>3,664</td>
<td>17,472</td>
<td>111</td>
<td>179</td>
<td>142</td>
<td>49</td>
<td>102</td>
</tr>
<tr>
<td>1902–06</td>
<td>3,175</td>
<td>4,355</td>
<td>1,605</td>
<td>2,445</td>
<td>11,767</td>
<td>72</td>
<td>135</td>
<td>64</td>
<td>32</td>
<td>66</td>
</tr>
<tr>
<td>1907–11</td>
<td>1,850</td>
<td>2,989</td>
<td>733</td>
<td>1,214</td>
<td>6,923</td>
<td>40</td>
<td>87</td>
<td>28</td>
<td>16</td>
<td>37</td>
</tr>
<tr>
<td>1912–16</td>
<td>1,131</td>
<td>2,180</td>
<td>322</td>
<td>652</td>
<td>4,349</td>
<td>23</td>
<td>60</td>
<td>12</td>
<td>8</td>
<td>23</td>
</tr>
<tr>
<td>1917–21</td>
<td>820</td>
<td>1,047</td>
<td>167</td>
<td>330</td>
<td>2,408</td>
<td>16</td>
<td>27</td>
<td>6</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>1922–26</td>
<td>224</td>
<td>280</td>
<td>60</td>
<td>123</td>
<td>710</td>
<td>4</td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>1927–31</td>
<td>98</td>
<td>204</td>
<td>40</td>
<td>46</td>
<td>403</td>
<td>2</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

\(^a\) The 4 regions are Piemonte, Toscana, Marche, and Umbria.
\(^b\) The total number of deaths for the 7 regions is the total for Italy and contains about 1 percent of the deaths of Liguria, Lazio, and Abruzzi.

Table 5  Index of Marital Fertility and Prevalence of Pellagra, 1881, in 111 Districts of Lombardia and Veneto

<table>
<thead>
<tr>
<th>LEVEL OF $I_g$</th>
<th>AVERAGE LEVEL OF $I_g$ (1880–82)</th>
<th>PERSONS WITH PELLAGRA PER 100,000 (1881)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>DISTRICTS</td>
<td></td>
</tr>
<tr>
<td>−.600</td>
<td>30</td>
<td>.571</td>
</tr>
<tr>
<td>.600−.649</td>
<td>33</td>
<td>.628</td>
</tr>
<tr>
<td>.650−.699</td>
<td>17</td>
<td>.677</td>
</tr>
<tr>
<td>.700−.749</td>
<td>20</td>
<td>.723</td>
</tr>
<tr>
<td>.750−.799</td>
<td>9</td>
<td>.778</td>
</tr>
<tr>
<td>.800 and over</td>
<td>2</td>
<td>.854</td>
</tr>
<tr>
<td>Mean</td>
<td>–</td>
<td>.653</td>
</tr>
</tbody>
</table>

Table 6  Variation of the Index of Marital Fertility, 1881 to 1911, and Prevalence of Pellagra in 1881, in 103 Districts of Lombardia and Veneto

<table>
<thead>
<tr>
<th>PERCENT VARIATION OF $I_g$, 1880−82 TO 1910−12</th>
<th>N. OF DISTRICTS</th>
<th>AVERAGE VAR. OF $I_g$ 1880−82 TO 1910−12</th>
<th>LEVEL OF $I_g$ 1910−12</th>
<th>PERSONS WITH PELLAGRA PER 100,000 (1881)</th>
</tr>
</thead>
<tbody>
<tr>
<td>−10 and over</td>
<td>9</td>
<td>−20.8</td>
<td>.544</td>
<td>23</td>
</tr>
<tr>
<td>−10 to 0</td>
<td>15</td>
<td>−5.5</td>
<td>.643</td>
<td>112</td>
</tr>
<tr>
<td>0 to +10</td>
<td>27</td>
<td>+5.6</td>
<td>.698</td>
<td>116</td>
</tr>
<tr>
<td>+10 to +20</td>
<td>29</td>
<td>+15.9</td>
<td>.721</td>
<td>166</td>
</tr>
<tr>
<td>+20 to +30</td>
<td>12</td>
<td>+24.1</td>
<td>.790</td>
<td>323</td>
</tr>
<tr>
<td>+30 and over</td>
<td>11</td>
<td>+38.0</td>
<td>.836</td>
<td>377</td>
</tr>
<tr>
<td>Mean</td>
<td>–</td>
<td>+10.2</td>
<td>.710</td>
<td>174</td>
</tr>
</tbody>
</table>

1881. The relationship is a direct one: the highest increase of $I_g$ took place in the circondari where pellagra had the maximum incidence in 1881; the few circondari of the two regions which experienced a decline in marital fertility had a low incidence of the disease. Table 6 also shows the level of $I_g$ for 1910 to 1912 according to classes of variation of $I_g$ for 1880 to 1882 and for 1910 to 1912 and the mean incidence of pellagra in 1881. In a regime of natural fertility, if pellagra were the only source of fertility differences, one would expect that the elimination of pellagra (as almost happened before World War I) would remove all source of variation in fertility. But this was not the case; in Table 6, $I_g$ for 1910 to 1912 is positively associated with the level
of pellagra in 1881 (which was *negatively* associated with $I_g$ for 1880 to 1882, as shown in Table 5).

This fact, however, is not surprising, since pellagra is not the only source of fertility variation during the period examined. Oversimplifying a very complex reality, we could say two classes of factors affect fertility over the period under investigation: first, factors affecting natural fertility (mainly pellagra); and second, factors determining the diffusion of fertility control. Although the evolution of the first factor determines the decrease in the variation of natural fertility levels, the second factor introduces a new source of variation (indeed, during the period, the diffusion of fertility control is evident in several areas). But both factors are also inversely associated—development was more rapid in the areas with better living conditions in 1881 and lower pellagra—and this fact explains why the negative association of pellagra (1881) with $I_g$ (1880–82) becomes positive when pellagra (1881) is associated with $I_g$ (1910–12).20

When the other indicator of the incidence of pellagra (the pellagra-specific mortality rate) is used, similar results are obtained. However, since death statistics by cause are only available from 1887 at the provincial level, all provinces with a significant death rate from pellagra have been considered. There are twenty-nine in all, including some provinces of Marche and Umbria and Emilia. Table 7, in full conformity with the results of Tables 5 and 6, shows the existence of a negative association between death rates by pellagra from 1887 to 1889 and marital fertility from 1880 to 1882 and a strong positive association between pellagra levels from 1887 to 1889 and marital fertility from 1910 to 1912.

The results are interesting but probably inconclusive; data on pellagra are far from flawless, although the agreement between two independent sources (surveys of pellagrins and death statistics) is reassuring.

20 The poor quality and doubtful completeness of the data on pellagra prevalence does not call for refined measures of the association between pellagra and fertility. The correlation coefficients for the *circondari* of Lombardia and Veneto between I (1881) and the prevalence of pellagra (1880–82) is weakly negative ($r = −.141$) and higher for Lombardia (35 *circondari*, $r = −.290$) than for Veneto (76 *circondari*, $r = −.122$). The correlation coefficient between the variation of $I_g$ between 1880 to 1882 and 1910 to 1912 and the pellagra prevalence of 1881 is $+.541$ for the pooled *circondari* of the two regions and higher for Lombardia ($+.711$) than for Veneto ($+.470$).
Table 7  Index of Marital Fertility, 1881 and 1911, and Death Rate by Pellagra, 1887–89, in 29 Provinces

<table>
<thead>
<tr>
<th>LEVEL OF Iₖ</th>
<th>NUMBER OF PROVINCES (29)*</th>
<th>AVERAGE LEVEL OF Iₖ</th>
<th>DEATHS OF PELLAGRA PER 100,000 INHABS. (1887–89)</th>
</tr>
</thead>
<tbody>
<tr>
<td>−600</td>
<td>7</td>
<td>.597</td>
<td>26.6</td>
</tr>
<tr>
<td>600–649</td>
<td>11</td>
<td>.619</td>
<td>33.7</td>
</tr>
<tr>
<td>650–699</td>
<td>6</td>
<td>.670</td>
<td>32.8</td>
</tr>
<tr>
<td>700–749</td>
<td>3</td>
<td>.727</td>
<td>20.0</td>
</tr>
<tr>
<td>750 and over</td>
<td>2</td>
<td>.759</td>
<td>19.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>.641</td>
<td>29.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1910–12</td>
<td></td>
</tr>
<tr>
<td>−600</td>
<td>5</td>
<td>.552</td>
<td>17.0</td>
</tr>
<tr>
<td>600–649</td>
<td>6</td>
<td>.629</td>
<td>20.7</td>
</tr>
<tr>
<td>650–699</td>
<td>7</td>
<td>.674</td>
<td>26.6</td>
</tr>
<tr>
<td>700–749</td>
<td>8</td>
<td>.728</td>
<td>36.6</td>
</tr>
<tr>
<td>750 and over</td>
<td>3</td>
<td>.806</td>
<td>54.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>.672</td>
<td>29.4</td>
</tr>
</tbody>
</table>

* The 29 Provinces belong to Lombardia (8), Veneto (8), Emilia (8), Marche (4), and Umbria (1).

Three aspects of the relationship between fertility and pellagra should be emphasized. The first concerns the striking coincidence between the pellagra-stricken areas and the area where marital fertility rose during the three or four decades preceding World War I. The second is the negative association between the level of marital fertility from 1880 to 1882 and the contemporary incidence of pellagra. The third is the obvious positive association between the rise in marital fertility and the level of pellagra during the 1880s. The areas where pellagra was highest in the 1880s and from which it disappeared before the war were also the areas with the largest increase in marital fertility.

IN SEARCH OF A CAUSAL LINK  Are the diffusion of pellagra in the 1870s and 1880s and its rapid decline at the beginning of this century sufficient elements to explain the rise in marital fertility in northeastern Italy? In theory, it is clear that endemic and chronic disease with the etiology of pellagra must have had a depressive effect on natural fertility. Weakness, apathy, and neurasthenia in the initial stage of the disease might easily have caused a loss of libido and affected the frequency of sexual intercourse. The diges-
Out of Africa: The Slave Trade and the Transmission of Smallpox to Brazil, 1560–1831

Among the “shock troops of the conquest” of the Americas—the variety of infectious diseases introduced to the New World from Europe and Africa—smallpox was undoubtedly the leading killer. Certainly that was the case in colonial Brazil. We suggest that for much of the period between the beginning of European contact (1500) and about 1831, when legal forms of the slave trade were ended, the primary source of smallpox contagion in Brazil was tropical Africa.

Our argument may be summarized as follows:

(1) Prior to the arrival of the first Europeans, smallpox (Varicella major, the only one of three modern species of the virus then existing) was unknown in the future Portuguese colony, or in any other part of the Americas.

(2) Because the pox survived in a community only as long as sufficient susceptibles existed to sustain the disease, and because of Brazil’s persisting low population densities, Variola probably did not become endemic there until the nineteenth century. New outbreaks of the sickness, at least massive ones, therefore followed reintroductions from external sources, initially European but subsequently African.

(3) As a prime New World importer of slaves, Brazil received regular infusions of potential African carriers of the disease, mainly from the Upper Guinea Coast (Senegambia) in the six-

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The authors gratefully acknowledge the stimulation and encouragement from panelists and members of the audience at the initial presentation of this research at the 1982 meeting of the American Historical Association. Ann C. Carmichael and Donald R. Hopkins made informed critiques of earlier drafts. Miller is grateful to the National Library of Medicine for financial and skilled staff support, and to the University of Virginia. Alden joins his thanks, especially to Dorothy Hanks at the National Library of Medicine, Dorothy Welker, and the John Simon Guggenheim Memorial Foundation.

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teenth century, from the southwestern littoral between 4° and 18° S. lat. (known as Angola) beginning in the early seventeenth century, from the Mina (or Lower Guinea) Coast of what is today Togo, Bénin, and southwestern Nigeria after about 1700, and by the end of the eighteenth century also from southeastern Africa (or Mozambique).

4 Periodic drought, famine, and epidemic smallpox in the parts of Africa that contributed significant numbers of slaves to Brazil corresponded reasonably well with the timing of major smallpox eruptions in the Portuguese colony from the seventeenth through the early nineteenth centuries.

5 Conditions in Africa and on the middle passage favored transmission of the infection in the bodies of the unfortunates sent as slaves to Brazil. Normally dispersed African populations became compressed in relatively moist regions during droughts, when infections quickly spread among non-immunes. Transmission of such diseases intensified further among slaves awaiting shipment in maritime barracoons and among undernourished, sick captives crowded closely together on the decks of slave ships. The thirty- to fifty-day Atlantic crossing permitted more than one cycle of infection among the several hundred chattels aboard the typical slaver.

6 Demographic patterns in Brazil explain the subsequent spread of the disease in epidemic form among American populations. When the slaves reached one of the major Brazilian ports—Recife de Pernambuco, Salvador da Bahia, and Rio de Janeiro on the east coast, and São Luis do Maranhão and Belém do Pará on the northern coast—they spread contagion rapidly from the waterside sheds in which they waited to be auctioned off throughout the commercial quarters of those towns. As they moved inland to plantations, mines, and settlements, the African newcomers transmitted the virus to virgin Amerindian populations and to previously unexposed individuals of African origin with whom they came into close contact.

ORIGINS AND IMPLANTATION OF SMALLPOX IN THE NEW WORLD
Smallpox is an orthopoxvirus, a genus that includes, among others, camelpox, cowpox, monkeypox, and vaccinia. The most common means of transmission was via virus-bearing moisture droplets exhaled by afflicted persons and inhaled by those with
smallpox epidemics during the remainder of the sixteenth century, although the disease broke out locally on at least three occasions in 1585, 1597, and 1599. The second of these minor epidemics, which forced a Portuguese commander to abandon a military campaign to occupy the coast of Rio Grande do Norte, in northeasternmost Brazil, may represent the first introduction of the pox from West Africa to Brazil. French ships frequented that part of the coast in search of brazilwood, and in that same year a French vessel that had earlier raided the Portuguese factory at Arguin island off the Saharan coast arrived in Bahia bearing smallpox. Droughts had struck the Cape Verde Islands off the West African mainland between 1580 and 1582 and again in 1594, and the years 1574 to 1587 witnessed a succession of failed rains, famine, and sickness in Angola.5

THE SPREAD OF SMALLPOX IN THE SEVENTEENTH CENTURY Slaves reached Brazil in increasing numbers—from around 4,000 a year to as many as 6,000 to 7,000—throughout the seventeenth century, and smallpox epidemics increased apace. Their African provenance also became more clearly established. The next outbreak of smallpox occurred in 1613, when extensive mortality depleted the slaves on the sugar estates in Rio de Janeiro. It was attributed explicitly by contemporaries to blacks. Three years later, what was termed sarampo e bexigas—measles (then difficult to distinguish from smallpox and several other rash-provoking maladies) and smallpox—attacked the northeast. Its source was identified as slaves from the Kongo in central Africa and from Allada on the Lower Guinea Coast.6


6 Vivaldo Coaracy, O Rio de Janeiro no século 17 (Rio de Janeiro, 1965), 38; Ambrosio
Two further outbreaks of pox occurred at the beginning of the 1620s. The first, another visitation upon the north coast, came via a ship from Pernambuco that brought the scourge to São Luís do Maranhão in 1621. The same epidemic swept on through Pernambuco and three other northeastern captaincies between 1621 and 1623. Although the extent of casualties is unknown, the contagion was sufficiently severe to provoke municipal authorities in Pernambuco to impose the first recorded quarantine for slaves arriving in Brazil.7

The simultaneous reappearance of smallpox in both northern and southern Brazil at this time coincided with new cycles of drought, attendant famine, and disease in both sahelian West Africa and Angola. The usually populous southern margins of the Sahara Desert appear to have enjoyed favorable rainfall during the fifteenth and sixteenth centuries, but they became increasingly arid during the next 200 years or so. As the dryness spread, numerous populations along the desert margins fell into conflict over shrinking living space. Their wars contributed captives to the streams of refugees that supplied slaves to Europeans buying labor along the West African coast. The populous interior delta of the Niger River experienced drought by 1617, and the Cape Verde Islands were beset by famine and sickness in 1620. Angola experienced a major smallpox epidemic, exacerbated by drought, from 1625 to 1628. In the mid-1620s, several slave ships from Luanda, the main port on the Angolan coast, reached Salvador after suffering extraordinarily high losses among their human cargoes. Nevertheless, smallpox-afflicted survivors were landed.8

Despite intensive fighting and population dislocations from the Dutch seizure of Brazil between 1630 and 1654, and the efforts of the Dutch to resupply the Northeast with fresh slaves from parallel conquests in Africa, the American colony appears to have

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8 Sources for climate history in Angola are nearly all cited in Miller, "The Significance of Drought, Disease, and Famine in the Agriculturally Marginal Zones of West-Central Africa," Journal of African History, XXIII (1982), 17–61.
escaped a serious encounter with the pox until 1641. That year excessively heavy rains fell in the Northeast, and a virulent pandemic of smallpox swept throughout Dutch-held Brazil and beyond. In the single captaincy of Paraíba (immediately north of Pernambuco, center of the Dutch presence) an estimated 1,100 blacks became its victims. Three years later, smallpox caused extensive losses among the Indian villages of Maranhão.  

Although no observer specified the source of these epidemics, they may again have been products of arid conditions then prevailing in western and central Africa. The pace of droughts in the sahel had quickened as rains failed from 1639 to 1643, and Portuguese traders in Cacheu, the principal Upper Guinea source of slaves for Brazil, reported famine in 1641. Two years earlier, an extended drought had afflicted Angola from the mouth of the Zaire River south beyond the Kwanza River to the Portuguese outpost of Benguela.  

Western and central African climates improved during the decade and a half after about 1645, and Brazil was spared further major smallpox outbreaks during those years. But a new wave of sickness hit what was then becoming Portugal’s most important colony during the early and mid-1660s. A “pestilential catarrh” (influenza or tuberculosis) swept Maranhão in 1660, and two years later a lethal attack of smallpox struck Maranhão and the adjacent captaincy of Pará. João Felippe Betendorf, a Jesuit father who ministered to the sick Indians of the north, described Maranhão as “burning with a plague of smallpox... Parents abandoned their children and fled into the forests to avoid such a pestilential evil.” Stricken Indians were so seriously afflicted that their skins became black, and their bodies became so fever-ridden that “pieces of their flesh fell off.”  

No casualty estimates survive for these Maranhão epidemics, and the same is true for the second Brazilian pox pandemic that

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Neither the African stations nor Brazil suffered a major contagion early in the century, but that hiatus ended abruptly in 1715. Smallpox ravaged Angola between 1715 and 1720, and Lower Guinea witnessed “thousands of Men . . . swept away” by the disease, according to a surgeon of the Royal African Company on the nearby Gold Coast. Smallpox appeared in Pernambuco on schedule in 1715 and caused serious losses in the Bahian backlands the following year. An Angolan ship bearing pox-infected chattels reached Salvador also in 1716, causing devastation in the city. By 1718 the disease appeared in Rio de Janeiro and by 1730 was in São Paulo. Between 1715 and 1730 the series of epidemics that swept the entire littoral of Brazil almost certainly originated in Africa.18

The 1720s, 1730s, and 1740s saw continuing frequent and severe epidemics in various parts of Brazil. The first outbreak seems to have been triggered from Angola, but West Africa may also have become a source by mid-century, when the most severe drought of the 1700s overspread the region. In 1724, the pox appeared in Maranhão and Pará and was carried from Belém to plantations and mission stations up the Amazon River, creating serious shortages of Indian laborers. São Paulo and its principal seaport, Santos, were also struck by pox between 1724 and 1725. Rains failed totally along the Angolan coast between 1724 and 1726, and food scarcities in its capital, Luanda, drove grain prices to unprecedented heights. Smallpox broke out in 1725 and, by 1727, the rise in slave mortality was ruining merchants there.19

The years 1729 to 1732, which were marked by low rainfall and famine in West Africa along the Senegal River, also saw renewed contagion in various parts of coastal Brazil. Between 1730 and 1732, smallpox returned to the city of São Paulo, as

18 Arquivo do Municipal da Bahia, Cartas de senado a sua magesdata, XXXVIII: 9, fl. 12; Luis Lisanti (ed.), Negócios coloniais (Uma correspondência do século XVIII) (São Paulo, 1973). I, 91–92; II, 131; Sergio Buarque de Holanda, “Movimentos da população em São Paulo no século XVIII,” Revista do Instituto de Estudos Brasileiros, I (1966), 77; Elena F. Scheuss de Studer, La trata de negros en el Río de la Plata durante el siglo XVIII (Buenos Aires, 1958), Fig. V.

well as to the captaincies of Pernambuco and Pará. In Angola drought again led to famine in 1735/36, and an epidemic of an unstated nature broke out in the latter year. Another unspecified malady claimed 3,000 lives in the captaincy of Rio de Janeiro in 1737. Very possibly its source was the pox which ravaged Bahia the same year.  

Infectious diseases along the entire American shore of the Atlantic reached their eighteenth-century peaks during the 1740s. In Brazil, the decade produced catastrophic losses from three separate epidemics of smallpox in São Paulo, but the captaincies of Maranhão and Pará were even more grievously stricken. The first serious poxian outbreak in two decades visited Belém in August 1743, with debilitating secondary infections of catarrhs, pleurisy, and bloody flux. Then, in 1749, just as those maladies had run their course, Belém, São Luís, and the vast Amazonian interior were hit by a lethal epidemic of measles. During most of the 1750s, waves of smallpox continued to wash over Maranhão and over all of Brazil. 

These middle decades of the century also saw widespread drought, hunger, and diseases among both Africans and Europeans in West Africa. Supposedly half of the population of the upper Niger River valley perished at this time. Food was in short supply along the Gold Coast in 1743 and again in the early 1750s. The drought-prone Cape Verde Islands, always a sensitive pluviometer for the climate of the adjacent West African mainland, experienced serious famine in the late 1740s and again in 1754. Slaves perished from malnutrition at the French post near the mouth of the Senegal River in 1751/52. Conditions were somewhat better in Angola, although smallpox was reported aboard a slaver en route from there to Pernambuco in 1759. 

20 Taunay, História da cidade, III, 161–164; APB/OR/26, no. 38; /27, no. 68; Inácio Accioli de Cerqueira e Silva (ed. Braz do Amaral), Memórias históricas da Bahia (Salvador, 1935), II, 378; Couto, “Desagrados do Brasil,” 184; Sor. Mariana Bernarda and Sor. Mª Margarida Bittencourt to king, ca. 1733, Arquivo Histórico Ultramarino (Lisbon) (hereafter AHU), Papes Avulsos (PA)/Pará cx. 7; Gomes Freire de Andrada to António Guedes Pereira, 9 June 1737, Arquivo Nacional, Rio de Janeiro (hereafter ANR), col. 60, 6, fl. 224; Conde das Galveas to king, 15 May 1738, APB/OR/35, no. 75. 

21 Numerous accounts scattered in AHU/PA/Maranhão, cx. 29 and maça 1, and AHU/PA/Pará, cxs. 1, 3, 12. Also Leduar de Assis Rocha, Efemérides médicas pernambucanas, séculos XVI, XVII e XVIII (Recife, c 1954), 53. 

Smallpox remained the principal scourge of Brazil during the second half of the eighteenth century. Northern Brazil continued to be a major theater of the infection, all the more because the state of Maranhão shifted after the mid-1750s from its traditional reliance on Indian labor to a dependency on imported African slaves. A royally chartered Maranhão Company initially purchased cargoes in Luanda at a time when prices there fell in response to drought, famine, and epidemics. The first shipments reached Belém in 1756, and that winter the city was beset by “a terrible epidemic of catarrhs” that had not abated before smallpox also broke out in the city. Company agents purchased more slaves at Luanda in the early 1760s, and the all-but-predictable smallpox epidemics followed in Maranhão in 1762/63 and 1766/67. The latter outbreak may well have followed renewed drought and a poxian upsurge in Angola between 1765 and 1767.23

Brazil’s east coast also suffered intermittent contagion between the late 1760s and the early 1780s. Smallpox, accompanied by jaundice and leprosy, appeared in São Paulo in 1768. Eleven years later the pox caused hundreds of persons to be admitted to Salvador’s two hospitals, where most of them died. In 1780, São Paulo again suffered from an eruption of smallpox, as did Pernambuco and adjacent northern captaincies on numerous occasions between 1774 and 1787.24

The last seven years of the eighteenth century witnessed another major Variola epidemic that was especially serious in northern Brazil. As the Portuguese minister of marine in Lisbon reviewed reports from the American colony, he lamented “the great damage that the pox has been causing, and continues to cause, all over Brazil.”25

25 Francisco Innocencio de Sousa Coutinho to Luis Pinto de Sousa, 7 June 1796, 12 Feb.
to delay debarkations from disease-ridden slave ships in Brazilian ports since the early seventeenth century. These measures had largely failed, because someone was always willing to purchase even mortally ill slaves. Inoculation, also known as variolation, had offered an alternative method of prevention since the middle of the eighteenth century. Inoculation procedures transferred a small portion of virally infected matter from a lesion on the skin of a sufferer to that of a healthy partner; a mild infection would usually result, conferring lifetime immunity from smallpox. Some Africans practiced variolation by the early eighteenth century, and the technique spread from Turkish sources to England in 1721 and to some French and British slavers in the Atlantic by the 1760s and 1770s. There and in Britain’s North American colonies it sharply reduced the extent and virulence of smallpox. No comparable decline had lessened Brazil’s sufferings, although missionaries in Portuguese America occasionally experimented with inoculation in the late 1720s and again in the 1740s. A standard Portuguese medical text published in 1761 reported that variolation was seldom employed, and advised against its use.31

Both the Portuguese Crown and its colonial agents finally became interested in the therapeutic possibilities of variolation by the 1790s, perhaps sparked by the Pernambucan-born Francisco Arruda Câmara, who wrote a thesis on smallpox inoculation at the French medical school at Montpellier, from which he graduated in 1790. The alarming reports of poxian virulence in Brazil in the 1790s led to the establishment of a special hospital in Lisbon to treat victims. In 1798, Diogo de Sousa, governor of Maranhão, promoted variolation during the smallpox outbreak there and achieved some success among whites and blacks, but less among Indians. The same year the governor of neighboring and fever-ridden Pará received royal authority to offer inoculations at state

expense to all persons in Belém willing to undergo the risk. Far
to the south, Francisco Mendes Ribeiro de Vasconcelos, a military
surgeon in Rio de Janeiro, won public acclaim for his efforts to
variolate against the “smallpox . . . [that had] alarmed sugar
planters and other agriculturalists at seeing their slaves die.” By
a general circular of 9 July 1799, the Crown directed all colonial
governors to initiate variolation programs, especially among
young black and Indian children, “since experience has shown
this to be the only effective defense against the scourge . . . which
has caused such considerable devastation in the Portuguese colo-
nies.”

That circular followed one year after Edward Jenner, an En-
glish physician, had published his confirmation of a longstanding
rural belief that persons whose skins were scored with cowpox
could gain immunity to smallpox at less risk than from variolation
with human viral matter. A small percentage of those inoculated
succumbed to the infection and could also transmit it to others at
dangerously uncontrolled levels. Jenner’s vaccine carried none of
these disadvantages and its use spread quickly throughout Europe
and abroad.

It is unlikely that the Portuguese Crown meant the inocula-
tion program it announced in 1799 to involve the novel use of
cowpox. Rather, variolation was probably intended. However,
vials of Jenner’s cowpox lymph were sent from London to the
University of Coimbra and to Lisbon in the same year, and in
1800 the Crown advised overseas authorities of the existence of
the new preventive. Copies of an account of the Jenner method
by de Paiva, a popularizer of current medical knowledge, were
sent to Brazil in 1801. The Jenner vaccine itself first arrived in
Brazil in 1804, when a wealthy Brazilian landowner, Francisco

32 Silva Araujo, *Immortalized Cow*, 18; Ernesto de Souza Campos, “Considerações sobre
a ocorrência de variola e vacina nos séculos xvii, xviii e xix sob a luz de documentação
coeva,” *Revista do Instituto Histórico e Geográfico Brasileiro*, CCXXXI (1956), 147–149;
Vianna, *As epidemias no Pard*, 45–46; “Representação de moradores do Rio de Janeiro sobre
a vantagem da vacina,” 15 June 1798, Biblioteca Nacional (Rio de Janeiro) (hereafter
BNRJ), II–32, 16, 9; another petition, 20 Feb. 1800, BNRJ, II–34, 15, 32; Teixeira Botelho,
“Acerca da vacinação,” 203.

33 The older literature preceding Hopkings, *Princes and Peasants*, includes: Genevieve
Miller, *The Adoption of Inoculation for Smallpox in England and France* (Philadelphia, 1957);
Peter Razzell, *Edward Jenner's Cowpox Vaccine: The History of a Medical Myth* (Firle, 1977);
Caldeira Brant, sent seven of his slaves to Lisbon to be vaccinated and to be returned to Brazil as fresh human reservoirs of the protective lymph. Their owner met them at the Salvador dockside and had his son become the first person to be vaccinated in Brazil on 31 December 1804. The boy obviously survived the experience, for he died in 1906 at the remarkable age of 104.\(^{34}\)

News of the Jenner method and supplies of the vaccine traveled rapidly along the Brazilian coast, reaching Rio de Janeiro and Pernambuco in 1805. Elsewhere enlightened administrators, hearing of the success of the innovative procedure, sent children to Bahia to bring the live vaccine back to their own captaincies. In Maranhão, where some of the lymph had recently arrived, a ship arrived from Angola in 1806 bearing infected slaves but failed to ignite an epidemic. That same year, the Crown distributed throughout the empire copies of a pamphlet prepared by the surgeon-general of Portuguese India to reassure remaining doubters about the safety of vaccination and asserting its superior effectiveness over other forms of prophylaxis. In 1811, three years after the exiled Portuguese court arrived in Rio de Janeiro, a municipal vaccine commission was created in Brazil’s most important city, and it continued to function down to 1835. In those twenty-four years its staff administered 102,791 vaccinations, concentrating on newly arrived slaves and on those already working on plantations. By 1819, plans were announced for the establishment of a similar clinic in São Paulo.\(^{35}\)

Public vaccination began that same year in Luanda, after several unsuccessful efforts to import the vaccine live from Brazil. Luanda officials had made their first attempt in 1806 during the onset of the smallpox epidemic that lasted until 1808, but the lymph expired during the crossing. The same experiment failed again the next year. A third attempt proved successful in 1819, and by early 1821, less than a year and half after the vaccine had


### APPENDIX

**Summary Chronology of Droughts and Epidemics in Africa and Brazil, c 1500–c 1840**

<table>
<thead>
<tr>
<th>Time line</th>
<th>West Africa</th>
<th>Angola</th>
<th>Mozambique</th>
<th>Brazil</th>
<th>Other American</th>
</tr>
</thead>
<tbody>
<tr>
<td>1500</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1507: Slavers alleged to have introduced smallpox-infected slaves in Hispaniola.</td>
</tr>
<tr>
<td>1510</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1518–20: Transfer of smallpox from Caribbean to Mexican mainland.</td>
</tr>
<tr>
<td>1520</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1520s: Smallpox epidemics in highland Mexico and Peru.</td>
</tr>
<tr>
<td>1530</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1549</td>
<td>1549: Drought in Cape Verde Islands.</td>
<td></td>
<td></td>
<td>1549: Sick slaves (disease unknown) at Bahia.</td>
<td></td>
</tr>
<tr>
<td>1550</td>
<td>1558–60: Hunger (?) and high mortality at São Tomé.</td>
<td></td>
<td></td>
<td></td>
<td>1558–69: Smallpox at the Rio de la Plata and elsewhere in Spanish South America.</td>
</tr>
<tr>
<td>Time Line</td>
<td>West Africa</td>
<td>Angola</td>
<td>Mozambique</td>
<td>Brazil</td>
<td>Other American</td>
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</tr>
<tr>
<td>1560</td>
<td></td>
<td>1561–63: Drought and famine in interior.</td>
<td></td>
<td>1562–65: SMALL-POX PANDEMIC, PERNAMBUCO TO SÃO VICENTE.</td>
<td></td>
</tr>
<tr>
<td>1570</td>
<td></td>
<td>c 1574–88: Drought, continuing intermittently. (drought)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1580</td>
<td>c 1580–82: Drought in Cape Verdes.</td>
<td>1584: Sickness noted at Luanda.</td>
<td></td>
<td></td>
<td>1581: Smallpox in Peru.</td>
</tr>
<tr>
<td>1590</td>
<td>1594: Drought in Cape Verdes.</td>
<td></td>
<td></td>
<td>1585: Smallpox in Ilhéus.</td>
<td></td>
</tr>
<tr>
<td>1600</td>
<td></td>
<td></td>
<td></td>
<td>1597: French vessel from Arguin (West Africa) introduced smallpox in Rio Grande do Norte.</td>
<td></td>
</tr>
<tr>
<td>1610</td>
<td>1609 (to 1614?): Drought in Cape Verdes.</td>
<td>1614–19: Serious drought.</td>
<td></td>
<td>1611: Smallpox at São Paulo (?).</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1613: Smallpox at Pernambuco.</td>
<td></td>
</tr>
</tbody>
</table>
1616: Smallpox in the northeast, attributed to slaves from both West Africa and Angola.

1620: Famine and sickness in Cape Verdes.

1620–28: First recorded smallpox epidemic, also great drought.

1625: Extraordinarily high mortality on board slavers from Luanda.

1621–23: Smallpox in the north and northeast.

1626: Ship carrying smallpox arrives in Bahia from Angola.

1621: Quarantine measures introduced at Buenos Aires.

1627: Smallpox at Anserma.

1630

1639–41: Excessive rains and smallpox widespread in north, northeast.

1641: Smallpox in Maranhão.

1633–34, 1636, 1639–41: Epidemics at Panamá.
<table>
<thead>
<tr>
<th>Time line</th>
<th>West Africa</th>
<th>Angola</th>
<th>Mozambique</th>
<th>Brazil</th>
<th>Other American</th>
</tr>
</thead>
</table>
1790
1789–91: Drought in Cape Verdes.
1790s: Generally dry in West Africa.
c 1790: Smallpox on River Sherbro (upper Guinea).
1795: Measles and smallpox on the Gold Coast.

1789–94: Extreme famine and disorder, repeated epidemics.
1793(?)-94: High mortality aboard slave ships.
1796-97: Second mortality peak on slavers.
1796: Smallpox aboard slave ship, suppressed by inoculation.

1791–96: Drought in the south.

1791: “Febres miasmaticas” in Ceará.
1793–99: Pandemic smallpox throughout Brazil.

1800
1803–04: Drought in Cape Verdes
1804–05: Drought in Sahara

1800: Recurrent epidemics. Smallpox implied in 1805
1806–08: Mortality rises on slavers at sea.
1799–1803: Dry period in Nguniland (Natal).

1801–02: Smallpox epidemics in New Granada, “less severe.”

1810
1810: Drought in Cape Verdes.

1810: Smallpox further specified in 1811, 1814.
Continuing episodes of drought.

1812: Drought and famine in lower Senegal.
1813–14: Drought in Cape Verdes.
1808: Smallpox in São Paulo
<table>
<thead>
<tr>
<th>Time line</th>
<th>West Africa</th>
<th>Angola</th>
<th>Mozambique</th>
<th>Brazil</th>
<th>Other American</th>
</tr>
</thead>
</table>
| 1820     | 1816: Possible dearth on Gold Coast.  
1822–23: Serious epidemics in Sahara.  
1824: Crop failure on Gold Coast.  
1825: Drought in Cape Verdes. | Continuing epidemics.  
Smallpox specified in 1822 and 1826, implied in 1825. | 1817: Drought in Madagascar.  
1822–32: Widespread drought.  
1824: Famine and wars at Delagoa Bay. | 1819: Smallpox at Belém from a ship from Africa. | |
1834–36: Smallpox reappears at Rio de Janeiro. |
McKeown deals with the periods 1848–1854 to 1901 and post-1901 separately, the significance of sudden changes in infant mortality after 1901 for longer term trends in life expectation may be blurred. McKeown is concerned only with national trends and presents, thereby, an impression of uniformity in his interpretation that belies the significant environmental variations which appear to have existed in both the level of mortality and the structure of cause-of-death patterns.4

Our approach to these issues develops an alternative yet complementary perspective by focusing in detail on the pattern and structure of mortality in the nineteenth century. We are particularly concerned with two aspects: variations in age-specific mortality together with regional or local differences in mortality. Although McKeown was to some extent concerned with the former, he took virtually no account of the latter. Yet the range of life chances experienced by Victorians was considerable. More specifically, we are concerned with two sets of problems: first, the estimation of model life tables which will assist the study of changes in the age-specific structure of mortality; and, second, the influence of environment on variations in the level of mortality. Although our solutions to these problems may not represent a reinterpretation of McKeown's position, they will at least add to the debate by providing a more sophisticated description of the underlying mortality conditions.5

We begin by considering the availability and quality of civil registration data, especially mortality statistics, in Victorian England and Wales. We proceed to a review of the official English Life Tables (ELTs) and the construction of English model life table systems (differentiated here and throughout from official ELTs). The last two sections consider regional variations in mortality


5 McKeown and Record, "Reasons for the Decline," 100. See also Woods and Woodward, *Urban Disease*, 39.
patterns and the influence of environmental factors, as well as the maldistribution of members of the medical profession in the nineteenth century.

VITAL STATISTICS Civil registration began in England and Wales in July 1837. The country was divided into forty-five registration counties and over 600 registration districts, each one of which had a superintendent registrar who was responsible for the compilation of information on births, marriages, and deaths. Of the three, mortality registration was, even from the 1830s, the most comprehensive, since both age at death and cause of death were recorded on a sex-specific basis. The registrar general, based in London, was responsible for the preparation of an annual report, which normally provided a commentary on the course of vital events together with a series of tables reporting annual vital statistics. Special reports on, for example, occupational mortality, were frequently appended, but the degree of detail reported was also liable to vary from year to year. Supplements were also prepared for the twenty-fifth (1862), thirty-fifth (1872), forty-fifth (1882), fifty-fifth (1892), sixty-fifth (1902), and seventy-fifth (1912) annual reports. They gave summaries of the vital statistics for the decades 1851–1860, 1861–1870, 1871–1880, 1881–1890, 1891–1900, and 1901–1910. In 1911 the districts were abandoned as civil registration units in favour of the new local government areas.

Registration districts should therefore provide a convenient scale for the analysis of mortality patterns between 1838 and 1910, but, apart from the decennial supplements, age at death data were only published in the eighteenth (1855) to the forty-seventh (1884) annual reports. Thereafter registration counties were employed and, for the forty-fourth (1881) to the forty-seventh (1884) reports, male and female ages at death were not published separately. Annual sex-specific age at death data are only available for the period 1855 to 1880 and can only be related to population at risk data from the 1861 and 1871 censuses.6

6 There is no comprehensive guide to the publications of vital statistics by the registrar general between 1837 and 1911. Most, but not all, of the reports, abstracts of statistics, supplements, and incidental items on specific issues are to be found in Parliamentary Papers (PP). John M. Eyler, *Victorian Social Medicine: The Ideas and Methods of William Farr* (Baltimore, 1979), provides an interesting insight on the work of the General Register Office and on William Farr, one of its most influential members.
Our analysis of mortality variations rests, therefore, on statistics drawn from the twenty-fourth (1861), twenty-fifth (1862), and twenty-sixth (1863) annual reports of the Registrar General of Births, Deaths and Marriages, together with the 1861 census of population, and is complemented by cause of death data from the Supplement to the Thirty-Fifth (1872) Annual Report for 1861 to 1870. It focuses on the registration districts and ignores those periods for which only decennial or county data are available. Decades and counties are not used for similar reasons: the former cover too long a time span and the latter too large an area; both obscure some of those very environmental variations that we seek to uncover.

The reliability of these data is difficult, if not impossible, to judge. There is no reason to believe that death reporting was particularly inaccurate, but there are grounds for suspecting some bias in the age at death and age structure data, and especially the data on cause of death. The former have been examined in some detail by Lee and Lam for England and Wales as a whole. They concentrate on the age structure of the female population from the 1821 and the 1841 to 1931 censuses, but they also analyze the male structures for 1861 to 1871. Their results suggest, for example, the need to adjust the number of 0–4 year olds given in the 1861 and 1871 censuses by 1.0162 for males and 1.0488 for females. They refer particularly to the high degree of age misreporting in the 1841 census.7

Unfortunately, the methods employed are not amenable to use with registration district data since the differences in age-structures that occur between two censuses are likely to be affected by age-specific net migration. Even Lee and Lam focused their analysis on the female population to avoid, among other things,

ELT 3 also provides the basis for Wrigley and Schofield’s attempt to construct a series of model life tables that would capture the pattern of age-specific mortality particular to England. They needed a family of life tables spanning a range of mortality levels in order to carry out aggregative back projection on the population of England prior to 1871. Looking first at the four families of models constructed by Coale and Demeny and comparing each with ELT 3, they observed that, although the North model provided the closest correspondence, the fit, particularly for females between ages twenty and fifty-five, was not close. To use ELT 3 unmodified as the basis for a family of specifically English life tables was considered undesirable because of the serious problem of age misreporting, especially among the elderly, in the 1841 census. Wrigley and Schofield’s solution to this problem was to splice rates of $nq_x$ from Coale and Demeny’s North model above age fifty onto those from ELT 3. The appropriate North model rates were found to be at level 9.45 and were obtained by linear interpolation between levels 9 and 10 of the English life table. As for level 10 of the English family, the other levels were calculated by applying the ratios of $nq_x$ between any two levels in the North tables to that English table. They present their new English family for both sexes combined for values of $e_0$ ranging from 23.54 to 45.66 (levels 3 to 12).

Wrigley and Schofield’s method produces a set of life tables which probably well reflects the English mortality experience. In order for the method to be of use in the analysis of mortality during the Victorian period, however, it needs to be extended to encompass a range of values of $e_0$ more appropriate to the late nineteenth century. Moreover, it is desirable to present the family in a form which is easier to use. In the following paragraphs we suggest ways in which these modifications could be made and compare the results with those from an alternative and entirely empirical method of constructing model life tables.

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2 females, in a letter to the registrar general by Farr on “The Finance of Life Assurance” (1853) and males in PP 1859/xii; ELT 3, in Farr, English Life Table. Tables of Lifetimes, Annuities, and Premiums (London, 1864); ELT 4, PP 1884/xx; ELT 5, PP 1895/xxiii, pt 1; ELT 6, PP 1905/xviii (also compares ELTs 3, 6); ELTs 7 and 8, PP 1914/xiv.


11 The most recent set of United Nations model life tables is presented in family patterns
We first followed Wrigley and Schofield’s method, and calculated single-sex life tables for levels 7 to 17 of the English family in which \( e_0 \) for males ranges from 32.34 to 56.39 and for females from 34.27 to 59.30, thereby encompassing most of the Victorian experience of mortality. The eleven life tables for each sex so generated were then treated analogously to the set of 326 used by Coale and Demeny. Regression equations were estimated for the relationships between \( nq_{xs} \) and \( e_{10} (1q_0, 4q_1, 5q_5, \ldots, 5q_{75} \) against \( e_{10} \)). Coale and Demeny’s original models used 80+ as the final open-ended age category. Here we prefer to use the more conventional 85+ category and are thus obliged to estimate \( 5q_{80} \). Like Wrigley and Schofield, we have based our estimate on the work of Gabriel and Ronen, who relate \( 5q_{80} \) to \( 5q_{75} \).  

Finally, we need to estimate \( e_{85} \), but here we are assisted by Coale and Demeny’s extended model system which gives \( e_{100} \). We therefore obtained an equation relating \( e_{85} \) to \( l_{85} \) by a simple regression of the values of \( e_{85} \) given in levels 7 to 17 of their extended North model tables on the corresponding \( l_{85} \) values. The final stage of the exercise involved the calculation of \( nq_{xs} \) for levels of \( e_0 \). In a fashion similar to Coale and Demeny, this calculation was accomplished by choosing values of \( e_{10} \), the independent variable, by an iterative procedure so that \( nq_{xs} \) corresponding to \( e_0 \) levels one year apart from 30 to 60 could be found for males and females separately. This complicated exercise yields thirty-one English model life tables for each sex: \( nq_{xs} \) for \( e_0 \)'s equal to 30, 35, 40, 45, 50, 55, and 60 are shown in Table 2.

The vital statistics and the census age structure data described above also provide the means of estimating sex-specific life tables for each of the registration districts in England and Wales in 1861 but organized so that \( e_0 \) is set at 35, 36, 37, 38, \ldots, 75 for males and females independently. This ordering differs from the Coale and Demeny system, where male and female \( e_0 \)'s are tied, and from Wrigley and Schofield, where they are combined. See United Nations, Model Life Tables for Developing Countries (New York, 1982).


13 The extended system is reported in the second edition of Coale and Demeny, Regional Model Life Tables. Table 1 above gives \( e_{85} \) so that all the abridged life table functions can be estimated. Additional \( nq_{xs} \) may be found by interpolation between adjacent \( e_0 \) levels. In life table notation, \( nq_x \) is the probability of dying between age \( x \) and age \( x + n \) (infant mortality is \( 1q_0 \)); \( l_x \) is the number of persons alive and aged \( x \) (\( l_0 \) is the radix of a life table, the assumed number of births per year); and \( e_x \) is life expectancy in years aged \( x \) (\( e_0 \) is life expectation at birth).
remain reliable so long as the North system fits closely to the English experience. In general, estimated model English life tables, but particularly the system based on Wrigley and Schofield’s method, appear to work well over the $e_0$ range 30–60, and especially 30–50.

Our purpose in estimating these English model life tables is twofold. First, we are concerned to emphasize the importance of changes in age-specific mortality in the late nineteenth century. Figure 1 helps to illustrate the important point that certain age groups made more substantial contributions to the rise in life expectations than others and thus that our understanding of mortality decline must focus on those particular ages. The movement of $e_0$ from 40 to 50 is related especially to the fall in mortality among children and young adults, but not among infants, adults, and the elderly. Once $e_0$ exceeds 50, further substantial improvements are only possible with a fall in infant mortality. In England and Wales, the seven-year increase in $e_0$ between 1851 and 1901 is closely linked with particular changes in the age structure of mortality between 1 and 24, whereas the five-year rise in $e_0$ between 1901 and 1911 is associated with additional changes in mortality among those under one year of age. This point is demonstrated most effectively if one scans across the rows for age groups 1–4 to 10–14 in Table 2.\(^\text{15}\)

Second, model life table systems offer a means of checking demographic data for internal inconsistencies and of enabling calculations to be made on estimates of local mortality and fertility in circumstances where vital statistics or census data are incomplete.\(^\text{16}\)


REGIONAL VARIATIONS Whereas life tables provide a most convenient means of representing the structure of age-specific mortality, life expectation at birth \( (e_0) \) and infant mortality \( (1q_0) \) give simple summary indices of the levels of mortality which avoid the distortion inherent in local variations in the age structure of the population at risk of dying. Life expectation and infant mortality are shown in Figures 2 and 3 respectively for both sexes combined in 1861. Both reveal a considerable range in the mortality experience. When \( e_0 \) for England and Wales as a whole was in the low 40s, many rural districts in the south and especially the southwest of England and Wales had \( e_0 \)s above 50 in the early 1860s; in other districts, particularly in the urban industrial areas of Lancashire, Yorkshire, and the northeast, \( e_0 \) was less than 35. Similarly, \( 1q_0 \) was about 0.130 nationally, but was below 0.100 in

Fig. 2 Life Expectation at Birth \( (e_0) \), England and Wales, 1861 (shown by registration districts with both sexes combined).
Several points emerge from Table 4 and the percentages cited above, but three need to be stressed. First, the comparison of cause-specific mortality patterns between areas experiencing extreme mortality conditions is thwarted by the existence of the large residual category "other causes." Second, infant mortality is relatively more important in the urban area and part of the reason for the excess of infant deaths must be attributed to water- and food-borne diseases. Third, there are sufficient similarities between the cause of death patterns of these contrasting populations to imply differences in the levels of morbidity and even the case fatality rates in circumstances where $e_0$ differs by at least fifteen years.20

A second approach to examining the patterns shown in Figures 2 and 3 involves an analysis of the relationship between life expectation at birth and infant mortality. One would expect to find a particularly strong association between $e_0$ and $1q_0$ since infant deaths, as we have seen, represented from 15 to 25 percent of all deaths. It should therefore be possible to predict $1q_0$ from $e_0$. Figure 4 shows the relationship among the 590 districts in 1861. Clearly the inverse relationship is significant and strong, but, when one proceeds a stage further and considers the regression residuals, a striking pattern of regional clustering emerges. Infant mortality is higher than would be predicted given a knowledge of life expectation at birth in Norfolk, Lincolnshire, the East Midlands, and the East Riding of Yorkshire, but is lower in the west of Wales and the extreme northeast and north of England (Figure 5). In the remainder of England and Wales $1q_0$ is either effectively predicted by $e_0$ or the residuals are scattered in an isolated and random fashion. This pattern also gives encouragement to our construction of national model life table systems.

Bideford, and Holsworthy (average $e_0$ and $1q_0$ in 1861, 52.04 and 0.088). The 6 Lancashire districts were Salford, Manchester, Oldham, Bury, Rochdale, and Haslington (average $e_0$ and $1q_0$ in 1861, 35.13 and 0.180).

20 Little seems to be known about variations in case fatality rates, but, in two populations subject to similar disease patterns, the one living in an urban environment may experience the higher case fatality rate and thus higher mortality. Although certain diseases were more prevalent in urban areas (the effect of population density on measles, for instance), the effect of high case fatality rates would have exacerbated the situation. In those populations with the very worst mortality levels both morbidity and case fatality rates may have been higher.
since, for most of the populous districts, $1q_o$ and $e_0$ were closely related.\textsuperscript{21}

It is not at all obvious why there should have been such differences between the east and the west, but an analysis of causes of death may help to resolve the matter. Table 5 is compatible with Table 4. It employs the same method on data drawn from six registration districts round the Wash, on the east coast of England, and from six districts along Cardigan Bay in Wales. Those in the east all had high positive residuals in Figure 5 and those in the west high negative ones. In the east 23 percent of all

\textsuperscript{21} In 590 districts the relationship between $1q_o (Y)$ and $e_0 (X)$ in 1861 was $Y = 0.3528 - 0.0049X (r^2 = 64.90\%$, significant at 99.99\%).
Fig. 7 The Relationship between Infant Mortality (1q0) and Log. Population Density in Persons per Square Kilometer, England and Wales, 1861 (based on registration district data with both sexes combined).

atation about the regression line. In the low population density rural areas $e_0$ does tend to be higher than in the high density urban areas, but there are clearly also rural areas in which $e_0$ is lower than one would expect from a knowledge of their population densities. Our comparison of east and west in Table 5 illustrates some of the possible reasons for finding relatively poorer life chances in certain low density areas. There is also variation among the urban districts most, but not all, of which have an $e_0$ less than 41. Similar comments could be made regarding the association between 1q0 and population density. There is a statistically significant association, but the degree of variation suggests that simple differences between urban and rural environments are either not entirely captured when measured via population density, or that
environmental conditions are rather more complex in their influence on mortality. Neither of these findings is surprising, but they do caution us against overhasty and oversimplified explanations.24

It has been argued by McKeown that medical science and the medical profession in general had a relatively limited influence on mortality in the nineteenth century. This being so, one would not expect a population well served by doctors to be any more healthy or to have any better life expectation than one with only limited access to members of the medical profession. Figure 6 shows that the well-supplied areas comprise parts of London and surrounding districts together with small southern towns, resorts, and spas. In London in 1861 there were 805 persons per doctor, but 304 in the St. George-Hanover Square district and 4,379 in Bethnal Green. Doctors were thinly distributed, especially in Lancashire and south Staffordshire, and in the most remote rural areas, particularly in Wales and in the rural hinterlands of well-supplied market towns.25

There appears to be no statistically significant link between mortality (either \( e_0 \) or \( 1q_0 \)) and the population:doctor ratio shown in Figure 8. But it is difficult to believe that morbidity and mortality could not have been ameliorated in, for example, the Lancashire cotton towns, if there had been more doctors to inform employers and local councils of the dangers to public and personal health, or in the East End of London, where few doctors worked and even fewer lived.26

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24 In 590 districts the relationship between \( e_0 \) (Y) and population density in persons per square kilometer (X) was \( Y = 56.4460 - 5.5433 \log X \left( r^2 = 46.59\% \right) \), significant at 99.99\% and between \( 1q_0 \) (Y) and X it was \( Y = 0.0762 + 0.0272 \log X \left( r^2 = 30.44\% \right) \), significant at 99.99\% in 1861. See Farr, Vital Statistics (London, 1885), 172–176, for analogous calculations for 1841–1850 and 1851–1860 using death rates. See also, Friedlander et al., “Socio-Economic Characteristics.”

25 The figures for population:doctor ratios are taken from the 1861 population census and relate to the total population and the number of physicians, surgeons, and apothecaries in that year. See Woodward, “Medicine and the City,” 69–70; M. Jeanne Peterson, The Medical Profession in Mid-Victorian London (Berkeley, 1978); Ivan Waddington, The Medical Profession in the Industrial Revolution (Dublin, 1984). Some 45% of the 768 physicians lived in just 3 districts (Kensington, St. George-Hanover Square, and Marylebone). In most of the East End districts there were 1,500 persons per doctor, but most of the doctors were surgeons or apothecaries.

26 The relationships of \( e_0 \) and \( 1q_0 \) to persons per doctor yield \( r^2 \) of 0.55\% and 1.15\%, respectively. Neither are significant. Marilyn E. Pooley and Colin G. Pooley, “Health, Society and Environment in Victorian Manchester,” in Woods and Woodward, Urban Disease, 148–175, deals with the case of Manchester in some detail. See also, Cheney,
eliminated, whatever remains, however, improbable, must be the truth) should be avoided in historical analysis: it rejects the need for strong positive evidence in order for the improbable to become acceptable. It is particularly unfortunate that this logic led McKeown to stress the importance of nutrition so forcefully. More recent research has demonstrated that the relationships between nutrition, morbidity, mortality, and fertility are very complicated. Extremely poor diets will exacerbate morbidity, increase mortality, and depress fertility; but improvements only in moderately poor nutrition will not on their own lead to dramatic changes in mortality. Second, the demographic analysis presented here stresses both the diversity of mortality patterns between environments (the composition effect) and changing age-related conditions (the cohort effect). It highlights new clues which should change our view of what was possible in the nineteenth century, especially in large towns and cities.\textsuperscript{30}

\textsuperscript{30} See the contributions to \textit{Hunger and History: The Impact of Changing Food Production and Consumption Patterns on Society}, a special issue of the \textit{Journal of Interdisciplinary History}, XIV (1983), 199–534, for a comprehensive survey of the debates and evidence relating to the effects of variations in nutrition on demographic and social history.
Diagnosis, Death, and Diet: The Case of London, 1750–1909

There are two basic approaches to historical study: the general and the particular. Most historians are more gifted at one, yet can appreciate and be stimulated by the other; the best history successfully combines the two perspectives. In interdisciplinary history above all, the two approaches meet in a complex and delicate relationship. The modern emphasis on statistical methods has added a further dimension of both vision and error to the historian’s craft. The history of mortality is one area where the use of statistical methods seems irresistible, and where the pitfalls of so doing are numerous.

When McKeown published his classic *Modern Rise of Population* in 1976, the novelty of the statistical survey technique, authoritatively applied by a medically qualified historian, gave great stimulus to historical mortality studies. The social history of medicine was a relatively new field, and the flaws in the technique were not immediately apparent. In the last decade, medical history has developed rapidly, and although the importance of McKeown’s work remains unchallenged, the validity of statistical surveys in furthering our understanding of historical mortality must be seriously questioned. In a recent article, Matossian used the statistical survey technique in an attempt to emphasize the importance of diet in mortality by showing a causal relationship between fungus contaminated cereals and declining mortality in eighteenth- and nineteenth-century London. Matossian’s essay is an excellent example of the misuse of eighteenth- and nineteenth-century statistics. The time has clearly come to draw attention to the shortcomings of these statistics, so that English mortality studies may be placed on a sounder footing.¹

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¹ Thomas McKeown, *The Modern Rise of Population* (London, 1976). For recent work critical of McKeown, see Robert Woods and John Woodward (eds.), *Urban Disease and...
than a variety of non-specific bowel and heat-induced disorders in young infants.\textsuperscript{7}

Although summer bowel complaints probably reflect a range of food and general hygiene problems, the role of bread and cereals in infant mortality throughout the year should be considered, in view of Matossian’s thesis. Adulterated bread was certainly considered a cause of infant mortality in the late eighteenth century. With the addition of chalk, alum, and bone-ashes to London bread, it would be surprising if vulnerable infants were not in one sense poisoned by being weaned on pap made with this bread. The substitution of potato flour for these additives in the late eighteenth and in the nineteenth centuries made for a healthier “wheaten” loaf. But if adulterated bread may be considered a possible element in deaths from convulsions, it is most unlikely that ergot of rye had a role to play in this mortality. By the 1780s, when deaths from convulsions began to decline, rye had for some time ceased to play any considerable role in the diet of London, and indeed of southern England. Rye flours continued to be used in some parts of the English counties throughout the nineteenth century, but there is no evidence of major local variations in convulsion mortality coinciding with rye areas.\textsuperscript{8}

Regional consumption of rye apart, the evidence for the presence of ergot in England is doubtful. The grounds for linking ergot with infant convulsions are even more debatable. Convulsive ergotism is a familial affection. Had convulsive ergotism been responsible for infant convulsion deaths in England on the scale which Matossian claims, the adult population would not have been unaffected, and there would certainly have been positive contemporary documentation. No such documentation exists.


Further, the regularity of the curve of infant convulsions deaths is at odds with the nature of egotism. Ergot is above all an epidemic infection: its presence in rye demands very particular weather conditions, which may not occur every year. In fact, rye becomes dangerous only in exceptional years. Quite apart from the disappearance of rye from the southern English diet in the eighteenth century, the suggestion that ergotism was responsible for an annual toll of infant deaths is untenable.9

**CONSUMPTION** The analysis of the pattern of deaths from consumption in eighteenth- and nineteenth-century London is both simpler and more complex than that of convulsions. On the one hand we are dealing with a recognizable disease, rather than with a symptom; on the other hand, the etiology of the disease is very complex. Nor was the historical recognition of pulmonary tuberculosis straightforward, and the bills of mortality are exceptionally unreliable with respect to this disease. The term “phthisis,” and hence also “consumption,” in its Greek derivation means simply “wasting,” and it was in this sense that it was used, during the eighteenth and much of the nineteenth centuries, to cover a wider range of diseases than pulmonary consumption alone. In later eighteenth-century veterinary texts, it can be found as a synonym for pneumonia. In the early nineteenth century five types of phthisis were identified, deriving from Cullen’s listing of five causes of phthisis: haemoptysis, suppuration of the lungs resulting from pneumonia, catarrh, asthma, and, the commonest, tubercule. Even by the end of the nineteenth century, old habits lingered. In 1892, for instance, the example of phthisis was used to demonstrate the unreliability of the Registrar-General’s statistics: Wales showed a very high phthisis mortality partly because the proportion of uncertified deaths was high, but also because “every death from wasting disease is apt to be certified as ‘consumption’.” But pulmonary tuberculosis was not the only disease

The difficulties which historians face in working on a disease like consumption are considerable. The detailed local investigations by medical officers of health and the central medical department, which provide most of our information on the communicable diseases for this period, are virtually absent for tuberculosis before the infectiousness of the disease was accepted in the early 1890s. Even then, specific, rather than general, epidemiological studies are rare. Medical officers seeking to study the disease met with formidable obstacles: patients gave false addresses or moved house to escape inquiry and publicity. Compulsory notification of consumption was not achieved before 1912, partly because of fears that official recognition would deprive patients of the means of livelihood.\(^{17}\)

Mortality figures remain our best guide to the incidence of pulmonary tuberculosis in a given area, but they should never be used without qualification, and they should always be considered in their local context. Local climate, demography, diet, social and sanitary conditions, occupational and institutional structure, all have a bearing on the interpretation of historical tuberculosis mortality. In wealthy urban areas, for example, registered mortality is more likely to reflect the incidence of the disease on the upper-class residential population: servants who fell terminally ill returned to their homes, often in the country, to die. Poor urban areas with many common lodging houses, on the other hand, often attracted a tubercular population whose infection had been acquired elsewhere: the lodging house was the last resort before the workhouse. The presence of hospitals could also swell local death rates from the disease, whether obviously as in Chelsea, the location of the Brompton Hospital, or unobtrusively as in St. Marylebone, where private hospitals and nursing homes became increasingly numerous in the Victorian period.\(^{18}\)


\(^{18}\) MOAR (St. Giles, 1863), 11; MOAR (Westminster, 1898), 47; Arthur Newsholme, Fifty Years in Public Health (London, 1933), 244–260.
The pattern of decline of mortality from respiratory tuberculosis before about 1840 is too uncertain to allow for chronologically precise explanations of its cause. Later nineteenth-century contemporaries were convinced that the disease was on the decline, just as their forebears, at the turn of the eighteenth century, had been convinced it was increasing. Consumption remained a major killer throughout the nineteenth century: in the 1890s the disease was still recognized to be a serious problem, responsible for some 70,000 deaths a year nationally. Overall, however, the reduction in tuberculosis mortality by over one third in the late nineteenth century is too large to be accounted for solely by greater accuracy in diagnosis and the disguising of tuberculous deaths.\textsuperscript{19}

Although the debate over the respective roles of nutrition and housing as contributory factors in causing respiratory tuberculosis continues, caution should be exercised in attempting to resolve the problem of the disease’s decline with blanket explanations. The lesson to be learned from local studies is that a wide variety of factors influenced regional tuberculosis mortality. In the case of London, the apparent sharp fall in tuberculosis deaths in the 1830s is suspect because of the change in methods of death registration, and the extreme unreliability of the bills of mortality in those years. Although it is probable that tuberculosis deaths were decreasing at this time, the available statistics cannot assess reliably the extent or chronology of the decline over the period 1750 to 1909. The very sharp fall between 1831 and 1838, with a further fall in 1841, after which registration procedures were tightened up, and the continuing but much more gentle decline thereafter, strongly suggests the operation of a redistribution factor.\textsuperscript{20}

The same statistical uncertainty bedevils attempts to estimate nutritional patterns in this period. Although it may be puzzling that deaths from pulmonary tuberculosis apparently began to decline in the “hungry half century,” there is little real evidence


tality declined in this period. The diagnostic unreliability of sev-
eral major "cause of death" categories invalidates this type of
statistical survey for the eighteenth and much of the nineteenth
centuries. If Matossian believes that the available statistics permit
London's mortality trends to be explained with "scientific preci-
sion," she is misguided. The suggestion that improved diet was
the significant factor in modern mortality decline is not a new
one, and is one that has been much debated since McKeown's
Modern Rise of Population. Here again, oversimplified explanations
should be treated with care: man did not live by bread alone, and
nutritional history, like the history of disease, cannot be deter-
mined within a framework of preconceived ideas and in that
vacuum in which the determination to be "scientific" so often
places historians. Our knowledge of the relationship between diet
and mortality, and of the historical behavior of different diseases,
can be advanced only by detailed local studies which take account
not only of a wide range of local circumstances, but also of
statistical pitfalls, the etiology of specific diseases, and contem-
porary medical observations.\footnote{It is possible that, overall, medical intervention contributed substantially to mortality. This argument is present in Smith, \textit{People's Health}. For positive evidence on this score, see Irvine Loudon, "Death in Childbed from the Eighteenth Century to 1935," \textit{Medical History}, XXX (1986), 1–41. For discussion of diet and mortality, see, for example, "Hunger and History," a special issue, \textit{Journal of Interdisciplinary History}, XIV (1983), 199–534.}
The Berkshire Conference of Women Historians will award its annual prizes for the best book and the best article on any historical subject written by an American woman and published in 1987. Two copies of each book should be sent to Amy Hacket and Harriet Alonso, 473 Westminster Road, Brooklyn, New York 11218. Three copies of each article should be sent to Toby L. Ditz, Department of History, The Johns Hopkins University, Baltimore, MD 21218. Deadline for submission is February 1, 1988.

The Journal of the History of Ideas is pleased to announce the establishment of the Morris D. Forkosch Prize ($1,000.00) for the best book in intellectual history published each year. The awards committee will favor books which are published in English and which display some interdisciplinary range, demonstrate sound scholarship, and make an original contribution to the history of thought and culture. For further information, contact Ms. Robin Ladrach, Editorial Assistant, Journal of the History of Ideas, Rush Rhees Library, The University of Rochester, Rochester, New York 14627.
residence and one or two additional establishments in the city. The daimyo had to spend half of their time in Edo to “pay attendance upon the Shogun,” but in reality this requirement enabled the Shogun to keep an eye on them—a polite hostage system. Although some of the smallest daimyo probably had only a hundred or so people in residence in Edo, some of the largest had thousands. The largest daimyo, the Maeda of Kaga, maintained a regular staff of about 1,000 retainers, who with their families constituted at least 4,000 permanent residents. When the daimyo himself was in residence, it is estimated that as many as 8,000 people were in attendance. Thus even a conservative estimate of the daimyo population would require adding a few hundred thousand to the population of Edo, and this number does not include servants.³

To estimate Edo’s population, from a base of 600,000 commoners, we would have to add a very conservative estimate of 100,000 for the Shogun’s direct retainers, a minimum of 200,000 in the samurai class from the daimyo establishments, plus an unknown number of servants. Clearly a total of 1 million for Edo’s population is not wild speculation. Edo was certainly much larger than London in 1700 and rivaled it in population in 1800.

Not only was Edo larger than any European city, but both Osaka and Kyoto were larger than the European capitals of Vienna, Moscow, and Berlin even at the beginning of the nineteenth century and were surpassed in size only by London and Paris. After the Battle of 1614/15, Osaka was built to become the commercial entrepôt of Japan. At its peak in 1763, it had a commoner population of 418,537. Even after Edo gradually took over many of the marketing and financial functions of Osaka, the city remained the second largest in the country with a population of 314,370 in 1858. Kyoto, the seat of the emperor, had a population recorded at 410,000 by 1534, and then, as other urban areas grew,

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³ The samurai were originally warriors, but by the Tokugawa period this was the general term for the warrior estate or class. During the Tokugawa period, the daimyo were regional military lords who held domains assessed at 10,000 or more koku (a unit of rice, approximately 5 bushels). The direct retainers of the Shogun were his vassals: a minority of daimyo (judai), the bannermen (hatamoto), and the housemen (gozenin). For information on the samurai, see Kozo Yamamura, *A Study of Samurai Income and Entrepreneurship* (Cambridge, Mass., 1974), 10; Naitō Akira, “Edo no toshi kōzō,” *Edo jidai zushi* (Tokyo, 1975), IV, 169; Toshio G. Tsukahira, *Feudal Control in Tokugawa Japan: The Sankin Kōtai System* (Cambridge, Mass., 1966), 95.
its population fell into the mid-300,000 range from the late 1660s on.^{4}

Japan's urban growth in the seventeenth century was not limited to the three largest metropolises; equally important in the long run was the proliferation of the castle towns in the same period. From the last quarter of the sixteenth century and into the seventeenth, numerous cities sprang up in the Japanese countryside. These originated as the headquarters of major daimyo and were known as castle towns, because at the center of each was the castle, which served as the domain's administrative center. Quartered here were the daimyo's forces, and to supply them there was a need for merchants and artisans. A domain's castle town usually maintained about 10 percent of the domain's total population. The commoner population of the major castle towns ranged from 10,000 up to more than 60,000 (Kanazawa) and, although their commercial activities began to be taken over by smaller towns in outlying areas in the late Tokugawa period, most of these towns remained vital centers. Half of the sixty most populous cities in Japan today originated as castle towns during the late sixteenth century.^{5}

Japan also had a score of ports by the sixteenth century that ranged in size from 10,000 to 50,000, and an even larger number of cities or towns with populations in excess of 5,000. With the establishment of formal shipping routes in the Tokugawa period, the number and size of port towns increased. By the late eighteenth and nineteenth centuries, villages in the countryside had become towns, and towns had grown into cities. After the formation of the castle towns, Japan was no longer dominated by its capital, and two centuries later, with the growth of outlying centers, the castle towns were no longer the only urban areas in many domains. The Tokugawa period witnessed not only the rise of one of the largest cities in the world, but also the urbanization of Japan.^{6}

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6 For discussions of pre-Tokugawa urban development, see Toyoda, Chūsei Nihon shō-
The most important difference between waste disposal in Japan and in the West was that human excreta were not regarded as something that one paid to have removed, but rather as a product with a positive economic value. The night soil of Japanese cities—and Chinese as well—was long used as fertilizer. With the growth of Japan’s population, the limitation of the amount of arable land and the increasingly intensifed use of land to feed the growing population, combined with the relative scarcity of animal wastes and other fertilizers, meant that human waste had a value as fertilizer that far exceeded its value in the West.

Long before Edo was even established, Osaka’s night soil was used as fertilizer for the surrounding farm villages. Most of it was collected, loaded onto ships, and distributed to nearby farm areas. The huge volume brought to the wharves resulted in such an unpleasant odor that there were complaints. In the Tokugawa period, the magistrates deliberated upon these complaints but concluded that “it was unavoidable for the manure boats to come into the wharves used by the tea and other ships.”

In the early years of the Tokugawa regime, boats were sent into Osaka loaded with vegetables and other farm produce which were exchanged for the night soil of the city. But as the price of fish and other fertilizers rose, the value of night soil rose correspondingly, and vegetables were no longer sufficient to pay for it. By the early eighteenth century, with the increase in new paddies in the Osaka area, the price of fertilizer had jumped to the point that even night soil had to be purchased with silver.

The value of human wastes was so high that rights of ownership to its components were assigned to different parties. In Osaka, the rights to fecal matter from the occupants of a dwelling belonged to the owner of the building whereas the urine belonged to the tenants. Feces were considered more valuable and hence commanded a higher price. Generally speaking, the price of fecal matter from ten households per year amounted to between two and three bu of silver, or over one half a ryō of gold. This was a considerable sum since a ryō during much of the Tokugawa period was sufficient to buy all the grain staple one person would eat during a year. Rent was adjusted on the basis of how many tenants there were and was raised if the number of occupants dropped.

11 Wakita Osamu and Kobayashi Shigeru, Osaka no seisai to kötsü (Osaka, 1973), 127.
12 Although urine is usually higher in nitrogen and potash than solid excreta and is
With the rapid growth of Osaka in the seventeenth century, the city government found by mid-century that it had to step in and form guilds to insure that waste disposal was handled properly. As the price of fertilizer rose, by the end of the century farmers from neighboring areas were forming associations for the purpose of obtaining monopsony rights to purchase night soil from various areas of Osaka. Eventually fights broke out over collection rights and prices. In the summer of 1724, two groups of villages from the Yamazaki and Takatsuki areas fought over the rights to collect night soil from various parts of the city. Other disputes arose between the guilds in the city and farmers’ associations, and examples exist for the neighboring provinces of Kawachi and Settsu as well, indicating that this type of conflict was neither a localized nor an isolated event.  

In the three major areas of Osaka, neighboring farm villages held the rights to collect night soil from households, but they either could not or did not want to collect all of the urine. The remainder was left to be collected by shōben nakagainin, literally, urine jobbers. The number of jobbers gradually increased, as did jurisdictional problems. Eventually they created their own association, and in 1772 they paid a fee to the Osaka authorities to establish a kabunakama (guild based on ownership of shares) with the authority to enforce jurisdiction and to set prices. However, the rights to collect the urine from containers left for passersby on the street corners in Osaka were given to an outcast village named Watanabe, but even though the price of urine was lower than for fecal matter, there were constant conflicts over these collection rights. Periodically, other people tried to get these privileges away from Watanabe, but the village managed to maintain its monopoly throughout the Tokugawa period, despite sabotage of its containers, challenges by others to its collection rights, and offers to buy the rights. 

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especially useful as an activator in converting crop residues to humus, it is more difficult to transport and store than solid excreta, which probably accounts for its lower price. For the price of fecal matter, see Watanabe Minoru, Mikaihō buraku-shi no kenkyū (Tokyo, 1965), 297. The value of a ryō was calculated from Yamasaki Ryūzō, “Edo chūki no bukkā dōkō to keizai hendō,” in Harada Toshimaru and Miyamoto Matao (eds.), Rekishi no naka no bukka (Tokyo, 1985), 78.

13 Wakisita and Kobayashi, Osaka, 128.
14 Watanabe, Mikaihō, 292–299. See also Osaka-shi, Osaka shishi, I, 866–868.
main streets in most castle towns were relatively narrow, about twenty-four feet wide, but they were “extremely well maintained and immaculately clean.” The regulations regarding the maintenance of public roads were detailed and infractions were reported. In Tottori, for example, streets had to be cleared and then sprayed with water (which probably reduced the incidence of respiratory disease). In Hirado orders were issued to the effect that all bridges, gutters, and waterways should be repaired, maintained, and cleared; to make certain that this was done, officers of the town were to inspect them constantly. “No corner shall be left uncleaned.” Judged by regulations alone, cleanliness and proper sanitation were of high priority among Tokugawa urban administrators.18

WHY JAPANESE CITIES WERE MORE HYGIENIC THAN EUROPEAN CITIES

Metropolitan sanitation in Japan from the mid-seventeenth through the mid-nineteenth centuries was almost certainly better than in the West in terms of quality and quantity of the water supply and in terms of waste disposal, resulting in a healthier environment for urban populations as measured by the size of the population and mortality rates. But Japanese city life was also more sanitary than that in the West because of various customs concerning hygiene, food, and drink, and because of a lack of domestic animals. Finally, the government played a major role in setting and maintaining standards of sanitation in the cities. Evidence to support these hypotheses comes not only from descriptive material, but from comparisons made by observers from the West who visited Japan, and from the few quantitative measures we have on water quality, mortality, and life expectancy.

As already elaborated, an important difference between Japan and the West was that human excrement was an economic good in Japan, and was carefully collected for use as fertilizer, thus protecting the water supply in all phases, from source to urban pipes, and also preventing people from coming into contact with waste matter while walking on the street or in or near dumping grounds. In contrast, Westerners traditionally relied on pits in the ground, such as cesspools, for the disposal of human wastes, and

18 Murai Masuo, “Hōken-sei no seiritsu to toshi no sugata,” in Morimatsu Yoshiaki et al. (eds.), Taikei Nihon-shi sōsho (Tokyo, 1965), XVI, 128.
the danger of polluting water supplies was ever present. Even in the 1880s, Cambridge, England, was described as "an undrained, river-polluted cesspool city." In the nineteenth century, "Leicester was typical of many towns in the way it tackled the problems of excrement removal. At mid-century it had almost 3,000 uncovered cesspits, covering 1¼ acres." Only by the end of the century had it managed to convert to a system of pails, which put an end to seepage into the sub-soil.19

Much has been made of the English invention of the water closet, but in the early years this system caused more problems than it solved. First, it required both a water supply and sewer system that could safely supply and remove the large quantities of water that the system used. When the water closet was first invented, Londoners flushed their wastes into the Thames, thinking that at last they were rid of a nasty problem in their houses. What they did not realize for decades was that the cause of the epidemics of infectious disease sweeping the city was the flushing of sewage into the upper Thames, since much of the city’s water was taken from it downstream. Furthermore, faulty drains caused sewer gases to waft up into homes, and people with fixed basins in their bedrooms often had to cover them with towels at night, a rather primitive method of coping with this problem.20

Nor was the new world immune: "As late as 1849, physician John H. Griscom described the unhealthy sanitary state created on Manhattan Island by ‘these thirty thousand cesspools studding it up and down, and filling the atmosphere with nauseous gases’." Stone comments that “even after the introduction of water supply systems, conditions in cities and towns remained unsanitary until properly engineered sewers replaced cesspools, beginning about 1850.” Pinkney’s assessment of Paris in the same year is that there was “one shockingly direct connection” between sewage disposal and the water supply. "The city drew part of its water supply from that main collector sewer, the Seine, and pumped it largely at points downstream from the mouths of sewers emptying into the river. Most of the remainder of the city’s water supply came from sources little more inviting."21

21 John H. Griscom, *The Uses and Abuses of Air* (New York, 1854), 183, as quoted in
unbearable stench caused by the necessities of nature which everybody discharges there daily."26

In contrast, João Rodrigues, a Jesuit, who was in Japan from the late sixteenth into the early seventeenth century, noted that the Japanese

provide their guests with very clean privies set apart in an unfrequented place far from the rooms. . . . The interior of the privies is kept extremely clean and a perfume-pan and new paper cut for use are placed there. The privy is always clean without any bad smell, for when the guests depart the man in charge cleans it out if necessary and strews clean sand so that place is left as if it had never been used. A ewer of clean water and other things needed for washing the hands are found nearby, for it is an invariable custom of both nobles and commoners to wash their hands every time after using the privy for their major and minor necessities.27

By the mid-nineteenth century, conditions in France were no longer so primitive, but the problems with sewage and contamination of the water supply indicated that major sanitation problems remained. In mid-nineteenth century Britain, even royalty was not immune from the effects of inadequate sewage disposal. Prince Albert, Queen Victoria’s consort, is thought to have died of typhoid fever contracted from faulty drains. In contrast, in Japan in the 1870s in the privies in the “better class of private homes,” an American visitor found “less annoyance and infinitely less danger . . . than are experienced in many houses of the wealthy in our great cities.” His description of the privies is similar to Rodrigues’ in the sixteenth century, except that attendants did not clean out the receptacle after every use, but rather it “was emptied every few days by men who have their regular routes.” Morse was taken not only by the cleanliness of Japanese toilets, but also by the amount of artful carpentrywork that decorated them in homes that he visited. He was not describing the

26 Frantz Funck-Brentano, *The Old Regime in France* (London, 1929), 156. Nicholas de la Mare was house steward to the Comte de Vermandois.
27 João Rodrigues, as quoted in Michael Cooper (ed.), *They Came to Japan* (Berkeley, 1965), 221.
toilet facilities of the elite but those in the houses that he visited during his extensive stays in Japan.28

This is not to say that contamination from human wastes did not occur in Japan, but it was probably less frequent than in the West. And custom helped prevent Japanese from becoming ill even when their water supply was not free from impurities. The Japanese customarily drank their water boiled, usually in the form of tea, a custom remarked on by foreign visitors to Japan from the sixteenth century. With the exception of Japanese “pickles,” preserved by fermentation and salt, the Japanese usually ate their food cooked, so that even if night soil was improperly applied as a fertilizer, it was less likely to make everyone sick. Within the household, each member had his own set of chopsticks, rice bowl, and teacup, which no one else used, so that it did not matter much that washing was perfunctory and in cold water. Food served outside the home was frequently finger food, and chopsticks used in restaurants were usually lacquered for easy cleaning and were not put in the mouth as were spoons in the West. By the mid-nineteenth century, disposable chopsticks had come into use.29

Moreover, the Japanese had strong notions about what was dirty and clean, many of which can be traced back to the Japanese native religion of Shinto and its concepts of pollution. Much of the pollution in Shinto is ritualistic, but what is considered polluting and what is purifying are related to contamination and cleanliness. Anything to do with blood, death, and illness—such as childbearing, menstruation, contact with a sick or dying person, and funerals—is considered unclean, and people used to be prohibited from participating in religious rituals, mixing with


29 Cooper, They Came to Japan, 198–199; idem, This Island of Japan (Tokyo, 1973), 263; Engelbert Kaempfer, The History of Japan Together with a Description of the Kingdom of Siam, 1690–92 (Glasgow, 1906), III, 238–240. Although raw fish became popular during the Tokugawa period, particularly in large cities, it was most frequently partially preserved and fermented and not served as slices of fresh raw fish as is common today. In any case, fish came from the sea and would not have been contaminated by fertilizers. For Tokugawa dietary habits, see Watanabe, Nihon shoku seizatsu shi (Tokyo, 1964), 190–269; Hanley, “The Material Culture: Stability in Transition,” in Jansen and Rozman (eds.), Japan in Transition, 454–461.
without their families and males who migrated to the city to work. But had the death rate been significantly higher, the city would not only have had difficulty maintaining a population above a million, but contemporary Japanese would also have noted the high death rates.\(^{36}\)

Tokugawa Japan and Europe in the same centuries had strikingly similar life expectancies. Life expectancy at birth in Europe in 1800 has been estimated as high as thirty-five to forty for some countries, a number higher than in the preceding centuries. Female life expectancy at birth was 42.18 in England and Wales in 1841 and 40.83 in France in 1817 to 1831. A composite figure for life expectancy for males in Western Europe in the nineteenth century, as calculated by the United Nations, is 30.6 in 1840, 41.1 in 1860, and 48.9 in 1900. Female life expectancy is estimated to have risen from 42.5 in 1840 to 52.1 by 1900. These estimates are similar to those we have for Tokugawa Japan.\(^{37}\)

Although the demographic statistics available for Japan and Europe indicate a similarity in the figures, by the nineteenth century Western European nations had available modern technology and by 1850 were well into the process of industrialization with its concomitant rising standard of living. Japan, however, did not begin to use any modern technology until nearly the end of the century. Given the high proportion of Japanese who lived in cities, had sanitation been poor and the level of living low, this combination would have been reflected in high rates of morbidity and mortality.

In fact, Japan seems to have been surprisingly free from the devastating effects of epidemics. The plague never reached Japan, and cholera came only in the nineteenth century when it spread throughout the world. Intestinal worms and enteric infections—those that enter through the mouth and are spread through contamination of food and water—tended to be localized and to

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appear in endemic rather than epidemic form. This situation is what would be expected in a society which used human wastes for fertilizer. It was true for Tokugawa Japan, which would explain the relatively high death rates for children between the ages of one and four or five. Children after weaning were particularly susceptible to these diseases, but if they did not die early, they tended not to succumb to them. However, the fact that the cities with a single water source, such as Edo, Osaka, and Kyoto, did not experience rampant epidemics meant that the water supply must have been generally good. 38

Based on the evidence available on water supply and sewage disposal systems in cities in Japan and in the West from the seventeenth through the nineteenth centuries, I argue that the level of sanitation was higher in Japanese cities than in Western ones during the same period. Not only was Japan able to provide better water and disposal systems, but Japanese customs led to better hygiene and sanitation than did Western modes of behavior. Thus Japan was able to maintain large urban populations from the sixteenth century because sanitation was better and because the Japanese had the control necessary to carry out large-scale engineering projects, to implement various systems connected with water supply and waste disposal, and to see that measures were enforced.

It might well be argued that none of the above systems, customs, or beliefs in themselves would necessarily have created more healthful conditions or been better than any sample found in the West. But the combination of them, combined with near universal application, resulted in more sanitary conditions in the city and more hygienic homes than were the norm in the West, either just prior to industrialization or in the first century of industrialization.

What has obscured the realization that the level of sanitation was higher in premodern Japanese cities than in the cities of the West in the same centuries is the fact that the situation has been the reverse in the twentieth century. In 1985, only 34 percent of Japanese communities had modern sewer systems and the resi-

I canvass the sixteenth-century texts for epidemiological and demographic insights. In the end, I favor the middle ground—somewhere substantially higher than what was common to sixteenth-century Europe, but lower than the crude figure of one-half attributed to Motolinía—closer to what the Franciscan actually wrote: “in some provinces half the people died, and in others a little less.”

**EARLY ACCOUNTS OF SMALLPOX**

Brooks argued that the story of smallpox devastation originated in “Motolinía’s” *Historia*, that it is the “basis (to say no more)” of subsequent descriptions of high smallpox mortality, such as those by López de Gómara and Díaz del Castillo. In Table 1, references are listed by approximate year of composition and their principal sources identified—eye-witness reports, annals, chronicles, and histories. Revisionist skepticism warns against the ready acceptance of later chronicles or histories. Yet we are faced with the reality that over the sixteenth-century many native records were destroyed by Christians in campaigns to eradicate vestiges of indigenous religion. For Spanish writers, on the other hand, there were few opportunities to publish; indeed, the important works by Cortés, López de Gómara, and Sahagún were suppressed for years, although not destroyed. Book publishing began in Mexico in the 1530s, rising to some 200 titles by the end of the century, but publication in New Spain or even Spain was an expensive and uncertain proposition. Some manuscripts on the conquest went through several copyings and enjoyed surprisingly wide circulation without being published. Others were copied by successive generations of local scribes, earlier versions having long since disappeared. Authors reworked, revised, and recopied, as new sources or interpretations appeared. Díaz del Castillo’s two versions of the *Historia Verdadera* is a well-known example. The earliest copy was sent to Spain in 1576, but he continued to revise a second copy in Guatemala until

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3 Toribio de Benavente o Motolinía (ed. Edmundo O’Gorman), *Memoriales o libro de las cosas de la Nueva España y de los naturales de ella* (Mexico City, 1971); idem, *Historia*, 13; in contrast to the embellished Historia: “in most provinces more than half died, and in others a little less.” Elsewhere the text reads: “in many provinces and towns half or more of the people died, and in others less than half, or a third part.” (Memoriales, 294). Provincia refers to a town or city and its surrounding villages and hamlets: “llaman provincias los pueblos grandes, y muchas de ellas tiene poco término y no muchos vecinos” (Memoriales, 245).
Table 1  The Smallpox Epidemic of 1520 in Early Colonial Accounts of the Conquest of Mexico

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>COMPOSITION</th>
<th>PUBLICATION</th>
<th>SOURCES</th>
<th>SMALLPOX EPISODES CITED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vázquez de Ayllón</td>
<td>1520</td>
<td>1866</td>
<td>eyewitness</td>
<td>Narváez' ships</td>
</tr>
<tr>
<td>Cortés⁴</td>
<td>1520s</td>
<td>15205</td>
<td>eyewitness</td>
<td>Maxixcatzin, caciques</td>
</tr>
<tr>
<td>Martyr</td>
<td>1526</td>
<td>15205</td>
<td>Cortés, reports</td>
<td>Cuitlahuatzin</td>
</tr>
<tr>
<td>&quot;Historia de los mexicanos&quot;</td>
<td>1530s</td>
<td>1882</td>
<td>pictographs</td>
<td>Cuitlahuatzin, many died</td>
</tr>
<tr>
<td>Anales de Tlateloco I</td>
<td>1540s</td>
<td>1903</td>
<td>oral tradition</td>
<td>cocoliztli</td>
</tr>
<tr>
<td>Vázquez de Tapia</td>
<td>1540s</td>
<td>1939</td>
<td>eyewitness</td>
<td>more than one-fourth died</td>
</tr>
<tr>
<td>Motolinía Memoriales</td>
<td>1530-40s</td>
<td>1903</td>
<td>eyewitness</td>
<td>black slave, plague, half-died, some provinces</td>
</tr>
<tr>
<td>&quot;Motolinía&quot; Historia⁴</td>
<td>1540s-?</td>
<td>1858</td>
<td>Memoriales</td>
<td>black slave, plague, half or more died, most provinces</td>
</tr>
<tr>
<td>Sahagún⁴</td>
<td>1540-76</td>
<td>1829</td>
<td>native eyewitnesses</td>
<td>pustules, etc.</td>
</tr>
<tr>
<td>López de Gómara⁴</td>
<td>1540</td>
<td>1552</td>
<td>Cortés, Motolinía</td>
<td>two episodes: black slave, Maxixcatzin</td>
</tr>
<tr>
<td>Cervantes de Salazar</td>
<td>1554</td>
<td>1554</td>
<td>López de Gómara, Motolinía</td>
<td>little-by-little, many incidents</td>
</tr>
<tr>
<td>Díaz del Castillo⁴</td>
<td>1550-70s</td>
<td>1632</td>
<td>eyewitness, López de Gómara</td>
<td>five episodes: black slave, great mortality, Maxixcatzin, smallpox weakened warriors, Cuitlahuatzin, Chalco</td>
</tr>
</tbody>
</table>

Anales de Techamachalco
Aguilar
Códice Ramírez
López de Velasco
Durán
Tezozomoc
Pomar
Muñoz Camargo
Sahagún
Anales de Tenochtitlán
Herrera
Chimalpahin
Codex Chimalpopoca
Anales de Tlateloco II

huey zahuati
women, soldiers
Cuitlahuatzin
never seen before
black slave, newness
Cuitlahuatzin
nursing, clothing
black slave; first, worst
pustules, etc.
totoonaliztli
much
many rulers died
many rulers died
bloody ears
phrase “han hecho mucho daño” (has caused great harm), but its significance required no elaboration for the emperor’s advisors or others who were familiar with the demographic catastrophe unfolding in the islands.\(^8\)

The introduction of smallpox among the Aztecs is frequently attributed to a black slave, given the name—Francisco Eguía—in one account. Hallowed by repetition, this story has become something of a trope—unlike the almost ignored tale of “Joan Garrido,” also a black slave, the first to sow and harvest wheat in Mexico. The anecdote of the smallpox-infected slave occurs in most Spanish chronicles of the conquest (those by Motolinía, López de Gómara, and Díaz del Castillo—but not Cortés or Sahagún), in native-mestizo accounts such as the *Relación Geográfica* for Tlaxcala by Muñoz Camargo, the *Codex Ramírez*, Ixtlilxochitl’s *Décima Tercia Relación*, and even in many modern textbook descriptions of the conquest of Mexico.\(^9\)

According to Brooks, reciting the story undermines the credibility of all would-be chroniclers of smallpox in two ways. First, it reveals their dependence on Motolinía, the first-known written account of the tale and, second, “Motolinía’s” own *Historia* is fable, an exercise in “mythopoiesis,” a strained allegory for the biblical account of the Ten Plagues. Brooks reasons that Motolinía needed an “Ethiopian” and that without a theory of contagion, no Spaniard would have made the connection much less remembered a source for the disease.\(^10\)

Whether smallpox was introduced by a black slave, by Cuban Indians, or by both is important only for determining the validity of sources. No historian, not even Brooks, doubts that smallpox reached central Mexico for the first time in April or May 1520,

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8 Vázquez de Ayllón, “Relación,” 42.
with the arrival of the Narváez expedition. As to the theory of contagion, the Spanish vernacular has long provided a simple, but credible notion of how the disease was spread—in a word, pegar (to stick or adhere).\footnote{Prem, “Disease,” 24. The first record of smallpox in the islands is late 1518, nearly six months after the Grijalva expedition departed from Cabo San Antón (Cuba) on May 1, 1518, and shortly before Cortés’ departure for Cozumel on February 18, 1519. Narváez probably left Cuba as the epidemic peaked, arriving at Cozumel, March 4, 1520. Juan de Grijalva (trans. Henry R. Wagner), The Discovery of New Spain in 1518 by Juan de Grijalva (Berkeley, 1942), 26; David Hengie, “When Did Smallpox Reach the New World (And Why Does It Matter)?,” in Paul E. Lovejoy (ed.), Africans in Bondage: Studies in Slavery and the Slave Trade (Madison, 1987), 17; López de Gómara, Historia, I, 14; Manuel Orozco y Berra, Historia Antigua y de la Conquista de México (Mexico City, 1880), IV, 365–367. After three decades of historiographical infighting, Dobyns’ smallpox chronology for the islands and central Mexico remains firmly in place (“An Outline of Andean Epidemic History to 1720,” Bulletin of the History of Medicine, XXXVII (1963), 494–495.)}

From May to September 1520, smallpox spread slowly inland, 150 miles to Tepeaca and Tlaxcala, and then on to Tenochtitlan in September or October. A second eyewitness account of smallpox is reported in another letter to Charles V, that by Cortés dated May 15, 1522, ten months after the fall of Tenochtitlan. Cortés, seeking to justify his transgressions and to position himself to claim vast rewards from the newly conquered lands, provided an exceedingly detailed report of his actions. He described the high regard which native leaders had for him and thereby justified his usurpation of royal authority in appointing native rulers. Cortés wrote that “many chieftains were dying and they wished that by my hand and with your approval and mine others be put in their place.” The many deaths of leaders were due to “the smallpox distemper which also enveloped those of these lands like those of the islands.” I emphasize what Brooks’ paraphrase omitted: Cortés’ explicit comparison of the impact in New Spain with what had happened in the islands.\footnote{Hernando Cortés, Cartas de Relación (Mexico City, 1971), Biblioteca Imperial de Viena Ser. Nov. 16000), 105; a modern translation is given in Crosby, “Conquistador y Pesticide,” 334. Where I translate comprendió as “enveloped,” a popular English edition of Cortés’ letters favors “raged”; see Cortés, Five Letters (New York, 1962), 136. Brooks, “Revising the Conquest,” paraphrase, 20.}

The guessing about how many natives peopled the islands before 1492 continues without relief (ranging for Hispaniola from 8 million to as few as 60,000), but there is widespread agreement that depredation and disease drove the native population to near extinction by 1520. Within a decade of first contact in 1492,
huatzin’s reign and death. The accompanying pictograph showed his enshrinded corpse encircled with tiny globes (ampollas), the symbol for smallpox according to Orozco y Berra. Chimalpahin, the historian of Chalco, also attributed the death to “pustules and ulcers from smallpox” (ampollas y llagas de viruelas [cahuatl]). 19

The evidence that smallpox ravaged the native elites pervades the historical record. The story is significant because the epidemic devastated native diplomatic and military capabilities precisely as Cortes prepared to renew his assault on Tenochtitlan. Chimalpahin, a native historian, reported the smallpox-inflicted deaths of some of the lords of Chalco, using the word cahuatl four times in a brief passage. A Nahuatl-French edition of Chimalpahin’s Séptima Relación first published a century ago, invariably translated cahuatl as “variola” (smallpox). Chimalpahin reported:

Year 2-flint, 1520. Then there was the plague [cahuatl] which caused great mortality. From it died the Huehue Yotzintli Tlayllótlac Teuhctli, Señor of Tzacualtitlan Tenanco Amaquemecan. He ruled thirty-three years.

19 All of the most important sources which mention Cuitlahuatzin’s death agree on the essentials, although none in the same words. Consider the following: Jesús Monjarás-Ruiz, Elena Limón, and María de la Cruz Paillés H. (eds.), Obras de Robert H. Barlow, Tlatelolco: Fuentes e Historia (Mexico City, 1989), II, 263; Tezozomoc, Crónica mexicaotl, 160; Bernardino de Sahagún (eds. Charles E. Dibble and Arthur J. O. Anderson), Florentine Codex: General History of the Things of New Spain (Santa Fe, 1955–75), VIII, 4, 22; idem, Conquest, 103; López de Gómara, Historia, II, 14; Díaz del Castillo, Historia Verdadera, I, 414; “Historia de los mexicanos,” in Icazbalceta (ed.), Nueva Colección, 233; Ixtlixochitl, Décima tercia relación, 13; Anales de Tenochtitlan in Lockhart, We People Here, 279; Chimalpahin (ed. and trans. Silvia Rendón), Relaciones originales de Chalco Amaquemecan escritas por Don Domingo Francisco de San Antón Muñón Chimalpahin Cuauhtlehuaniitzin (Mexico City, 1965), 236.

Notwithstanding the relative late recording of the Tenochtitlan annals, Lockhart concluded that “in all likelihood he [the writer] has given us quite untouched and authentic elements of Tenochca oral and written tradition.” Most authorities think the Annals were written in 1528, but Lockhart argued that internal linguistic evidence suggests the 1540s as a more likely date of composition (Ibid., 39). In any case, this is an early indigenous source based on oral tradition.

The Spanish loan word for smallpox, viruelas, does not enter written Nahuatl until the late sixteenth-century. The imputation of smallpox in this translation is by Lockhart; see also Alonso de Molina, Vocabulario en lengua castellana y mexicana y mexicana y castellana (Mexico City, 1970; orig. ed., 1571), 150v. Other episodes of smallpox in 1520 are discussed in Chimalpahin, Relaciones originales, 158. Although written in Nahuatl after 1620, these relaciones were based on pictographs and oral traditions (Lockhart, We People Here, 39). The interpretation of the pictographic evidence is Orozco y Berra, Historia Antigua, IV, 493, n. 1.
And from this same thing died his adviser. . . . Also of smallpox [cahuatl] died Señora Tlacochuatzin . . . From the same cause died Itzcahuatzin y Tlatquic, from Itzcahuacan, who succeeded in governing thirty-five years and his own son, the said Necuametzin [also died of smallpox] . . .

Díaz del Castillo attested as well that the lord of Chalco died of smallpox, but López de Gómara only noted the death without stating the cause. Cortés scarcely mentioned the Chalco incident, and Motolinía ignored it entirely. The absence of comments by Cortés or Motolinía is insignificant because there is ample independent evidence of smallpox striking down many of the native elite.20

Other Nahuatl sources related the epidemic in a single sentence, such as the Anales de Tlatelolco, where some 4,000 words were allotted to the conquest but only two lines to the epidemic: “Then a plague [cocoliztli] broke out of coughing, fever, and pox [cahuatl]. When the plague [cocoliztli] lessened somewhat, (the Spaniards) came back.” In Cuauhtitlan, the entry for 1520 reads: “Then Yohtualtonatiuh was inaugurated. It was in his time that the Spaniards arrived. Both Citlalcoatl and Yohtualtonatiuh died of the smallpox.” Outside the central basin and fifty miles southeast of Puebla in the district of Tepeaca, the surviving copy of the Annals of Tecamachalco, which dates from the 1590s, chronicled the event-of-the-year for 1520 as “very frightful great smallpox” (“cena temahmauhhti yniic mo chiuh huey zahuatl”). This chronicle omitted any mention of conquest or deaths due to war. Smallpox was the event for 1520.21

The best test would be to tally all the Nahuatl annals by whether smallpox is or is not mentioned for 1520. Pending that exercise, it is evident that any survey of native annals and picto-


se pega). Pegar also describes how fire is spread and the means by which one gets vices, customs, opinions, knowledge and, even, jokes. Thanks to the word pegar, by the seventeenth-century, as the theory of communicable disease became respectable among educated Europeans, Spanish folk discourse required little adjustment to explain the transmission of contagious diseases ("Vale también comunicar una cosa a otra. Comunmente se dice de las enfermedades contagiosas.").

Motolinía, like Vázquez de Ayllón and many other eyewitness chroniclers, used pegar to describe the spread of smallpox. To explain transmission from the natives of Fernandina to those of Cempoala, han pegado was chosen. Two decades later, Motolinía’s Memoriales employed the same imagery: smallpox spread to the Indians ("pegar a los indios"). Sahagún and his assistants, translating from the Nahua, described death from smallpox as "the sticky [spreading?] disease" ("la muerte pegajosa") ‘of which many died, but others died only of hunger because no one cared for anyone else.” When the first measles epidemic struck in 1531, Motolinía described it as “jumping” (saltar) from a Spaniard:

... and from him it jumped to the Indians, and if there had not been much advance warning so that they could be told, prepared and even preached that they not bathe or take other remedies contrary to the illness; and with this pleased the Lord so that not as many died as from smallpox; and they called this the year of the small leprosy (lepra) and for the first, the year of the great leprosy.

The notion of the communicability of disease might not enter formal European discourse until the middle of the sixteenth-century, but, as early as 1431, the imagery was circulating in a Spanish medical manual written in the vernacular to facilitate its dissemination. Later, pegar appears frequently in the writings of the first conquistadores of New Spain.

26 Brooks, “Revising the Conquest,” 26. Nebrija, Vocabulario de Romance, s.v. "contagión"; Real Academia Española, Diccionario de la lengua castellana (Madrid, 1726–39), s.v. "pegar." Cortés used se pegó to describe how pestilence was inflicted upon Spanish judges who were sent in 1530 to inquire about his conduct and thus was spread to New Spain (Cartas, 276).
27 Motolinía, Memoriales, 30.
28 Alonso de Chirino (ed. Maria Teresa Herrera). Menor Daño de la medicina de Alonso de Chirino, edición crítica y glosario (Salamanca, 1973); completed before 1431, Chirino’s work
Also mistaken is the argument that Motolinía simply wrote to emphasize parallels between the suffering of the chosen people in Egypt and the natives in New Spain. Motolinía recounted the Ten Plagues, but then, in a passage expunged from the Historia, challenged popular Spanish beliefs by contrasting the plagues of biblical Egypt with those of contemporary New Spain:

Well considered, there are differences, great differences, between these plagues and those of Egypt. First, in only one of those [of Egypt], and that in the last, were there deaths of people; but here, in each of these there have been many deaths. Second, in each one of the houses there remained someone to mourn the dead, and here, of the plagues already described, many houses were left abandoned, because all their occupants died. Third, in Egypt, all the plagues lasted only a few days, and here, some a very long time. Those, by the commandment of God: most of these by the cruelty and depravity of men, although God permitted it.29

The Franciscan was not guilty of mythopoeisis, as Brooks suggested. Motolinía engaged his Catholic readers’ religious beliefs—that the natives’ afflictions were due to God’s wrath—then he disputed the commonplace thesis of conquest as fulfillment of biblical prophesy by emphasizing the vast differences between the plagues of Egypt and those of New Spain. Historians who traffic in English translations miss the subtlety of Motolinía’s argument because they, like Brooks, favor the Historia (first published in 1858; first English edition 1949) over the Memoriales (first published in 1903; no English translation). Confusion reigns because

survives in six slightly varying copies. Chapter VII examines diseases that stick (“De las enfermedades que se pegan”) and places smallpox among them, 34. Vázquez de Ayllón, “Relacion,” 42. Translated as “muerte pegajosa” by Alejandra Moreno Tosanco in Daniel Cosío Villegas (ed.), Historia General de México (Mexico City, 1976), II, 9–10. Other sixteenth-century writers who used pegar are: Díaz del Castillo to describe the transmission of mororra (Historia Verdadera, II, 263); Diego Muñoz Camargo (ed. Rene Acuña), Descripción de la Ciudad y Provincia de Tlaxcala de las Indias y del mar océano para el buen gobierno y enoblecimiento dellas (Mexico City, 1981; reprint ed.), 35v: “las viruelas que trujo y pego”; Herrera y Tordesillas, Historia General, Decada II, libro 10, cap. IV (398): “las viruelas pegándose con los indios”; Códice Ramírez; Francisco Cervantes de Salazar (ed. Manuel Magallón), Crónica de la Nueva España (Madrid, 1971, Biblioteca de Autores Españoles vol. 245), II, 98; Juan López de Velasco, Geografía y descripción universal de las Indias (Madrid, 1971), 14.

29 Motolinía, Memoriales, 30.
In contrast, the smallpox which struck Amerindians, adults as well as children, was severe and often fatal. The impact of smallpox in New Spain was wholly unlike that in Spain. In Spain, it was a disease of childhood, whereas in New Spain the attack of 1520 struck all ages, including many native leaders. Spanish eyewitnesses compared the outbreak with what had happened in the islands, not with anything in Spain or Europe. In New Spain, unlike Spain, smallpox was a lethal pestilence. If we assume that children made up one-third of the native population, then the crude rate of smallpox mortality among the natives would start at three times the rate for European populations that were subject to regular outbreaks of the disease. Among Amerindians, the absence of care and caretakers propelled smallpox mortality to catastrophic levels, but genetic factors probably played a role as well.

**SMALLPOX MORBIDITY AND GENETIC DIVERSITY**

Genetic immunity is a common explanation for the enormous difference in death rates between Europeans and Amerindians, but there is no proof for this hypothesis. Few genetic differences distinguish new-world populations from the old, and none has a demonstrated advantage against the smallpox virus. Genetic diversity, rather than immunity, may be the key, as Francis Black, a viral epidemiologist, recently argued. Human geneticists reported that Amerindians (along with Polynesians and New Guineans) are unusually homogeneous genetically. The smallpox virus adapts quickly to a host’s immunological response—not mutating into a new strain, but rather preparing for battle with other hosts of nearly identical genetic makeup.

Field research on measles is the most convincing. Measles acquired from a member of one’s family tends to be more virulent than that acquired from a stranger. According to Black, “virus grown in one host is preadapted to a genetically like host and thereby gains virulence.” The genetic key to successfully defending against an attack of virulent smallpox is the production of histocompatibility antigens. Unfortunately, in this regard, Amerindians show only one-sixty-fourth the genetic diversity of Africans or Europeans. The odds worsen when exposure is simultaneous and from multiple sources, particularly from members of one’s own family. The close living quarters, described by López de Gómara and Sahagún, would heighten virulence as the smallpox spread through families and compactly settled communities.37

CARE Whatever geneticists ultimately teach historians about immunity or diversity in explaining the virulence of the disease, the role of social agency is also important. We know, and the Spaniards knew, as Chirino’s medical manual made clear, that nursing reduces smallpox mortality. Whereas Europeans possessed no herbs, antibiotics, or prophylaxes, they, unlike the natives, understood that chances of recovery improved with care—water, food, and clean, warm clothing. What astonished Spanish eyewitnesses of this first epidemic was that it struck adults as well as children. In reaching everyone, the attack left the population without caregivers or nurturers, a fact frequently noted in both Spanish and Nahuatl chronicles. Motolinía’s Memoriales recounted the lethal effects of this horror: “because they all fell ill at a stroke, [the Indians] could not nurse one another, nor was there anyone

Native annals, unlike Spanish chronicles, recorded the most important events for each year. Before 1519 native annals reported pestilence or famine only when devastation was prolonged, often for a year or more. In the century before European instruction (1420–1519), the Annals of Cuauhtitlan reported seven famines and two epidemics—all multiyear phenomena. Then, for 1520, the most notable event was smallpox, when the death of two leaders from the disease was chronicled. Since smallpox outbreaks remain in any one place only for a couple of months, it should be surprising to find smallpox recorded. Yet, 1520 is often named the year-of-the-pox in native annals, such as the Annals of Tlatelolco and the Annals of Tenochtitlan. One of the most interesting annals, the Códice Telleriano Remensis, is rendered useless by the loss of pictographs for 1516 through 1527. Of the surviving pictographs for the postconquest period, 1528–60, this Códice recorded four epidemics: measles (1531), smallpox (1538), a great mortality (“una gran mortandad,” 1544–45), and mumps (1550).

In Nahuatl texts written in the Roman alphabet, the horror of smallpox is told again and again. The longest native account is in Sahagún’s monumental ethnohistorical treatise, the General History of the Things of New Spain. A distillation of testimony of Nahuatl leaders and informants in three towns, the clinical, yet melancholic descriptions have made this one of the most widely cited Nahauatl texts on the conquest. English translations are available in three editions. The first, published in 1955 by Dibble and Anderson, is often cited in extenso. Brooks used the second translation, published in 1975. I favor a third by Lockhart (1993), the first to offer English translations of both the original Nahuatl (probably completed in 1555) and the accompanying Spanish gloss (written before 1586). The most informed, comprehensive account of smallpox epidemic is in the

Twenty-ninth chapter, where it is said how, at the time the Spaniards left Mexico, there came an illness of pustules of which many local people died; it was called ‘the great rash’ (smallpox).


44 Lockhart, We People Here, 259, 279; José Corona Nuñez (ed.), Antiguiedades de Mexico: Basadas en la recopilación de Lord Kingsborough (Mexico City, 1964–67), I, 42–43; Bierhorst, History and Mythology, 79.
Before the Spaniards appeared to us [again], first an epidemic broke out, a sickness of pustules. It began in Tepeihuitl ['which is at the end of September,' according to the accompanying Spanish gloss]. Large bumps spread on people, some were entirely covered. They spread everywhere, on the face, the head, the chest, etc. (The disease) brought great desolation; a great many died of it. They could no longer walk about, but lay in their dwellings and sleeping places, no longer able to move or stir. They were unable to change position, to stretch out on their sides or face down, or raise their heads. And when they made a motion, they called out loudly. The pustules that covered people caused great desolation; very many people died of them, and many just starved to death; starvation reigned, and no one took care of others any longer.

On some people, the pustules appeared only far apart, and they did not suffer greatly, nor did many of them die of it. But many people’s faces were spoiled by it, their faces and noses were made rough. Some lost an eye or were blinded.

The disease of the pustules lasted a full sixty days; after sixty days it abated and ended. When people were convalescing and reviving, the pustules disease began to move in the direction of Chalco. And many were disabled or paralyzed by it, but they were not disabled forever. It broke out in Teotlco, and it abated in Panquetzaliztli. The Mexica warriors were greatly weakened by it.

45 Lockhart, *We People Here*, 180, 182; Sahagún (eds. Dibble and Anderson), *Florentine Codex*, XII, chap. 29 (1st and 2d eds.). The fact that Brooks “cannot recall one [historian] who quotes the qualifications in the second paragraph” is due to faulty memory and flawed transcription (“Revising the Conquest,” 28, n. 40). He omitted text and erased the division between paragraphs two and three. Crosby and Padden quoted paragraphs one and two in their entirety. They omitted paragraph three, perhaps because it seemed less important in the translation from which they worked, but they are in good company. The sixteenth-century Spanish digest of the Nahuatl text omitted all but the first sentence of paragraph three (Códice florentino, III, book 12, chap. 29, fol. 53). See Crosby, “Conquistador y Pestilencia,” 336; Robert C. Padden, *The Hummingbird and the Hawk: Conquest and Sovereignty in the Valley of Mexico, 1503–1541* (New York, 1970), 206; Miguel León-Portilla (ed.), *The Broken Spears: The Aztec Account of the Conquest of Mexico* (Boston, 1962), 93.

Both the English and Spanish translation of the last line of paragraph three changed greatly over the years and with it the significance of the passage for understanding the impact of smallpox. The 1955 edition by Dibble and Anderson (XII, 81) reads: “Then the Mexicans, the chieftains, could revive.” A second edition published in 1975 (cited by Brooks) favors a translation with broader demographic implications (XII, 83): “At that time the Mexicans, the brave warriors were able to recover from the pestilence.” Then, in 1993, Lockhart’s translation elicits nuances unnoted by earlier philologists: “The Mexica warriors were greatly weakened by it.” This reading fits neatly with Díaz del Castillo’s account of smallpox-weakened warriors encountered on the Spaniards’ return to Texcoco in January 1521 (*Historia Verdadera*, I, 438). Likewise, Muñoz Camargo (*Descripción de la
demographic catastrophe of the century for the Nahuatl-speaking people of central Mexico.\textsuperscript{52}

Consensus is emerging on the scale, causes, and consequences of the demographic disaster which struck sixteenth-century Mexico. There is agreement that a demographic catastrophe occurred and that epidemic disease was a dominant factor in initiating a die-off, beginning, in Mexico, with smallpox in 1520. But the role of disease cannot be understood without taking into account the harsh treatment (forced migration, enslavement, abusive labor demands, and exorbitant tribute payments) and ecological devastation that accompanied Spanish colonization. Killing associated with war and conquest was clearly a secondary factor, except in isolated cases, such as the deliberate destruction of Cholula or the leveling of Tenochtitlan.

A fair-minded cross-examination of the broad range of primary sources for the epidemic of 1520 leaves little doubt that smallpox swept throughout the central Mexican basin, causing enormous mortality. The epidemic ranked with the deadliest disasters that native annals customarily recorded. Whether the fraction of smallpox deaths was one-tenth or one-half, we have no way of knowing, but from my reading of the texts discussed here, the true fraction must fall within these extremes, perhaps near the midpoint.

If we leave aside the controversy over the size of Amerindian populations at contact, there emerges a broad agreement in the Spanish and Nahuatl narratives and in the patterns of decline sketched by historians.

For historians who abide quantification, expert estimates point to overall levels of demographic destruction in sixteenth-
century central Mexico exceeding 50 percent, probably ranging beyond 75 percent, and even topping 90 percent in some large regions such as the tropical lowlands. Vociferous debates over population sizes often overlook similarities in the scale of demographic collapse. To reduce historiographical uncertainty further will require much additional, careful sifting of archival and archaeological evidence—tasks which, in recent decades, few seem inclined to undertake.\textsuperscript{53}

In the meantime, I find convincing the testimony of Licenciado Francisco Ceynos, who sums up the opinion of many enlightened sixteenth-century Spanish observers. Ceynos, after five years as fiscal on the Royal Council of the Indies, arrived in Mexico in 1530 to sit on the Real Audiencia of Mexico City. A royal judge (oidor) for more than thirty years, he fought against the widespread practice of enslaving Indians and against the extreme labor and tribute burdens common in that era. On March 1, 1565, he completed a lengthy recommendation on colonization policies suitable for newly conquered regions. As preamble he reviewed briefly the demographic tragedy of Spanish colonization in Mexico:

\dots and it is certain that from the day that D. Hernando Cortés, the Marquis del Valle, entered this land, in the seven years, more or less, that he conquered and governed it, the natives suffered many deaths, and many terrible dealings, robberies and oppressions were inflicted on them, taking advantage of their persons and their lands, without order, weight nor measure; \dots the people diminished in great number, as much due to excessive taxes and mistreatment, as to illness and smallpox, such that now a very great and notable fraction of the people are gone, and especially in the hot country.\textsuperscript{54}

We do not know what number, percentage, or ratio that Ceynos had in mind for "grandes muertes," "gran cantidad," or "faltó


as measured by the stature of young adults, could deteriorate during periods of putative economic expansion. Measurements of stature appear to capture a way to assess shortcomings in the output and distribution of economic, and specifically nutritional, resources. Anthropometric historians have expanded upon this implication by suggesting that the poorer nutritional status can be associated also with survivorship prospects. Poorer nutritional status, as revealed by lower adult height, is taken to signal a higher risk of death. Moreover, in the view of some of these scholars, heights provide a way to make comparisons about health status across countries and time. This article examines a fresh body of manuscript information about the anthropometric status of a large group of British males measured during the 1860s. The evidence is used as an opportunity to review and to challenge some of the assumptions most often made in anthropometric histories.2

Anthropometric research is based on the recognition of a triangular relationship among food intake, demands for energy, and growth. Calories consumed by an individual provide the energy needed for metabolic processes, work, recreation, disease resistance, and growth. Since metabolic processes make a prior claim, the other claimants may suffer when caloric intake is insufficient to sustain all of these activities at their maximum levels. Shortfalls in final height, compared to the height potential of a population, are taken to reflect prior nutritional stress, and stress is equated with malnutrition.

According to nutritionists, the proportion of potential height that an individual achieves is determined by net nutrition and by the composition of the individual’s diet. Maximum height would be achieved by an individual whose caloric intake regularly equaled or exceeded the demands of metabolism, growth, work, and other claimants, and whose diet included large quantities of protein-rich foods, especially meat and dairy products, which promote growth. Although it is widely recognized that certain foods are rich in protein—for example, some 38.4 percent of the

energy in beef is protein—protein needs amount to only 10–15 percent of the energy supply, and less than 10 percent among adults whose energy intake is high. Thus, it is useful to distinguish the composition of diets that promote growth, which should be rich in protein, from diets that satisfy present-day nutritional recommendations, in which protein needs may be high or low. Many foods other than meat and dairy products also supply generous allowances of protein energy; for example, wheat flour furnishes about 13.2 percent of its energy in protein. Protein needs can be met by diets poor in meat and dairy products, but they can be surpassed, and growth in infancy and childhood promoted, by diets particularly rich in such high quality protein foods.3

Diets are often divided into groups of the “good” and the “bad.” In anthropometric histories, good diets are those that result in rising heights, or in heights that are stable at a high level. For example, eighteen-year-old entrants to Harvard University from private schools (that is, schools charging tuition, indicating the high socioeconomic status of their pupils) averaged 180.59 cm in the 1930s, and did not gain in height between then and the 1950s, whereas their public school counterparts did gain significantly in height.4 The implication is that the diet of wealthy Americans in the 1910s and 1920s, when university students of the 1930s were growing up, allowed their height potential to be achieved, whereas diets in other socioeconomic groups did not, for some decades yet, allow them to reach their potential. Bad diets are those that result in low heights, compared to the stature achieved by counterparts in other cohorts, or in declining heights.

Lower heights can be reached along two bad-diet paths. On one, they are achieved because caloric intake fails to match the demands of metabolism, growth, work, and other claimants, especially at moments in the life cycle of particular importance for growth. On the other, they are achieved by a change in dietary composition, and most efficiently by a shift away from protein-rich foods. Shortfalls in height may actually reflect prior dietary composition rather than nutritional stress.

body of information that formed the basis for a monograph in which he divided the individuals about whom he had received reports into groups based on place of birth; averaged their heights and weights, converting those to equivalents without clothing or footwear; and where possible discussed coloring of hair, eyes, and complexion. In extended footnotes, Beddoe reported what he believed he had learned about the physical traits of racial groups, discussing such topics as "the Northamptonshire breed." Although he received some information about men of lower and higher ages than the limits he stated, twenty-three and fifty, Beddoe did not publish those data. Since the manuscript reports from and correspondence with his collaborators have been preserved in the archives of the University of Bristol, individual-level information was extracted from the original records, including height, weight, age, occupation, place of residence at measurement, and place of birth. These characteristics form the basis of this investigation.  

Beddoe made a large number of decisions about how to collect and interpret information, and many of those decisions influence the evaluation and interpretation given here. Altogether, Beddoe published information about 8,583 men born in England, Ireland, Scotland, and Wales. The body of data to be discussed here, referred to as the Beddoe group, deals with 3,498 men—the entire part of the full set that met the standards of data accuracy and selection that I applied. The largest number of records omitted deal with men listed in reports in which the collaborator gave weights only to the nearest stone (14 lbs. or 6.35 kg) or score (20 lbs. or 9.07 kg). Rounded figures suggest inaccuracy in reporting and measurement. Some collaborators acknowledged this possible inaccuracy by writing Beddoe that they had been unable to obtain precise values. Beddoe gathered information about the weights of clothing and shoes worn by subjects, and the height added by footwear. He distinguished subjects who were measured wearing footwear from those that were not. This information, along with other such distinctions, gave Beddoe a way to adjust

10 The phrase "taken indiscriminately as to size" and others like it appeared repeatedly on the forms that Beddoe provided. John Beddoe, On the Stature and Bulk of Man in the British Isles (London, 1870), 73, 75. A Paradox3 version of the file is available from the author on request, as is also a more detailed description of the decisions that Beddoe made about collecting and interpreting data.
values for clothed subjects to the unclothed values that he wanted to consider.  

Like other records used in anthropometric research, Beddoe's group of 3,498 cannot be called a random sample of the overall population. Nevertheless, it is useful to compare the Beddoe group, especially those aged twenty-five to forty-nine, with the entire population. This age group included 2,801 men out of a total population of that age numbering 3.59 million. In the Beddoe group, 21.0 percent were born in Scotland, a significantly larger proportion than the 11.9 percent of all British males of that age living in Scotland in 1866. Since the Scots were, on average, taller and heavier than their counterparts from Wales and England, this distribution pushes the overall average upward. The results that follow consider average heights and weights adjusted for the proportions of men living in England and Wales compared to Scotland.  

Concerning age groups, the distribution is much more similar, as shown by comparing the Beddoe group with averaged proportions from the 1861 and 1871 censuses:

<table>
<thead>
<tr>
<th>AGE</th>
<th>% BEDDOE GROUP</th>
<th>% OVERALL POPULATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>25–29</td>
<td>28.2</td>
<td>25.0</td>
</tr>
<tr>
<td>30–34</td>
<td>22.9</td>
<td>22.3</td>
</tr>
<tr>
<td>35–39</td>
<td>19.3</td>
<td>19.4</td>
</tr>
<tr>
<td>40–44</td>
<td>16.1</td>
<td>18.1</td>
</tr>
<tr>
<td>45–49</td>
<td>13.4</td>
<td>15.2</td>
</tr>
</tbody>
</table>

Although they made no use of the Beddoe manuscripts, Floud, Wachter, and Gregory consulted Beddoe's published study, noting

11 Beddoe, _Stature and Bulk_, 12–113. This total, 8,583, represents my count of the number of unique individuals included in Beddoe's central tables. In presenting those tables, Beddoe sometimes summed up information provided earlier, thereby duplicating it. I have tried to eliminate duplications, but Beddoe's language is not always clear. Statistics concerning lunatics, criminals, and others reported on pg. 113 are excluded from the figure of 8,583, as are men who fell outside Beddoe's age limits.

Heights are available for 3,497 subjects, weights for 3,484, and ages for 3,494. A sample of the excluded reports was analyzed to determine whether, as a group, the reports with rounded figures differ in their overall averages from the reports with more precise figures. Containing 434 individuals, the sample shows only trivial differences in age-standardized height, weight, and body mass index (BMI) from the Beddoe group, considering either the group as a whole or its Scots and English members separately.

12 This comparison and the next are based on data taken from Brian R. Mitchell, _British Historical Statistics_ (Cambridge, 1988), 9, 12–13.
RESULTS The records concerning these 3,498 men differ from the sources most commonly discussed in anthropometric history because they report on stature and bulk not toward the end of growth, around age twenty, but across a wider spectrum of age. Only a few men in the Beddoe group were less than twenty-three or more than fifty; Beddoe specified these limits in order to avoid being misled by men still growing or by those of advanced age who had lost height.\textsuperscript{16} Figure 1 presents crude data for heights among men age twenty-five to forty-nine, whose average height was 170.61 cm (5 ft. 7.17 in.).

Assuming that all individuals were measured in 1866, Beddoe’s subjects age twenty-five to forty-nine in 1866 were born between 1817 and 1841 and grew up in the period 1817–1841 to 1841–1866. According to Floud, Wachter, and Gregory, that period included major differences in nutritional status during growth years and, therefore, in final heights achieved by people who grew up during that time. In the way that they appear in Figure 1, however, Beddoe’s findings give no evidence of such differences. They suggest, to the contrary, that across the period little or no variation occurred in nutritional status.\textsuperscript{17}

Figure 2 provides crude data for weights. At age twenty-five to forty-nine the weights, like the heights, are nearly regular. Across the life cycle, however, weight increased, rising from an average of 64.8 kg (142.6 lbs.) at age twenty-five to 70.0 kg (154.3 lbs.) at age forty-nine. The men in this survey, like men in twentieth-century populations, tended to gain weight with age.

Figure 2 also shows the body mass, or Quetelet index. The body mass index is a standard measurement of weight by height in a simple formula devised by Quetelet, a Belgian statistician: \( \text{BMI} = \frac{\text{kg}}{\text{m}^2} \), where \( \text{BMI} \) = body mass index, kg equals kilograms, and \( \text{m}^2 \) equals height in meters squared. Although other means of combining weight and height values have been developed, the Quetelet index is the one that is used most often and the one most appropriate for this study. It is valuable both as an

\textsuperscript{16} Some of Beddoe’s subjects rounded their ages to numbers ending in 0 or 5, presumably to the 0 or 5 nearest their true age. No attempt has been made to allocate men at these ages because significant numbers of men rounded their age only when they were between ages 30 to 50, a time when growth was complete.

\textsuperscript{17} Floud, Wachter, and Gregory, \textit{Height, Health and History}, 136–137. Some men may have been measured in 1865.
**Fig. 1** Average Heights

![Graph showing average heights over different ages.](image)

**SOURCE**  John Beddoe Collection, Special Collections, University Library, University of Bristol, Bristol.

**Fig. 2** Average Weight and Body Mass

![Graph showing average weight and body mass over different ages.](image)

**SOURCE**  See Figure 1.
born in rural areas (population < 5000) achieved heights and weights greater than men born in towns. For example, their heights averaged 171.04 cm versus 169.54 cm. This difference was more marked for Scotsmen, among whom it was 1.62 cm, compared to 0.91 cm for Englishmen.

By finding the size of places of birth and residence, it is also possible to consider whether body mass varied with population size. Limiting the issue again to men age twenty-five to forty-nine, larger cities tended to produce shorter and slighter men and also to attract shorter and slighter men. The association between the size of birth cities, estimated from 1831 population data, and body mass is small; the same is true of the association with height. Between residence city size, estimated from 1871 census data, and body mass and height, however, the associations are larger. The more populous a residence city was, the more likely were the men in the Beddoe group living in it to be shorter and slighter.20

Since at least half of the men in the Beddoe group had moved away from their place of birth, it appears that the men who moved and those who did not alike sorted themselves by stature and weight into patterns of movement. Larger men seem to have moved to smaller cities in preference to larger cities, and slighter men to have moved in preference to larger cities. Taken in combination with the findings reported above about occupational categories, the implication is that shorter and slighter men migrated to urban jobs in manufacturing. In any case, migration

---

20 In this comparison, no effort has been made to adjust the threshold between rural and urban areas for growth in city size between 1831 and 1871. Population data for 1831 derive from John Gorton, Population of Great Britain According to the Returns made to Parliament in 1831 (London, 1832); those for 1871 from Great Britain, House of Commons, Sessional Papers (1872) LXVI, pt. 1; (1872) LXVIII. Regressing age, 1831 city size, and 1871 city size on body mass for 1,533 men, and on height for 1,538 men, for whom the size of the place of residence in 1831 and 1871 is known, produces these coefficients:

<table>
<thead>
<tr>
<th></th>
<th>BMI</th>
<th>HEIGHT</th>
</tr>
</thead>
<tbody>
<tr>
<td>age</td>
<td>.05015^a</td>
<td>-.00232463</td>
</tr>
<tr>
<td>1831 population</td>
<td>-.000001625</td>
<td>-.000002399</td>
</tr>
<tr>
<td>1871 population</td>
<td>-.000006257^a</td>
<td>-.000008784^a</td>
</tr>
<tr>
<td>constant</td>
<td>21.26027^a</td>
<td>170.23647^a</td>
</tr>
</tbody>
</table>

^aSignificant at the .01 level.

Most of the missing information is for 1831 city size, and most of the missing values are probably villages. The results imply unrealistically low values for Londoners, suggesting that city size was not linearly related to BMI or height.
reduced body mass differentials between the town and the countryside. For men age twenty-five to forty-nine as a whole, the body mass differential dropped from 0.71 when birth locales are divided into rural and urban sites to 0.54 when residence locales are divided on the same grounds. Similar declines occurred in height and weight taken separately.

Comparing place of residence in 1980, the tallest Britons lived in southeastern England; Englishmen were slightly taller than Scotsmen and markedly taller than Welshmen. This difference in height was not true in the Beddoe group. On average, the Scots age twenty-five to forty-nine in the Beddoe group were taller than their English counterparts, by 3.71 cm, and taller than their Welsh counterparts, by 2.72 cm. Table 3 summarizes differences in average height, giving for 1866 both raw averages for the Beddoe group and averages adjusted to the division of population between rural and urban areas. Englishmen have gained height, which is especially noticeable in comparing younger adults in the 1980 sample with men of any age in the Beddoe group. It is less noticeable, however, when comparing older men in the 1980 sample with their counterparts in the Beddoe group, which implies that most of the interim height gain has occurred during the lifetimes of men measured in 1980.

Regarding Scotsmen, Beddoe himself provided three separate estimates of the average height of Scotsmen. The full number of

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Average Heights (Ages 25–49 in the Beddoe Group; All Adult Males in the 1980 Survey)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1866</td>
</tr>
<tr>
<td>Englishmen</td>
<td>169.86 cm</td>
</tr>
<tr>
<td>Englishmen Adjusted (Rural/Urban Division)</td>
<td>169.78</td>
</tr>
<tr>
<td>Scotsmen</td>
<td>173.57</td>
</tr>
<tr>
<td>Scotsmen Adjusted Rural/Urban</td>
<td>173.42</td>
</tr>
<tr>
<td>Highland/Other</td>
<td>173.76</td>
</tr>
</tbody>
</table>

were nearly twice as likely to die in the study period as those 180–184 cm tall (about 6 ft.). This finding has proved of most interest to anthropometric historians. Waaler also examined survivorship by body mass (that is, height-for-weight). His findings for males are reproduced in Figure 5, which shows that low death rates (given in the vertical column in a log scale) are associated with Quetelet index values toward the mean. At the lower ages in this comparison, twenty to forty-nine, the difference in survival prospects is so great as to produce a series of bathtub-shaped curves, in each case with a broad base. In that base area, differences in survival prospects are evident, but they are comparatively small. The differences are large only at the extremes, where individuals had either markedly low or high weight-for-height. For example, a male age 45–49 with a Quetelet index value of 33 was more than twice as likely to die as a male of the same age with a value of 25.23

In order to compare these findings with the Quetelet index values of men in the Beddoe group, consider Figure 6. It shows the range of Quetelet index values that Waaler found to be associated with lower mortality, at the lower bound always a value of 19, and at the higher bound a value rising with age from 27 to 30. Two curves appear within that range. One shows the distribution of average values among males in the Norwegian study population. The other depicts average values calculated for the Beddoe group. On average, the men in Waaler’s study fall closer to the middle of the range of desirable Quetelet index values, but the Beddoe group also falls toward the middle of that wide range. Compared to the men in Waaler’s study, the Britons that Beddoe and his collaborators measured in 1866 were nourished toward lower average heights and body mass. But, the body mass that they achieved as adults appears to be associated with good survival prospects.

Costa’s study of a small group of 377 veterans of the Union Army in the American Civil War promotes the same conclusion. The lowest mortality risk in her study group was associated with a Quetelet index value of 22 which, unlike the Waaler results, was decisively more favorable for survival than slightly higher or

23 Waaler, “Height, Weight, and Mortality,” 3–4. These proportions are inferred from the figure on p. 13. Waaler does not provide actual values.
Fig. 5 Survivorship by Body Mass

comparison made on the basis of height is to be sustained, then their life expectancy in 1866 should exceed that in 1980, which is far from being true.

When the analysis takes up body mass index rather than height, the evidence drawn from Beddooe's research suggests that the men in his study were smaller in proportion to their late twentieth-century counterparts, but not at greater risk. Judging by twentieth-century standards, those who prefer Waaler's estimates of the association between body mass and survivorship will find that the Beddooe group is slightly less favored than modern populations, which are heavier and have higher Quetelet index values. Those who prefer the more conservative advice of the Anglo-American nutritionists will find that Beddooe's group is favored. Its members were closer to the slender ideal recommended by present-day nutritionists. In either case, there are no grounds for supposing that nutritional deficiency in adulthood played a significant role in the higher mortality of adult males in Britain in the 1860s. Judged by a small body of evidence from that period, most men in the Beddooe group had nearly ideal body mass.

When stature alone is considered in comparison with mortality, the comparison has often been taken to suggest that mortality decline may have been associated with whatever factors have promoted gains in stature. But, weight matters. The addition of information about weight, which allows calculation of body mass, calls into question the use of height as a gauge of quality of life, nutritional status, and mortality risk. The favorable body mass of men in the Beddooe group challenges the conclusions of anthropometric historians, who have based their findings and interpretations primarily on information about height.
The Social Context of Child Mortality in the American Southwest

The health of children was an important subject of debate among doctors, reformers, and government officials at the beginning of the twentieth century, as evidenced by a grand tide of proposals for reform and research studies. The United States Department of Labor established the Children’s Bureau to marshal information and set up procedures for improving child health and reducing infant and childhood sickness and mortality. Individual states also acted for improved child welfare and health, spurred on by a women’s reform movement that saw children’s health and welfare as fundamental. The emphasis on children’s health reform was inspired, in part, by the poor health and sanitation conditions of the poorest and least well-educated families, relative to the healthier families within the privileged class of the reformers. The early studies of rural health conditions, such as Bradley and Williamson’s study of North Carolina, focus on how the differences between families affect their health. Studies of rural health in the Midwest, when compared with those of conditions in the South, further highlighted the wide variations between the childhood health and mortality

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This research was supported by Grant # R01 HD23693 from the National Institute of Child Health and Human Development, and by grants from the University of Texas Project Quest and the University Research Institute of the University of Texas, Austin. We are grateful to the other members of the Texas Historical Demography project staff, and especially to John Vetter, Jane Zachritz, Judy McArthur, and Christie Sample for their assistance and advice. This article was originally presented to a seminar organized by the International Union for the Scientific Study of Population on the subject of “Infant and Child Mortality in the Past.” We are grateful to the organizers for giving us the opportunity to present our work, and to the other participants for their helpful suggestions. We also wish to thank the Economic History Seminar at the University of Arizona for useful comments.

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Table 1  Characteristics of Women in the Texas Project Sample Counties, 1900

<table>
<thead>
<tr>
<th>ETHNIC ORIGIN GROUPS</th>
<th>TOTAL NUMBER OF WOMEN</th>
<th>ANGELINA</th>
<th>DEWITT</th>
<th>GILLESPIE</th>
<th>JACK</th>
<th>RED RIVER</th>
<th>WEBB</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>African</td>
<td></td>
<td>66</td>
<td>113</td>
<td>18</td>
<td>5</td>
<td>454</td>
<td></td>
<td>656</td>
</tr>
<tr>
<td>%</td>
<td>14.0</td>
<td>30.5</td>
<td>3.1</td>
<td>1.2</td>
<td>1.2</td>
<td>32.6</td>
<td>0.0</td>
<td>19.0</td>
</tr>
<tr>
<td>German (all generations)</td>
<td></td>
<td>10</td>
<td>72</td>
<td>369</td>
<td>19</td>
<td>21</td>
<td>7</td>
<td>498</td>
</tr>
<tr>
<td>%</td>
<td>2.2</td>
<td>19.4</td>
<td>63.2</td>
<td>4.9</td>
<td>1.5</td>
<td>2.8</td>
<td></td>
<td>14.4</td>
</tr>
<tr>
<td>1st generation</td>
<td></td>
<td>1</td>
<td>29</td>
<td>38</td>
<td></td>
<td></td>
<td>1</td>
<td>70</td>
</tr>
<tr>
<td>%</td>
<td>0.1</td>
<td>7.9</td>
<td>6.6</td>
<td>0.0</td>
<td>0.0</td>
<td>0.5</td>
<td></td>
<td>2.0</td>
</tr>
<tr>
<td>2d generation</td>
<td></td>
<td>0</td>
<td>28</td>
<td>230</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>264</td>
</tr>
<tr>
<td>%</td>
<td>0.1</td>
<td>7.5</td>
<td>39.3</td>
<td>0.8</td>
<td>0.1</td>
<td>0.8</td>
<td></td>
<td>7.6</td>
</tr>
<tr>
<td>3rd generation</td>
<td></td>
<td>9</td>
<td>15</td>
<td>101</td>
<td>16</td>
<td>20</td>
<td>3</td>
<td>164</td>
</tr>
<tr>
<td>%</td>
<td>2.0</td>
<td>4.0</td>
<td>17.3</td>
<td>4.1</td>
<td>1.4</td>
<td>1.4</td>
<td></td>
<td>4.8</td>
</tr>
<tr>
<td>Mexican (all generations)</td>
<td></td>
<td>0</td>
<td>31</td>
<td>12</td>
<td>0</td>
<td>2</td>
<td>202</td>
<td>247</td>
</tr>
<tr>
<td>%</td>
<td>0.0</td>
<td>8.3</td>
<td>2.0</td>
<td>0.1</td>
<td>0.1</td>
<td>84.8</td>
<td>7.2</td>
<td></td>
</tr>
<tr>
<td>1st generation</td>
<td></td>
<td>0</td>
<td>8</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>108</td>
<td>121</td>
</tr>
<tr>
<td>%</td>
<td>0.0</td>
<td>2.0</td>
<td>0.8</td>
<td>0.1</td>
<td>0.0</td>
<td>45.3</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>2d generation</td>
<td></td>
<td>0</td>
<td>12</td>
<td>3</td>
<td>0</td>
<td>38</td>
<td>5</td>
<td>73</td>
</tr>
<tr>
<td>%</td>
<td>0.0</td>
<td>3.2</td>
<td>0.5</td>
<td>0.0</td>
<td>0.0</td>
<td>15.9</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>3rd generation</td>
<td></td>
<td>0</td>
<td>11</td>
<td>5</td>
<td>1</td>
<td>56</td>
<td>74</td>
<td></td>
</tr>
<tr>
<td>%</td>
<td>0.0</td>
<td>3.1</td>
<td>0.8</td>
<td>0.0</td>
<td>0.1</td>
<td>23.6</td>
<td>2.1</td>
<td></td>
</tr>
<tr>
<td>Other whites</td>
<td></td>
<td>393</td>
<td>155</td>
<td>185</td>
<td>370</td>
<td>919</td>
<td>30</td>
<td>2,050</td>
</tr>
<tr>
<td>%</td>
<td>83.8</td>
<td>41.7</td>
<td>31.7</td>
<td>93.8</td>
<td>65.8</td>
<td>12.4</td>
<td>59.4</td>
<td></td>
</tr>
</tbody>
</table>

| OCCUPATIONAL STATUS OF HUSBAND |               |         |        |         |       |         |      |       |
| Farm owner               |               | 133     | 67     | 353     | 147   | 364     | 11   | 1,075 |
| %                       |               | 28.4    | 18.2   | 60.4    | 37.3  | 26.1    | 4.7  | 31.2  |
| Farm tenant              |               | 101     | 124    | 98      | 127   | 721     | 9    | 1,179 |
| %                       |               | 21.5    | 33.4   | 16.8    | 32.2  | 51.7    | 3.9  | 34.2  |
| Farm laborer             |               | 73      | 83     | 36      | 36    | 95      | 84   | 406   |
| %                       |               | 15.5    | 22.3   | 6.2     | 9.1   | 6.8     | 35.0 | 11.8  |
| High white collar        |               | 13      | 16     | 28      | 25    | 47      | 11   | 139   |
| %                       |               | 2.7     | 4.2    | 4.8     | 6.3   | 3.4     | 4.6  | 4.0   |
| Low white collar         |               | 31      | 20     | 25      | 33    | 49      | 17   | 174   |
| %                       |               | 6.5     | 5.3    | 4.3     | 8.3   | 3.5     | 7.3  | 5.1   |
| Skilled work             |               | 38      | 29     | 32      | 15    | 43      | 48   | 204   |
| %                       |               | 8.1     | 7.8    | 5.5     | 3.9   | 3.0     | 20.0 | 5.9   |
| Non-farm laborer         |               | 74      | 11     | 5       | 5     | 47      | 48   | 190   |
| %                       |               | 15.8    | 2.9    | 0.9     | 1.2   | 3.4     | 20.2 | 5.5   |
| No occupation            |               | 7       | 22     | 6       | 7     | 30      | 10   | 83    |
| %                       |               | 1.5     | 6.0    | 1.1     | 1.8   | 2.2     | 4.2  | 2.4   |

<table>
<thead>
<tr>
<th>HOUSEHOLD WEALTH (DOLLARS)</th>
<th>Mean</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>359</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>740</td>
<td>105</td>
</tr>
<tr>
<td></td>
<td>1,171</td>
<td>740</td>
</tr>
<tr>
<td></td>
<td>673</td>
<td>355</td>
</tr>
<tr>
<td></td>
<td>372</td>
<td>107</td>
</tr>
<tr>
<td></td>
<td>627</td>
<td>295</td>
</tr>
</tbody>
</table>
We use the same general scheme for identifying Mexican-Americans as German-Americans. The first and second generation categories are defined by place of birth and parents' place of birth. The third generation is defined by a dictionary of surnames. We began with the Hispanic surname dictionary from the 1980 United States census, which we supplemented with information from the Mexican-born and Mexican-parentage populations in our sample.8

MORTALITY IN TEXAS AT THE TURN OF THE CENTURY The census asked women who had been married various lengths of time how many children they had borne, and how many of them still survived. An empirical investigation showed that it is possible to estimate the average ages of the children, according to the number of years their mothers were married. For all women who had been married fewer than twenty-five years (the group we used), the mortality estimates refer to a sixteen-year period centered eight years prior to the census. Thus, the 1900 census yields information centered by a period around 1892, and the 1910 census yields information centered by a period around 1902. We refer to these two infant mortality periods as “1900 Census mortality” (roughly 1884–1900), and “1910 Census mortality” (roughly 1894–1910), in order to keep our description as simple as possible.9

The procedures that we have followed are based on those described by Preston and Haines in their influential study of late nineteenth-century child mortality in the United States, Fatal Years; our use of these measures is described in the Appendix. The point of the analysis is to use values of the \( q(x) \) parameter of a model life table to compute an expected number of dead children for every mother, based on the number of years she had been married. The expected number of dead children is the product of the total number of children that she bore, and the proportion that the model life table indicates were likely to have died by certain ages. For women married zero to four years, the analysis uses the \( q_z \) parameter (the proportion expected to have died in

Table 3  Child Mortality Characteristics of the Population for Six Texas Counties, by Ethnic Origin

<table>
<thead>
<tr>
<th>ETHNIC ORIGIN OF MOTHER</th>
<th>CHILDREN DEAD</th>
<th>MORTALITY ESTIMATES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>WOMEN</td>
<td>ACTUAL</td>
</tr>
<tr>
<td>African</td>
<td>656</td>
<td>605</td>
</tr>
<tr>
<td>German</td>
<td>498</td>
<td>216</td>
</tr>
<tr>
<td>Mexican</td>
<td>247</td>
<td>255</td>
</tr>
<tr>
<td>Other white</td>
<td>2,050</td>
<td>1,324</td>
</tr>
<tr>
<td>Total</td>
<td>3,452</td>
<td>2,400</td>
</tr>
</tbody>
</table>

Note: Based on once-married women married fewer than twenty-five years, with spouse present and no child from a previous marriage belonging to either woman or spouse.

The first two years of life, for marriages of five to nine years, the $q_5$, and so on. Once we have calculated an expected number of children born, we can compare that number with the actual number who have died. The analysis of differences in mortality is based on an index that is the ratio of the actual to the expected number of births, either for each woman, or for groups of women (see Table 3). This index is the important part of the analysis, and any related life table measures—such as $e_0$—are less important and less precise. With that caveat, we report a few life-table estimates, in order to put rural Texas childhood mortality in context, before turning to differentials in the Texas childhood mortality experience.10

The mortality experience of the children of mothers enumerated in the 1900 census shows that the expectation of life for rural Texas children at birth was similar to that in a Coale-Demeny west model life table with an expectation of life of nearly forty-nine years. Ten years later, the expectation of life at birth had grown by approximately two years. Overall, life expectancy in

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10 See the works cited in note 9. See the Appendix for further discussion.
Table 5  Child Mortality Index and Estimated $q_5$ for Six Texas Counties, by Ethnic Origin and County of Residence

<table>
<thead>
<tr>
<th>COUNTY</th>
<th>AFRICAN</th>
<th>GERMAN</th>
<th>MEXICAN</th>
<th>OTHER WHITE</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>INDEX</td>
<td>$q_5$</td>
<td>INDEX</td>
<td>$q_5$</td>
<td>INDEX</td>
</tr>
<tr>
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<td>0.53</td>
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</tr>
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</tr>
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<th>GERMAN</th>
<th>MEXICAN</th>
<th>OTHER WHITE</th>
<th>TOTAL</th>
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<tbody>
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<td></td>
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<td>$q_5$</td>
<td>INDEX</td>
<td>$q_5$</td>
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<td>0.68</td>
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NOTE  The 1900 and 1910 index values cannot be compared; estimated $q_5$s can be compared. Empty cells have fewer than ten actual child deaths. For other limitations on data inclusion, see Table 3 and text.

mediate level, with Angelina somewhat higher. Red River and Webb counties had the highest mortality levels. County and ethnicity are not independent in this analysis, given the structure of our sample. The large concentrations of Germans in Gillespie County (and to a lesser extent DeWitt County), of African-Americans in Red River County, and of Mexicans in Webb County shape the overall level of mortality in the respective counties. But it is significant that the “other white” category shows unmistakable county differences that demand further explanation. In the 1900 census data, the other whites in DeWitt, Gillespie, and Webb counties had much lower index values than did those in Angelina, Jack, and Red River counties. In the 1910 data, those in Gillespie, DeWitt, and Jack counties had the lower child mortality, and Angelina, Red River, and Webb counties the higher.

Tables 4 and 5 demonstrate that child mortality in Texas is complicated by the interrelationships between ethnicity, occupa-
tion, and location. The families of farm owners generally had lower mortality than did those of farm tenants, except in the African-American population. Moreover, some counties had better child mortality experiences than others. These interactions demand a more complex analytic scheme that will reveal genuine patterns—namely, a multiple regression analysis in which each woman's child mortality index is the dependent variable. We must also understand the forces that shaped infant and child mortality in rural Texas.

THE DETERMINANTS OF DIFFERENTIAL CHILD MORTALITY Reformers at the beginning of the twentieth century concentrated on three issues to lengthen the lives of children. Their first concern was medical care for mothers, before and during the process of giving birth. A mother who received competent prenatal care, and who was attended at birth by a well-trained physician or midwife, was more likely to produce a healthy child and be healthy herself, than one who was not. The second concern of children's health reformers was good infant and child nutrition. Mothers were urged to breast-feed their children and to provide a balanced diet to those who had been weaned. The third concern involved sanitation. In the rural context, representatives of reform groups and the Children’s Bureau of the Department of Labor taught the need for properly protected water supplies and proper disposal of human and animal waste. They argued for deep water wells with pumps, protected from the contaminants that could foul shallow, open-topped wells. They also advocated properly designed domestic sanitation systems, consisting of, at minimum, well-constructed privies that would not foul ground water and, at maximum, modern septic systems for containing human waste.14

Although it would be desirable to measure the extent to which any household had access to the improved maternal, infant, and child health provisions that the reformers proposed, the available sources make it difficult to track down individual health, nutrition, and sanitation conditions. Instead of measuring the causes of good childhood health directly, we measure them indirectly. Some of the differences between mortality experiences are explained by the individual characteristics of women and their

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14 Bradley and Williamson, Rural Children; Moore, Maternity and Infant Care.
Table 8: Regression Models for 1910

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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<td>R²</td>
<td>0.080</td>
<td>0.100</td>
<td>0.085</td>
<td>0.104</td>
</tr>
<tr>
<td>Adjusted R²</td>
<td>0.080</td>
<td>0.099</td>
<td>0.084</td>
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</tr>
<tr>
<td>Constant</td>
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<td>−0.093</td>
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<tr>
<td>Age</td>
<td>0.017****</td>
<td>0.018****</td>
<td>0.016****</td>
<td>0.017****</td>
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<td>Household wealth ($1,000s)</td>
<td>−0.019****</td>
<td>−0.016****</td>
<td>−0.018****</td>
<td>−0.015****</td>
</tr>
<tr>
<td>Ethnic origin (without generations)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African</td>
<td>0.241****</td>
<td>0.156****</td>
<td>0.243****</td>
<td>0.157****</td>
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<tr>
<td>German</td>
<td>−0.334****</td>
<td>0.049</td>
<td>−0.266****</td>
<td>0.077</td>
</tr>
<tr>
<td>Mexican</td>
<td>0.517****</td>
<td>0.759****</td>
<td>−0.364****</td>
<td>0.033</td>
</tr>
<tr>
<td>Other white (reference group)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African</td>
<td>0.263****</td>
<td>0.157****</td>
<td>0.262****</td>
<td>0.077</td>
</tr>
<tr>
<td>German 1st generation</td>
<td>−0.364****</td>
<td>0.033</td>
<td>−0.283****</td>
<td>0.065</td>
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<tr>
<td>German 2d generation</td>
<td>−0.662****</td>
<td>0.951****</td>
<td>−0.621****</td>
<td>0.527****</td>
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<tr>
<td>Mexican 1st generation</td>
<td>0.214****</td>
<td>0.524****</td>
<td>0.252****</td>
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<tr>
<td>Mexican 2d generation</td>
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<td>0.252****</td>
<td>0.524****</td>
</tr>
<tr>
<td>Mexican 3d generation</td>
<td>0.252****</td>
<td>0.524****</td>
<td>0.252****</td>
<td>0.524****</td>
</tr>
<tr>
<td>Other white (reference group)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Husband’s Occupation</td>
<td></td>
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<tr>
<td>Farm owner (reference group)</td>
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</tr>
<tr>
<td>Farm tenant</td>
<td>0.061****</td>
<td>0.009</td>
<td>0.062****</td>
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<tr>
<td>Farm laborers</td>
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<td>−0.102**</td>
<td>−0.155****</td>
<td>−0.115**</td>
</tr>
<tr>
<td>High white collar</td>
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<td>−0.019</td>
<td>−0.008</td>
<td>0.024</td>
</tr>
<tr>
<td>Low white collar</td>
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<td>−0.065</td>
<td>−0.052</td>
<td>−0.057</td>
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<tr>
<td>Skilled work</td>
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<td>0.315****</td>
<td>0.260****</td>
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<tr>
<td>Non-farm laborer</td>
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<td>0.413****</td>
<td>0.371****</td>
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<tr>
<td>No occupation</td>
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</tr>
<tr>
<td>County</td>
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<td></td>
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</tr>
<tr>
<td>Angelina</td>
<td>0.455****</td>
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<td>0.457****</td>
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<td>0.134****</td>
<td>0.134****</td>
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<tr>
<td>Gillespie (reference group)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Jack</td>
<td>0.300****</td>
<td>0.295****</td>
<td>0.295****</td>
<td>0.295****</td>
</tr>
<tr>
<td>Red River</td>
<td>0.699****</td>
<td>0.695****</td>
<td>0.695****</td>
<td>0.695****</td>
</tr>
<tr>
<td>Webb</td>
<td>0.209****</td>
<td>0.134**</td>
<td>0.209****</td>
<td>0.134**</td>
</tr>
</tbody>
</table>

* Significant at the .1 level.
** Significant at the .05 level.
*** Significant at the .01 level.
**** Significant at the .001 level.

white farm laborers in Red River County. The greatest difference distinguished the two non-farm working-class groups—the skilled workers and the non-farm laborers—a large proportion of whom lived in Webb County. We discuss the importance of that connection below.
We expected the counties to display different child mortality experiences, and model 2 of tables 7 and 8 offer confirmation. Gillespie County, the reference, had the lowest mortality of all the counties, with age of woman, wealth, ethnicity, and husband’s occupation taken into account. DeWitt’s was slightly higher, Angelina and Jack Counties were intermediate, and Red River and Webb counties had the highest mortality. The models that include the counties are especially interesting because they reduce the importance of the German ethnic variable, suggesting that the advantage experienced by the Germans was a consequence of living in Gillespie and DeWitt counties, or that, because virtually all of the Germans lived in these counties, the consequences of German sanitation and cleanliness affected other groups. Put another way, there is good reason to believe that by the first decade of the twentieth century (the 1910 data reflects sixteen years centered on 1902), the child mortality experiences of German-Americans and other whites were similar in those counties where they lived in proximity. The other whites have higher mortality in the overall index values because of their residence in other counties and their different distributions of occupation and wealth. We speculate further about these relationships in the conclusion.16

In the third and fourth models presented in tables 7 and 8, we divide the Mexican- and German-origin populations according to generation—the foreign born (first generation) from the native born of foreign parents (second generation) and the native born with the Mexican and German surnames of native parents (third generation). Model 3 includes only ethnicity; model 4 also includes the county variables. The results for the occupation, age, wealth, and county variables, and for the models as a whole, are not too different from the simpler models in which generation was not taken into account. The results for generation raise the question of whether second- and third-generation Mexican-Americans and German-Americans were more like their immigrant parents or more like members of other ethnic groups. The answer is revealing. German-Americans were relatively consistent in all three generations. They were likely to have had lower

16 We verified the relationship between ethnicity and county of residence in another set of regression models, in which we interacted residence and ethnicity, finding that other whites had mortality that varied from county to county, but child mortality was similar to that of German Americans in DeWitt and Gillespie Counties.
benefited, in relative terms, the life chances of German-Americans, above and beyond their wealth and occupation? It is unlikely that genetic susceptibilities to disease were sufficient to explain the child mortality differences between these groups. Although there may have been cultural differences—that is, members of these groups may have been more likely to live in some situations than others—those cultural differences were shaped largely by social and economic opportunities.

In a recent article, Fliess shows that the Wendish population of Serbin, Texas (not included in our sample, but living in conditions similar to those in DeWitt County), reduced their levels of infant mortality between the 1850s and the 1880s, by limiting air- and waterborne contagious diseases. By the 1890s, the German-American population probably were able to do so because they lived in well-constructed farm houses and were relatively prosperous. Apparently, by 1910, other whites were making progress as well, at least where they had German-American models to follow. The higher-mortality, Mexican-American and African-American families, however, did not have the same opportunities. Studies of rural living conditions in the South suggest that the poorest whites and blacks may have been ignorant about how to improve their health and too poor to do much about it anyway. The complexity of this story is made greater by differences in mortality within each county, even for the group, “other whites.”

We began with the idea that differences in child mortality by county and ethnic background might have had something to do with location, in a purely geographical sense. Many of the German-Americans in our sample lived in Gillespie County. This area of the Texas “hills” has an arid climate, porous limestone soil, and relatively clean surface water. Many of the African-Americans lived in Red River County, which is at a lower elevation, has a heavy clay soil, and more surface water. DeWitt County, with its mixed population, had an intermediate geography. The greater possibility of drawing contaminated water in Red River County than in Gillespie County should have resulted in higher infant mortality. The rate in DeWitt County should have fallen between those of the other two. The presence of families in our other-

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19 Fliess, “Mortality Transition.”
white category in all three counties allows us to test this hypothesis. Table 5 shows that mortality for other whites was higher in DeWitt than in Gillespie County, and highest in Red River County. Terrain seems to play a role, but it is only part of the explanation.

What makes the story more complicated than just a combination of geography, wealth, and ethnic origin is that the diseases that killed the children of rural Texas were contagious. There may have been a relationship between proximity of disease and level of mortality, even for groups that were otherwise not likely to have undergone high levels of child mortality. We anticipated that in counties with dense populations of poor people living amid unsanitary conditions and using contaminated water, everyone would have had ample contact with disease, and that in counties with dense populations per se, the exposure would have been worse. These hypotheses were impossible to test with the data at hand. Nonetheless, it is noteworthy that non-Mexican and non-German whites lived in a poor, or a densely populated county, as did Germans. Living in DeWitt County, with its dense, poor population was more dangerous for children than living in Gillespie County, all other things being equal.

The reformers in the early years of this century who attempted to reduce infant mortality sought to work within the contemporary social system and to improve the health of women and children by teaching them about nutrition and sanitation. They knew that money made a difference; the children of well-to-do parents are always more able to survive childhood. However, they also argued that improvements in drinking water and sanitation would help the poorer population. The reformers of the Children’s Bureau did not have our sophisticated statistical approaches, but they had a wealth of case studies that showed the importance of the measures that they advocated. Our analysis shows that they were correct. Wealth and ethnicity—along with occupational status, to some extent—were the driving forces behind differences in child mortality in rural Texas. It is just as important to note that some parts of rural Texas provided a better environment for the survival of children than others. But we wish to emphasize that the conclusions that we draw are similar to those that the reformers drew. The measurable differences in the social context of child mortality discussed in this article seem mostly to
expected to have died. If more of her children died than expected, the index is large; if fewer died than expected, the index is small. The numbers of actual and expected children can be summed for a group of women, and an overall index computed. In the main body of this article, we used indexes computed for groups of women according to specific categories (by ethnic group, or by ethnic group and occupation or county), and we used the indexes computed for individual women in the multivariate regression analyses.

It is sometimes necessary to convert child mortality indexes back to model life tables. Preston and Haines show that it is sufficient to multiply the index by the \( q_5 \) of the reference model life tables (those we use are shown in Table A2) to determine the \( q_5 \) of the model life table that is represented by the child mortality index. The \( q_5 \) can be converted to any other parameter of the life table with the Coale-Demeny model life tables (and either the published volume or the Match program). For convenience, we usually present these life tables by showing an expectation of life at birth, sexes combined.