Environment, Disease, and Mortality in Early Virginia

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Disease was one of the principal causes of death in early Virginia, and Jamestown was the locus of mortality. This association between disease and place has long been observed, but seldom understood. A geographic model of disease mortality, however, can account for the spatial, seasonal, and annual mortality variations in Jamestown and the James River estuary between 1607 and 1624. This model, derived from the first year in Jamestown, suggests the probable causes of disease-related deaths and offers a logical and consistent account of the pathogenic organisms; the sources of infection; the incidence of infection, morbidity, and mortality; and the recurrence of epidemics. This essay presents the derivation of the model, based on the first year at Jamestown; examines the application and testing of the model for the years 1608 to 1624; discusses the colonists’ and the Virginia Company’s inability to lower mortality rates; and, last, offers some speculation on the causes of declining death rates in Virginia after 1624.

The first year in Virginia portended the dreadful mortality that ravaged the colony until 1624. Initially things went well. The expedition of three vessels and 144 persons left England in December 1606, headed south and west to the West Indies, and then veered north to the Chesapeake Bay, entering it on April 26, 1607. Shortly thereafter, the colonists established Jamestown on the north side of the James River, nearly fifty miles from its mouth.1 The Virginia spring was beneficent, and when Captain Newport departed on June 22, he left 104 healthy colonists. But soon the colony took on a somber attitude. On July 6 George Percy’s journal mentioned John Asbie’s death by the “bloudie Flix.” Three days later George Flowre died of the “swelling.” In the space of a month 21 colonists died, causing Percy to lament that “our men were destroyed with cruel diseases, as Swellings, Flixes, Burning Fevers, and by warres, and some departed suddenly, but for the most part they died of meere famine.”2 By the end of September, 46 were dead, and in January, when the first supply ship arrived in Virginia, just 38 of 104 colonists were barely alive.3

The final version of this paper has been improved by the comments and criticisms of Daniel Doepers, James Knox, Allan Kulikoff, and Russell Menard. Map credits go to the Cartographic Laboratory of the University of Wisconsin-Madison and Cartographic Services of the University of Maryland, Baltimore County.


3. Arber and Bradley, eds., Travels and Works of Captain John Smith, I, lxvi, 9, 95; Alexander Brown, The First Republic in America, An Account of the Origin of This Nation,
The abundance of death demanded an explanation. But Percy's speculation that "meere famine" was the cause of death is unconvincing. In support of his thesis we can say that the colonists' daily ration consisted of just half a pint of wheat and another of barley, mixed in a gruel that yielded roughly half the caloric intake required for an active man of the colonists' stature. But we should not hastily accept Percy's "meere famine," if only because of the political disputes and intrigues rending the first colony. Percy may have had an axe to grind. The selection of Jamestown over the Archer's Hope site displeased him, and conceivably he chose to support the faction that accused President Edward Maria Wingfield of hoarding the colony's food and drink for presidential favorites. Whatever Percy's motives, his emphasis on famine spotlighted President Wingfield. The president, of course, denied such allegations. His rebuttal drew indirect support from one of his enemies, Captain John Smith. Smith made little of the shortage of provisions, stating matter-of-factly on several occasions that the colony still had many weeks of supplies remaining. He knew that the annual sturgeon run would provide a supplementary source of food. Thousands of these fish entered the James estuary in April and May, and their run to freshwater spawning grounds continued through the summer, when the big fish came in. "From the later end of May till the end of June," wrote Smith, "are taken few, but young Sturgeon of 2 foot or a yard long. From thence till the midst of September, them of 2 or three yards long and fewe others. And in 4 or 5 hours with one nette were ordinarily taken 7 or 8: often more." A few years later John Rolfe related that two men in a few hours had axed forty sizable sturgeon. Since the Atlantic sturgeon averages over one hundred pounds, the output of two axe-wielding men would have totaled four thousand pounds, or nearly forty pounds per colonist per day. A daily intake of two pounds of sturgeon, some crabs, and the wheat-barley gruel was more than adequate for the colonists' metabolic needs. Furthermore, two pounds of fish daily would have provided 90 percent of the daily thiamine requirement, and would thus have thwarted the outbreak of beriberi that has been postulated by the medical historian Wyndham Blanton.

The food supply during Jamestown's first summer, though unappealing, provided sufficient nourishment to ward off starvation and vitamin deficiency diseases. Starvation was not the principal cause of death at Jamestown, but the possibility was constantly feared by the colonists. By mid-September they perceived that starvation was imminent. Newport had left them supplies for thirteen or fourteen weeks, and even though the death of 50 colonists had reduced the drain on the supplies, by September they had only enough for four to eight weeks and did not expect additional supplies until October at the earliest. Moreover, the sturgeon run fell off.

Although Percy blamed famine, his list of clinical symptoms brings us closer to the actual causes of death—typhoid, dysentery, and perhaps salt poisoning. Medical historians generally agree that Percy's "flixe" or "bloudie Flixe" describes dysentery, and "Burning Fevers" are symptomatic of typhoid fever. The "Swellings," though perhaps associated with dysentery, could also result from salt intoxication from the salty river water. These three diseases are also indicated by the incidence and rapidity of death, as chronicled by Percy. Typhoid fever progresses rapidly after infection by the bacterium Salmonella.

Written from the Records Then (1624) Concealed by the Council. Rather than from the Histories Then Licensed by the Crown (Boston and New York, 1898), 55.
6. Ibid., 8-9, 51.
10. Very useful are John Duffy, Epidemics in Colonial America (Baton Rouge, La., 1953); Wyndham B. Blanton, Medicine in Virginia in the Seventeenth Century (Richmond, Va., 1930), 3-77; Thomas P. Hughes, Medicine in Virginia, 1607-1699, Jamestown 350th Anniversary Historical Booklets, no. 21 (Williamsburg, Va., 1957); and Richard Harrison Shryock, Medicine and Society in America, 1660-1860 (New York, 1960), 82-116.
typhosa. The first week may be symptomless, as the organisms spread through the bowel wall and into the lymphatic glands. In the second week the organism enters the bloodstream, causing a rapid rise in body temperature, recognized by colonists as the "Burning Fever." The illness peaks in the third week, and death may result. Before the use of antibiotics, it is estimated that 15 to 20 percent of infected persons died.\textsuperscript{12} Dysentery, caused by amoebic parasites, produced the "bloody Flxie." While several types of amoebic parasites reside in the human intestinal tract, most are harmless commensals or organisms that may cause diarrhea or mild dysentery. More dangerous is Endamoeba histolytica; it may invade the bowel wall, causing ulceration and the bloody stools that gave the disease its seventeenth-century name. More serious complications result when these parasites bore into a large blood vessel, causing massive hemorrhage, or when the amoeba get into the bloodstream and travel to other organs. Dysentery is often fatal, especially when populations are weakened by other illnesses or undernourishment. Pre-antibiotic mortality rates of 12 to 25 percent have been recorded.\textsuperscript{13} Like typhoid, dysentery can act quickly, though the rates of incubation vary with the individual. Controlled experiments with human volunteers have shown "that the prepatent period, i.e., from exposure until the amoeba appeared in the stools, averaged nine days, varying from one to 44 days in the 17 of 20 exposed individuals who became infected."\textsuperscript{14} Clinical symptoms usually appear within one to four weeks, but the range may be from a few days to several months.

12. The fever lasts 21 days usually and occasionally up to 33 days. Frederick P. Gay, Typhoid Fever Considered as a Problem of Scientific Medicine (New York, 1918), 13–24; William Budd, Typhoid Fever: Its Nature, Mode of Spreading, and Prevention (New York, 1931); Jacques Meyer May, The Ecology of Human Disease (New York, 1958), 171–188. Gay shows 15 to 27 percent mortality for the London Fever Hospital, 1848 to 1870. In general I agree with Jones that typhoid killed numerous Virginians in 1607. I disagree with him on the following points: (1) that typhoid, aided by beriberi, was the principal killer; (2) that typhoid was probably introduced by Reverend Robert Hunt; and (3) that the Jamestown environment was essentially passive in the typhoid epidemic. Gordon W. Jones, "The First Epidemic in English America," Virginia Magazine of History and Biography, LXXI (1963), 3–10.


14. Faust, Amebiasis, 58.

An epidemic of typhoid fever and dysentery is consonant with Percy's description of death and sickness at Jamestown. Percy first noted disease-related deaths on July 6, and sickness and death continued "for the space of six weeks"—a time span in keeping with the progression of typhoid fever and dysentery. Furthermore, 50 of the 104 colonists had perished by mid-September. The actual mortality rate of 48 percent just slightly exceeds the sum of expected pre-antibiotic mortality rates for typhoid (15 to 20 percent) and dysentery (12 to 25 percent), or a total rate of 27 to 45 percent. Our estimated rate of mortality may be criticized as too high on the grounds that some colonists died of both pathogens, and therefore the separate rates are not additive. The evidence does not permit an estimate of multiple causes of death. On the other hand, my guess is that the correct estimate of disease mortality probably lies in the 27 to 45 percent range and near the upper end. This supposition is based on several factors. First, the Jamestown population was probably under thirty years of age, and hence included the most susceptible age groups for typhoid (15 to 25 years) and dysentery (26 to 30 years).\textsuperscript{15} Furthermore, the concentrated and confined population facilitated the spread and incidence of these diseases and perhaps their virulence as they passed rapidly from one infected colonist to another (via the feces and ingestion).\textsuperscript{16}

The role of typhoid and dysentery may be further clarified by identifying the disease agents, the process of introduction, and the sources of human exposure. The introduction of these two diseases is not problematic. The colony contained numerous carriers of both diseases. According to modern laboratory diagnostic surveys, Endamoeba histolytica is carried by 40 percent of the population from 26 to 30 years old, with a decreasing incidence away from that peak.\textsuperscript{17} And typhoid bacilli are carried by 2 to 30 percent of the general population.\textsuperscript{18} In both diseases carriers may be symptomless and therefore almost

15. Ibid., 28. About 50 percent of all typhoid cases occur between the ages of 15 and 25. Gay, Typhoid Fever, 13.
16. Faust, Amebiasis, 26. Modern surveys for Endamoeba histolytica in the southeastern United States average 11 percent positive in the general population, but among the concentrated populations of a mental hospital and an orphanage, the positives rose to 40 and 55.5 percent respectively. Willard H. Wright, "Parasitism in Southeastern United States: Current Status of Parasitic Diseases," Public Health Reports, LXX (1955), 966–975; Gay, Typhoid Fever, 14–15, 43–45.
18. Gay, Typhoid Fever, 43.
impossible to detect in the absence of laboratory diagnoses. The
Jamestown carriers passed millions of disease organisms in their feces
and also their urine in the case of typhoid. The diseases were then
transmitted, in all probability, through a contaminated water supply.

But what was the water supply, and why was it contaminated in
July and August and not earlier or later? The colonists drank river
water. In spring the water was safe. With river discharge at a max-
imum—owing to high precipitation, low evaporation, and high runoff
—the fresh running water swirled around Jamestown Island and
flushed disease organisms downstream. But the water supply became
contaminated as summer set in. River discharge fell, water levels re-
ceded some ten to fifteen feet, and Jamestown Island became a pen-
sula attached to the mainland.19 Pools of standing water and stagnant
marshes rimming the mainland side of the island created a wetland
environment ideal for the retention of Salmonella typhosa and
Endamoeba histolytica. Even more deadly was the summer contamina-
tion of the river water with salt, sediment, and fecal material. As fresh-
water discharge fell, saltwater invaded some thirty miles up the James
estuary from Hog Point in the spring to Jamestown by mid-summer.
And along the landward-moving freshwater-saltwater boundary, sed-
iments and organic wastes were trapped by the salt plug—particu-
larly on the north side of the James, owing to the rightward deflection
of the marine incursion by the earth’s rotation.20 Percy put it succinctly: “Our drink was cold water taken out of the River, which was

19. The normal regime of Chesapeake estuaries is described here. Discharge,
however, will depart from the norm of spring high and summer lows under atypi-
cal meteorological conditions, e.g., prolonged drought or excessive rainfall, variable
evapotranspiration, variable snow-melt water. Virginia, Virginia Conservation Com-
mission, Division of Water Resources, Surface Water Supply of Virginia: James River
Basin, nos. 5, 13, 17, 25 (Charlottesville and Richmond, Va., 1944–1961). As the James rose in
spring and receded in summer, Jamestown occupied alternately an island and a
peninsula attached to the mainland. C. A. Browne, “Reverend Dr. John Clayton and
His Early Map of Jamestown, Virginia,” William and Mary Quarterly, 2d Ser., XIX (1939),
5–6. The recession in river depth is estimated from depths of the main channel at
Jamestown. Percy gives 6 fathoms (36 feet) in spring, an English pilot, interrogated by
the Spanish in 1611, put the depth at 3½ fathoms (21 feet) at the same point. The river’s
annual range is 15 feet. “Observations by Master George Percy,” in Tyler, ed., Narratives
of Early Virginia, 15; Alexander Brown, The Genesis of the United States, A Narrative of the
Englishmen, ... Set Forth Through a Series of Historical Manuscripts ... (Boston and New
York, 1890), 514; Samuel H. Yenge, The Site of Old “James Towne,” 1609–1698 ... (Richmond, Va., 1904).

20. An excellent survey of the James is Maynard M. Nichols, “Sediments of the
James River Estuary, Virginia,” CN rods Society of America, Memoir 133 (1972), 169–212.

at a flood verie salt, at low tide full of slime and filth, which was
the destruction of many of our men.”21 At flood tide the colonists
drank water containing salinity concentrations of over five parts per
thousand—far above the recommended standard for constant daily
usage of one part per thousand. The colonists suffered from salt
poisoning, with its characteristic symptoms of “swellings” (edema),
lassitude, and irritability. The idle, lazy, and factious behavior of early
Virginians was, in part, the result of a steady summer diet of salty
water.22 The ebb tide, though less saline, was very turbid, organically
polluted, and deadly. The trapped pathogens of typhoid and dysen-
tery, thus floated back and forth past Jamestown with the summer
tide. The danger from contaminated water faded in September. River
discharge increased, pushing the salt incursion and its deadly associ-
ates downstream toward Hog Point.

The 1607 epidemic of typhoid and dysentery was the first of many
summer epidemics in early Virginia. Fevers, fluxes, sickness, and
death visited the colony recurrently between 1607 and 1624. One
decisive factor underlying these repeated epidemics is the limited
immunity conferred by the diseases themselves. Dysentery survivors
acquire no immunity to subsequent attacks. Severe dysentery attacks
do invoke a limited antibody response for two weeks after the infec-
tion, but thereafter the survivor is again susceptible to infection.23

Typhoid attacks confer slightly more immunity. Typhoid recurrence is
usually put at .75 to 4.2 percent; however, the recurrence rate rises to
8 to 15 percent in especially virulent and massive infections, like those
in Jamestown.24 Thus, the survivors of dysentery and of typhoid
epidemics at Jamestown were only slightly less susceptible to these
diseases than newly arrived immigrants. Survivors of a Virginia sum-


22. Drinking water preferably should contain not more than 0.5 parts per thousand
salt content; however, some contemporary municipal water supplies use two parts per
thousand without public complaint. Thomas R. Camp and Robert L. Meserve, Water
and Its Impurities, 2d ed. (Stroudsburg, Pa., 1974), 2; Keitel, Pathophysiology and Treatment
of Body Fluid Disturbances, 162–164, 209–210; Bland, “Clinical Physiology and Four
Avenues of Loss and Gain,” in Bland, ed., Clinical Metabolism, 133–164. A composite of
early Virginians’ behavior would include irritability, laziness, short tempers, factious-
ness, and hypocritical perceptions. The extremity of their situation accounts for some of
these behaviors; salt poisoning accounts for them all. On idleness in early Virginia, see
Edmund S. Morgan, “The Labor Problem at Jamestown, 1607–1618,” American Historical

23. Faust, Abemiasis, 30–32.

mer did become "seasoned" to a new disease environment, but they were not particularly immune to future epidemics of typhoid, dysentery, or salt poisoning. These epidemics recurred for another reason—the annual summer invasion of saltwater up the James that contaminated the Jamestown water supply.

This close relationship between environment, disease, and mortality in 1607 Jamestown may be stated more generally for all Chesapeake estuaries. For our purposes, an estuary is an ecological unit wherein freshwater from the land is mixed with encroaching water from the sea, producing three salinity zones: a zone of freshwater, with salinity less than .5 parts per thousand; a zone of freshwater-saltwater transition (the oligohaline), with salinities of .5 to 3 parts per thousand; and a zone of salty water, with salinity above 3 parts per thousand (includes the mesohaline, polyhaline, and marine). Sediment and fecal material entering an estuary are flushed out of its freshwater portion, temporarily trapped or plugged up by the salt incursion in the oligohaline until a large portion is eventually flushed downstream into the saltier water. Thus, pathogenic river-borne organisms are least common in the freshwater zone, maximum in the oligohaline zone, and intermediate in the mesohaline and polyhaline zones near the estuary mouth. Contamination also varies by bank side. Left bank contamination exceeds that of the right bank owing to the deflection of the salt incursion by the earth's rotation. This geographic distribution of estuarine contamination is, in turn, directly correlated with human exposure, infection, and mortality from the pathogens of typhoid and dysentery. Mortality also varies seasonally with the migrations of the salt incursion. In the oligohaline zone the probability of infection increases when the saltwater-freshwater boundary passes by; clinical symptoms and mortality lag behind during the incubation period, with a normal lag of about one week to one month. The location of this deadly boundary zone migrates with river discharge. In the Chesapeake estuaries low discharge usually occurs in summer, and the saltwater invades the estuary to its landward maximum; on the James it penetrates thirty miles to the vicinity of Jamestown, where as a result seventh-century mortality rates should have peaked in July and August. Highest discharge customarily comes in the spring, and pushes the saltwater to its seaward maximum; on the James the retreat is to Hog Point, where mortality rates should have peaked in April and May. Within the saltwater zone, mortality rates should have risen slightly in spring because of the proximity of the salt trap; however, this zone would have received tidal flows of fecal material throughout the summer, thus assuring summer sickness and death. Recurrent epidemics were possible when population occupied the freshwater-saltwater and saltwater zones.

Having put forward a geographic model of mortality, I hastily remind the reader of its crudity. The model coarsely subdivides estuaries into three salinity zones and hypothesizes their variable mortality. A more refined model might specify the precise concentrations of contaminants, as a function of estuarine flushing and of the transport and the life expectancies of pathogens, and the expected levels of infection and mortality. The hydrologic information for early Virginia hardly warrants such refinements.


26. The distribution of disease organisms within an estuary depends on their point of entry, the circulation and flushing time of the estuary, and the life expectancy of the disease organisms. Laboratory experiments show that coliform bacteria, an indicator of disease contaminants, die off rapidly to one-tenth their original population in a period of one half to two or three days. The extent of downstream contamination increases when river circulation is rapid and the pollutants are flushed downstream before death. Bostwick H. Ketchum, “Distribution of Coliform Bacteria and Other Pollutants in Tidal Estuaries,” Seine and Industrial Wastes, XXVII (1955), 1288-1296; Clarence J. Velz, Applied Stream Sanitation (New York, 1970), 339-379; Wastes Management Concepts for the Coastal Zone: Requirements for Research and Investigation, Committee on Oceanography and Committee on Ocean Engineering, National Academy of Engineering (Washington, D.C., 1970).

27. The expected timing and location of disease morbidity and mortality rest on the assumption of “average” climatic conditions and “normal” estuarine circulation, i.e., peak discharge and salt retreat in spring, and low discharge and salt incursion in late summer. Atypical weather conditions could alter the timing and location of disease incidence. The timing of epidemics is affected also by physiological factors. An increased incidence of typhoid and dysentery in later summer may have to do with increased human output of pathogens at that time. Counts of coliform bacteria in the Detroit River rose steadily in spring, reaching a peak in August; the reasons underlying this increased productivity are incompletely understood. For our purposes, this increased summer output should produce higher mortality in the landward edge of the oligohaline than on the seaward edge. Velz, Applied Stream Sanitation, 239-242.
The data for early Virginia afford several opportunities of testing the geographic model of mortality. For the period from 1607 to 1624, deaths may be estimated from contemporary statistics and estimates of population and immigration, which must be handled with circumspection. Figures can lie, and early Virginians regularly juggled population estimates to suit their purposes. The most probable causes of death are deduced from colonists’ descriptions of the timing and symptoms of death and the reasonableness of their explanations as to the causes of death. The locations of population and of mortality derive from contemporary accounts. Particularly useful is the geographic census of the living and dead for 1623–1624.

The first question at issue is the relationship of mortality and the location of population in early Virginia. A chronological survey of the period from 1608 to 1624 reveals the recurrent deadliness of Jamestown summers. When population was concentrated in the town, mortality rates invariably rose above 30 percent; and when the population dispersed, death rates declined sharply.

The first two summers in Virginia were disastrous; the third offered the first glimmer of hope. As of October 1608 perhaps 244 colonists had come to Jamestown, and 144 of them had subsequently died. But the death rate fell abruptly between October 1608 and the summer of 1609, when by the most liberal estimates just 21 of 130 persons died, including eleven by drowning. This anomaly of survival deserves comment. Captain John Smith claimed credit for this success, and rightly so. Smith, though a vainglorious man, was also a sensitive ethnographer. He carefully recorded the Indians’ seminomadic economy and undoubtedly understood its survival value. In the spring the Indians congregated along the James estuary, subsisting on marine life while they planted their crops of corn, pumpkins, beans, and so forth. As summer approached, the tribes dispersed into smaller groups, residing usually on a hill with a fresh water spring, yet near the river where they gathered fish, oysters, and crabs. By dispersing, the Indian bands avoided the deadly estuarine zone, while exploiting scattered edible plants and animals during this leanest of seasons. But survival had its price. The scattered bands were politically and militarily weak. They sniped at their vulnerable, sick, and weak English enemies, but a summer war of attrition was impossible. As this flux in Indian power eluded most Virginians, they were terrified by late summer. Percy fully expected annihilation in 1607, and he marveled that God had saved them by putting “a terour in the Savages hearts.” Smith saw things more clearly; the Indians were almost as vulnerable as the whites. In 1609 he dispersed his men with impunity. Smith also understood the Indians’ generosity in the fall. Then they reassembled, harvested their crops, and gorged themselves. Their full bellies made them charitable, and they brought “Bread, Corne, Fish and Flesh in great plente” to the confounded colonists. With the onset of winter, the Indians once again fragmented into small bands and migrated upland into their piedmont hunting grounds, where they stalked deer, bear, and other game animals. Smith’s genius was in placing the puzzling Indian behavior and subsistence strategies into a coherent ecological whole. He realized that the colony’s survival, no less than the Indians’, depended on seminomadism, at least during the deadly summer season.

In late May 1609 President Smith scattered the Jamestown settlers into the surrounding countryside. His scheme infuriated Captain Gabriel Archer, who described more than he understood: “Howbeit when Captaine Argoll came in [about July 10, 1609], they were in such distress, for many were dispersed in the Sauages townes,

28. In December 1606, 144 colonists went to Virginia; 104 were left by Newport in June 1607, 38 to 40 survived in January 1608, 100 to 120 immigrants arrived between January and September 1608, and 60 were alive in October 1608. The mortality rate in the text is from Irene W. D. Hecht, “The Virginia Colony, 1607–1640: A Study in Frontier Growth” (Ph.D. diss., University of Washington, 1969), 68; Brown, First Republic in America, 55, 58–59, 68. Brown’s population and immigration figures are usually accurate, and I rely on them frequently. However, his friendliness toward the Sandys administration from 1619 to 1624 and its “democratic” character leads him to minimize the mortality problem then, while he is excessively critical of mortality under the crown and under Thomas Smythe.

29. Friends of Smith claimed that only 7 or 8 died out of 200. Brown shows 130 alive in October 1608, 11 drowned in mid-January, and not more than 109 survived.


32. Ibid.
living upon their almes for an ounce of Copper a day; and fourescore lived twenty miles from the Fort and fed upon nothing but oysters eight weeks Space.”33 Smith’s scheme of dispersal, though repugnant to Argall, was the wisest to date. But the scheme encountered opposition in August with the arrival of between 185 and 270 immigrants. Smith was able to dispatch a third of the colonists to Nansemond on the south side of the river in the saltwater zone and another third to the freshwater zone at the falls near the head of the James. But the rest stayed in Jamestown, assuredly against Smith’s better judgment. Predictably, sickness ravaged 100 Jamestown colonists, and 50 died by October. Yet at Nansemond and at the falls, few sickened and none died.34 Indian behavior had given Smith the key to life in the James estuary, but this precious knowledge was soon lost. He was relieved of the presidency in October and returned to England: with him went the schemes of seminomadism and summer dispersal. The colony once again clustered at Jamestown, and death hung heavy over the settlement.

Between Smith’s departure and Thomas Gates’s arrival in May 1610, the colony experienced the infamous “starving time.” The accounts of hundreds starving, of cannibalism and other inhumanities, have proved irresistible. But these accounts are biased, sensationalized, and exaggerated. They have warped the death rate and its causes out of all proportion and have diverted attention from the summer epidemics. In the first place, the death toll in the winter of 1609–1610 was much less than is usually assumed. The most common error has been the belief that 490 to 500 immigrants came to Virginia in October, with just 50 to 60 surviving when Gates arrived in May 1610—meaning that over 400 died.35 In fact the Virginia population in October stood at 250 or less, and after Smith departed with 30 unruuly youths, 220 colonists remained. At least 15 of them were killed by Indians, and 25 to 30 others returned to England, leaving 180 in the colony.36 When Gates arrived in May, he found 40 men in good health, along with President Percy, at Point Comfort near the mouth of the James. And at Jamestown 60 ragged men dragged out to meet Gates.37 In other words, 100 survived the winter, 15 were killed by the Indians, and 80 died from other causes.

Was starvation the cause of death? Enemies of the company and of Sir Thomas Smythe’s administration placed the blame on starvation resulting from inadequate provisions. Purportedly, “famine compelled us wholly to devour those Hogs, Dogges and horses that were then in the Collony,” along with vermin and human flesh.38 Yet there are serious inconsistencies surrounding the “starving time.” Gates reported that 600 hogs were destroyed, which at conservative dressweights of 50 pounds per hog amounted to 30,000 pounds for 200 colonists or less—or about 150 pounds per capita during the seven months.39 And supplemented by 500 chickens, seven horses, dogs, rats, snakes, and other vermin, the colonists’ diet seems sufficient to have warded off starvation—even without human flesh.40 Gates offered a different interpretation. He noted that Powhatan stepped up hostilities, confining the colonists to Jamestown between October and May. Some of the colonists were murdered, others fled, “and most by drinking of the brackish water of James Fort weakened and endangered, famine and sickness by all these means increased.”41 Brackish water, probably contaminated with typhoid and dysentery, is implicated once again, but this time in winter. One source of salty


37. Percy’s abandonment of Jamestown for the healthier Point Comfort site was pragmatic, but not the most heroic of gestures. Wright, ed., Voyage to Virginia: Two Narratives, 62–63.


40. Gates’s hog estimate was confirmed by Smith. Ibid.; Arber and Bradley, eds., Travels and Works of Captain John Smith, 1, 167.

41. The tragedy was also blamed on “idleness,” perhaps the result of salt intoxication. Wright, ed., Voyage to Virginia: Two Narratives, 98–99.
water was, of course, shallow wells, tapping brackish aquifers contaminated by pathogens percolated downward into the ground water. Another possible source of bad water was the river, contaminated during the severely cold winter of 1609–1610. Climatologists have observed that cold temperatures and subsiding air depress rainfall. A cold, dry winter—common in many parts of the mid-latitudes during the late fifteenth and early sixteenth centuries—would have lowered river discharge and delayed the retreat of the estuarine salt incursion, fecal material, and sediment from Jamestown.42

We cannot say conclusively that typhoid, dysentery, and salt poisoning were the principal causes of death in that winter; however, we can suggest that the case for massive starvation is far from proven. For instance, the mortality rate of 44.4 percent is much lower than the rate usually suggested by proponents of the "starving time." Starvation appears dubious given the livestock available to be consumed in the winter. Moreover, the mortality rate is very similar to expected and observed rates of death from typhoid and dysentery for 1607 and 1608. Finally, winter mortality in early Virginia was rare except in extremely severe winters (for example, 1607–1608 and 1609–1610).43 Cold, dry winters and estuarine hydraulics could have produced a contaminated water supply and epidemic typhoid and dysentery in the so-called starving time.

With the arrival of Lord De la Warr in June of 1610, Jamestown was retained as the colony’s center. In mid-June 350 people were alive, the sickness began one month later, and 150 (43 percent) had died by the end of the summer. By April 1611, 50 more died.44 Colonial leaders strongly suspected the Jamestown water supply as the cause of death. Gates and William Strachey stated as much, and Gates and De la Warr, on their return to England in the fall of 1610, communicated their fears to the Virginia Company leaders.45 Jamestown’s days were numbered, or so it seemed.

The establishment of a healthier town site took time, and meanwhile the summer death continued. Thomas Dale arrived in Virginia on May 22, 1611, with 300 colonists, bringing the colony’s strength to 480.46 By mid-June, Dale had chosen a new town site—the falls at the head of the James—but building did not commence until September. The colonists spent the summer in Jamestown, with the sickness beginning in early July. A few days later Dale instituted martial law, but tough discipline did not thwart disease. At least 240 of the colonists became so sick that they could not work.47 A death toll of about a third of the population, or 160, would be consistent with summer mortality and with later population estimates. At summer’s end in 1611 the colony’s population stood as follows: Dale’s 320 survivors, plus 300 immigrants brought by Gates in August, all of whom were evacuated to the healthier falls site, and 62 brought by Argall in late September putting the colony total at 682—a figure just slightly below the 700 estimated for early 1612 by a Spanish prisoner at Jamestown.48

43. C. P. Brooks, Climate Through the Ages: A Study of the Climatic Factors and Their Variations, 2d rev. ed. (New York, 1970), 359–378. The extremely cold winter of 1609–1610 is noted in Brown, First Republic in America, 113. Salty water at Jamestown in 1609–1610 would explain the presence of water too cold to wade in for oysters, yet unfrozen because the salt incursion lowered the freezing point. “A Briefe Declaration,” Virginia Col. Records, 71. Twentieth-century records of the James River provide evidence of winter discharge falling below late summer levels. However, salinity records are too recent and spotty to indicate a winter saltwater incursion. Virginia Conservation Commission, Surface Water Supply.
44. Arber and Bradley, eds., Travels and Works of Captain John Smith, I, 23, 98. Brown estimates that 57 of 110 colonists died between January and April 1608, and that 25 of 83 died between April and October 1608. The winter mortality of 52 percent exceeds the 44 percent rate (80/180) for the cold winter of 1609–1610. Brown, First Republic in America, 57; Arber and Bradley, eds., Travels and Works of Captain John Smith, II, 398, 407, 434.
45. For Strachey’s insights, see Wright, ed., Voyage to Virginia: Two Narratives, 82–83; Morton, Colonial Virginia, I, 28–29.
46. In March 1610, 150 were alive; 30 arrived soon after, and Dale brought 300 in May. By October 1610—a total of 480. Brown, First Republic in America, 138–139; 149; Ralph H. Hamor, A True Discourse of the Present Estate of Virginia, and the success of the affairs there till the 18 of June, 1614 . . . (London, 1615), 26.
48. Brown, First Republic in America, 156, 172; Arber and Bradley, eds., Travels and
Rome was not built in a day, nor was Henrico, the new town at the falls. Construction began in the autumn of 1611 and continued through winter. But the schedule was interrupted by spring planting in 1612, and full-scale settlement of the new town awaited the end of the harvest. My guess is that the majority of colonists spent the summer of 1612 at Jamestown. The mortality figures suggest as much. According to the Spanish prisoner Molina, 350 died out of a total population of 700. Molina’s report appears accurate; 700 colonists seems about right for the spring of 1612. Molina, however, gives two estimates of the survivors in May 1613—either 305 or 350. The death rate for 1612-1613 was probably 50 percent or more.49

With the establishment of Henrico and the general dispersal of population between 1613 and 1616, early Virginia enjoyed its healthiest era. By 1614 Jamestown had dwindled as the colony’s center, and the population shifted toward the head of the James River. Rolfe’s description of settlement in 1616 revealed that Jamestown contained just 19 percent of the colony population, and just 32.3 percent resided in both the oligohaline (Dale’s Gift and Jamestown) and the saltwater (Kecoughtan). The remaining 67.7 percent occupied the freshwater zone at Henrico, Bermuda Nether Hundred, and West Sherley Hundred.50 Mortality was rarely mentioned in the contemporary correspondence or accounts of these years, and for good reason. The population in May 1613 consisted of 305 to 350 persons, and by May 1616, 45 immigrants had arrived and 351 colonists survived.51 The mortality rate had declined sharply. Assuming no natural increase, the Virginia population either held steady or declined at a rate of about fifteen deaths per year—an astonishing annual mortality rate of about 3.8 percent.

The marked improvement in mortality rates following the redistribution of population into healthier freshwater environments is consistent with the model used here; however, the causal role of dispersal is clouded by a simultaneous reduction in immigration. The latter explanation for declining mortality is favored by proponents of the “seasoning” thesis. They maintain that mortality rates among immigrants were very high, but fell sharply among the survivors, who were “seasoned” to the Virginia disease environment. The seasoned survivors were less susceptible (immune) to disease in future years. An alternative hypothesis, and the one favored here, maintains that seasoned colonists were nearly as susceptible as newcomers to typhoid, dysentery, and salt poisoning and that their vulnerability can be demonstrated for the period under discussion. Immigration to Virginia came to a virtual standstill in the summer of 1611. The seasoning thesis would posit a sequence of high mortality rates in that summer, the survival of seasoned colonists, and a sharp mortality reduction in the summer of 1612. In fact, we have shown that mortality remained high in both summers. Death rates dropped dramatically in 1613, after the Virginians shifted their settlements into the freshwater zone. Environment and location were the decisive factors lowering mortality between 1613 and 1616; immigration and seasoning were largely irrelevant.52

The healthy era, 1613 to 1616, was the product of a lengthy and painful process of environmental learning and adjustment. The three years from 1607 to 1610 were spent enduring death and identifying its nature.

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49. As late as May 1613, Jamestown contained almost half of the colony’s population and Henrico only a third. The pattern surely changed by the summer of 1613. Works of Captain John Smith, II, 509; “Letter of Don Diego de Molina, 1613,” in Tyler, ed., Narratives of Early Virginia, 220, 223-224.

50. John Rolfe, A True Relation of the state of Virginia Left by Sir Thomas Dale, Knight, in May last 1616 (New Haven, Conn., 1951), 33-41; Hamor, A True Discourse, 26-33; Charles E. Hatch Jr., The First Seventeen Years: Virginia, 1607-1624, Jamestown 350th Anniversary Historical Booklet, no. 6 (Williamsburg, Va., 1957), 32-33.

51. John Rolfe, A True Relation of the state of Virginia Left by Sir Thomas Dale, Knight, in May last 1616 (New Haven, Conn., 1951), 33-41; Hamor, A True Discourse, 26-33.

52. Seasoning was at a once a well-recognized process in Virginia and a theory of curative medicine, i.e., treatment of individuals. By exposing the individual to infection in a new disease environment, future susceptibility was reduced. The theory worked fine for self-immunizing or debilitating diseases such as malaria, bacillary dysentery, paratyphoid, but curative medicine worked miserably on non-immunizing, virulent diseases. Exposure to these diseases brought death year after year. The only effective remedy against them, at least before vaccines and antibiotics, was the preventive medicine of environmental modification or avoidance. Since Virginians were powerless to change the oligohaline, the best course was to avoid the zone. An excellent discussion of the seasoning process and the role of malaria is Darrett R. Rutman and Anita H. Rutman, “Of Aigus and Fevers: Malaria in the Early Chesapeake,” WMQ, 3d Ser., XXXIII (1976), 31-60. Blanton, Medicine in Virginia, 37-41; Colonel P. M. Ashburn, The Ranks of Death: A Medical History of the Conquest of America, ed. Frank D. Ashburn (New York, 1947), 118-123, 159-160; Duffy, Epidemics in Colonial America, 214-218; May, Ecology of Human Disease, 26.
geographic pattern and its causes. By the spring of 1610 colonial leaders had associated death with the water supply and the Jamestown environment. They persuaded the company in London of this pattern by the fall of that year. Implementation of a new settlement distribution consumed the next three years from the winter of 1610–1611 to the fall of 1613. During these years Dale reconnoitered, chose a site, began construction of Henrico, cleared land for crops, and instituted an aggressive Indian campaign. Thus, in seven years the company had perceived the solution to summer mortality, and Dale worked swiftly toward that end. But the resettlement scheme was greatly facilitated by the Indians. Dale’s provocative encroachment into Powhatan’s territory at the head of the James met little resistance from the chief. Perhaps Powhatan’s advanced age and the capture of his daughter Pocahontas tempered his retaliation, but his mysterious behavior suggests intrigue. The chief remained incommunicado from May 1613 to March 1614, and he removed his quarters from the James to the Pamunkey River. Is it not possible that Powhatan had his hands full with the hostile Monacans on his western flank, and until they were subdued, he temporarily conceded Dale the James River head? Whatever Powhatan’s motives, the English colony profited from his passivity.53

When Dale left Virginia in the spring of 1616, he felt confident that the colony would endure. The mortality problem had been solved by diminishing Jamestown’s importance and locating the settlements in healthier zones. With sickness and death on the wane, the healthy colonists produced a surplus of food. Trade relationships altered. Formerly the colonists begged, stole, or traded for Indian food; now the Indians came seeking the colony’s corn.54 Healthy conditions continued through the summer of 1616. There were no reports of widespread mortality, and the colony probably contained 335 to 351 colonists. But with the arrival of a new governor in the spring of 1617, all of Dale’s insights were abandoned, to be painfully relearned.


54. Rolfe, A True Relation, 36.

Governor Samuel Argall was not one to learn from his mistakes. This was the same Argall who earlier had condemned Smith’s dispersal of colonists in the summer of 1609. As governor in the spring of 1617, Argall was again appalled by the state of the colony and Jamestown where “he found but five or six houses, the Church downe, the Palizado’s broken, the Bridge in pieces, the Well of fresh water spoiled; the Store-house they used for the Church; the marketplace, and streets, and all other spare places planted with Tobacco: . . . the Colonie dispersed all about, planting Tobacco.”55 On June 9 Argall wrote the company that he liked “James Town better than Bermudas 40 miles above it, [and] will Strengthen it.”56 Argall must have succeeded in realigning settlement, for that summer a great mortality ensued. Death struck 105 to 115 of the 415 colonists, and suddenly the mortality rate had risen from almost nil to 25 percent.57

The realignment of settlement begun by Argall and continued under the Sandys administration was one of the principal causes of death until 1624. The hard-won knowledge of the environment and the adjustments made between 1607 and 1617 were abandoned. Between 1617 and 1623, 36 new settlements dotted the James estuary, and 13 of them occupied the oligohaline and the saltier water.58 More important, population shifted into the lower estuary, and Jamestown was reaffirmed as chief city and center of government. The extent of realignment is revealed by the census of 1623–1624, and by the records of deaths from the massacre of March 1622, both of which help provide a more accurate picture of population distribution from 1618 to 1622 (see table 1).59 These sources show that 72 percent of the


57. Ibid., 92. The mortality rate is based on the following: probably 335 (my estimate) were alive in May 1617, plus 80 brought in by Argall, or a total of 415. In May 1618 the colony contained about 400, 90 to 100 of whom had arrived between March and May 1618. Subtracting these from the 400 yields 300 to 310 alive in March 1618. Thus from May 1617 to March 1618, 105 to 115 had died. Arber and Bradley, eds., Travels and Works of Captain John Smith, II, 330–336; Brown, First Republic in America, 253–256, 260, 277; Hecht, “The Virginia Colony, 1607–1640,” 333–334; Evarts B. Greene and Virginia D. Harrington, American Population before the Federal Census of 1790 (New York, 1932), 135.


TABLE 1. Population Distribution in 1622–1624 (and the Probable Distribution from 1617 to 1624)

<table>
<thead>
<tr>
<th>Estuarine Zone</th>
<th>Massacre Deaths</th>
<th>Living, 1623–1624</th>
<th>Dead, 1623–1624</th>
<th>Total 1623–1624</th>
<th>Percentage of Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Freshwater</td>
<td>209</td>
<td>289</td>
<td>57</td>
<td>555</td>
<td>28.5%</td>
</tr>
<tr>
<td>Oligohaline</td>
<td>145</td>
<td>693</td>
<td>211</td>
<td>959</td>
<td>49.3%</td>
</tr>
<tr>
<td>Mesohaline, Polyhaline</td>
<td>0</td>
<td>303</td>
<td>101</td>
<td>431</td>
<td>22.2%</td>
</tr>
</tbody>
</table>

Note: This table does not include population on the Eastern Shore or on recently arrived vessels.

Virginia colonists resided in the oligohaline and saltwater zones; 28 percent occupied the freshwater—almost a direct reversal of the pattern under Dale, when 68 percent lived in the freshwater zone.

Increased mortality accompanied the shift in population. Several thousand colonists died between 1618 and 1624, and disease was an important cause. Comments on summer sickness and death increasingly punctuated colonial correspondence. But disease was not the sole killer. Indian attacks, starvation, and plague also contributed. While the surviving evidence precludes a precise bill of mortality, some estimates of disease-related deaths can be made from the census of 1623–1624.

Here 1623–1624 is used as a benchmark year for estimating the usual mortality rate from typhoid, dysentery, and salt poisoning from 1618 to 1624. Several bits of evidence suggest these diseases as the principal causes of death in 1623–1624: the reports of summer sickness and death in that year; the absence of other reported causes of mortality; an abundant food supply, making starvation an unlikely cause of death; and the census listing of colonists killed, presumably by the Indians, so that these deaths can be excluded from our disease estimate.\(^{60}\) Typhoid and dysentery are also implicated by the spatial pattern of death recorded in the census of 1623–1624. Within those settlements reporting deaths during the year, 16.7 percent died in the freshwater zone; 37.1 percent in the oligohaline; and 23.3 percent in the saltier portion of the James estuary (table 2).\(^{61}\) The match between reality and our estuarine model is good, but not perfect. Freshwater death rates are higher than expected, perhaps reflecting the severe disruptions in this area caused by the massacre of 1622. Another peculiarity is Hog Island in the oligohaline, where only 8.8 percent died. A safer right-bank location, the removal of pollutants toward the north bank by a river meander, and the small population probably combined to make Hog Island a healthier micro-environment. Otherwise the census pattern points toward death by typhoid and dysentery in the oligohaline and the salty lower James.

Having isolated these diseases as probable causes of death, we can estimate their usual contribution to Virginia mortality. The annual

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60. Brown, First Republic in America, 569–570; Edmund S. Morgan, American Slavery, American Freedom: The Ordeal of Colonial Virginia (New York, 1975), 104–105. The year following the massacre of 1622 was very sickly, but the resultant mortality probably antedated the census of 1623–1624. Tyler, ed., Narratives of Early Virginia, 438; Morton, Colonial Virginia, 1, 83–90.

TABLE 4. Disease-Related Death Estimates in Virginia, 1618–1624

<table>
<thead>
<tr>
<th>Time Period</th>
<th>Population at Beginning</th>
<th>Population at End</th>
<th>Immigrants</th>
<th>Overall Mortality Rate</th>
<th>Disease-Related Deaths</th>
<th>Disease Death Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dec. 1618–Mar. 1620</td>
<td>600</td>
<td>887</td>
<td>814–914</td>
<td>37.3%–41.4%</td>
<td>402–430</td>
<td>402–430</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(527–627)</td>
<td>(28.3%)</td>
<td>(527–627)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(1,414–1,514)</td>
<td>(68–76%)</td>
<td></td>
</tr>
<tr>
<td>Mar. 1620–Mar. 1621</td>
<td>887</td>
<td>843</td>
<td>1,051</td>
<td>56.5%</td>
<td>550/1,095</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(1,095/1,938)</td>
<td>(50.2%)</td>
<td></td>
</tr>
<tr>
<td>Mar. 1621–Mar. 1622</td>
<td>843</td>
<td>1,240</td>
<td>1,580</td>
<td>48.8%</td>
<td>688/1,183</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(1,183/2,423)</td>
<td>(58.2%)</td>
<td></td>
</tr>
<tr>
<td>Mar. 1622–Apr. 1623</td>
<td>1,240</td>
<td>1,241</td>
<td>695</td>
<td>35.9%</td>
<td>347/694</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(694/1,935)</td>
<td>(50.0%)</td>
<td></td>
</tr>
<tr>
<td>Apr. 1623–Feb. 1624</td>
<td>1,241</td>
<td>1,275</td>
<td>405</td>
<td>22.5%</td>
<td>371/371</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(371/1,646)</td>
<td>(100.0%)</td>
<td></td>
</tr>
</tbody>
</table>

Summary:

- Population in Feb. 1624: 1,275
- Disease-related deaths, 1618–1624: 2,538
- Other causes of death or return to England: 1,332
- Total: 5,145

Notes:
- Includes deaths from all causes as well as those returning to England alive.
- Using estimates of 28.4% per year.
- Since 347 known deaths occurred in the massacre, the remainder are assigned to disease.
- Overall death rate fell below the disease rate, hence all were assigned to disease.
principal killers in some years, and they were significant contributors in all.

The leaders of the company and the colony tried desperately to reduce summer mortality but failed because of their misconceptions of its causes. Preventive measures were aimed at the immigrants and not at the environment and at population distribution. Guest houses (hospitals) were established, and immigrant arrivals were scheduled for fall after the sickly summer months, all done on the false assumption that seasoned colonists would survive. But seasoned colonists stood little chance of survival in the oligohaline zone, as revealed in the muser of 1625. Then 57 settlers gave arrival dates before 1616; 24 resided in the fresher, 25 in the salt, and just 9 in the oligohaline. Older settlers, those arriving before 1620 of all giving arrival dates, made up about one-fifth of the population in the oligohaline, one-third in the saltwater zone, and two-fifths in the fresh. These spatial and environmental patterns of death went undetected by the company, and that oversight was instrumental in its dissolution.

The demise of the Virginia Company in 1624 signaled a new era in Virginia demography. The old constraints focusing the colony on Jamestown and the oligohaline were relaxed, and mortality fell. Between 1625 and 1634 Virginia's population grew from 1,210 to 4,914.

63. These estimates of annual disease mortality permit an assessment of other causes of death. Morgan, for example, has suggested starvation and malnutrition, occasions by control of Virginia's food and labor supply by a handful of private capitalists. The most likely years for such class behavior were 1620 to 1622, when causes other than Indian killings and diseases contributed 40 to 50 percent of all deaths. Morgan, American Slavery, American Freedom, 92-107. Note, however, that immigration was also heaviest in these years—suggesting Craven's point of inadequate provisioning of the immigrants by the company. Probably both company and private wheeler were responsible for the increased death rate; in any case, the critical years were 1620-1622. Wesley Frank Craven, Dissolution of the Virginia Company. The Failure of a Colonial Experiment (New York, 1932), 152-153.


66. Morgan, American Slavery, American Freedom, 159, estimates 1,000 immigrants per year. Morgan understimates the magnitude of declining mortality rates after 1624. A lower death rate is not inconsistent with his literary evidence. Ship captains experienced high mortality (42 percent in 1636) precisely because they piled in the oligohaline zone. And 1,800 deaths in 1636, given the population and increased immigration in that year, produces a mortality rate in between that of the period 1618 to 1624 (28.3 percent) and our post-1624 estimated rate (14.2 percent).

67. Morgan's 1625 to 1640 estimate of 1,000 immigrants per year has been questioned as too high by Menard. He suggests that immigration varied directly with tobacco prices, and therefore Virginia immigration peaked at about 2,000 in 1635-1636 and in preceding years (1625-1635) immigration generally fell substantially below 1,000 per year. If Menard is correct, then annual mortality for the period 1625 to 1634 falls even lower than the 14.2 percent presented here. Russell R. Menard, "Economy and Society in Early Colonial Maryland" (Ph.D. dissertation, University of Iowa, 1975), 167-170.

### Table 5. Population Growth, 1625-1634, under Pre-1625 Disease Mortality Rates and a Fitted Mortality Rate

<table>
<thead>
<tr>
<th>Date</th>
<th>Population &amp; Immigrants</th>
<th>Survivors</th>
<th>Population &amp; Immigrants</th>
<th>Survivors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1625-1626</td>
<td>1,210 + 1,000</td>
<td>1,582</td>
<td>1,210 + 1,000</td>
<td>1,896</td>
</tr>
<tr>
<td>1626-1627</td>
<td>1,582 + 1,000</td>
<td>1,849</td>
<td>1,896 + 1,000</td>
<td>2,485</td>
</tr>
<tr>
<td>1627-1628</td>
<td>1,849 + 1,000</td>
<td>2,040</td>
<td>2,485 + 1,000</td>
<td>2,990</td>
</tr>
<tr>
<td>1628-1629</td>
<td>2,040 + 1,000</td>
<td>2,177</td>
<td>2,990 + 1,000</td>
<td>3,423</td>
</tr>
<tr>
<td>1629-1630</td>
<td>2,177 + 1,000</td>
<td>2,275</td>
<td>3,423 + 1,000</td>
<td>3,794</td>
</tr>
<tr>
<td>1630-1631</td>
<td>2,275 + 1,000</td>
<td>2,344</td>
<td>3,794 + 1,000</td>
<td>4,113</td>
</tr>
<tr>
<td>1631-1632</td>
<td>2,344 + 1,000</td>
<td>2,394</td>
<td>4,113 + 1,000</td>
<td>4,387</td>
</tr>
<tr>
<td>1632-1633</td>
<td>2,394 + 1,000</td>
<td>2,430</td>
<td>4,387 + 1,000</td>
<td>4,622</td>
</tr>
<tr>
<td>1633-1634</td>
<td>2,430 + 1,000</td>
<td>2,456</td>
<td>4,622 + 1,000</td>
<td>4,824</td>
</tr>
<tr>
<td>1634-1635</td>
<td>2,456 + 1,000</td>
<td>2,475</td>
<td>4,824 + 1,000</td>
<td>4,997</td>
</tr>
</tbody>
</table>

Note: The actual population in 1634 was 4,914. The above calculations assume no natural increase. Some children were born in the colony during the period, but the imbalanced sex ratio favoring males, and other evidence suggests that children contributed little to population growth at this time. While receiving an estimated 9,000 immigrants. Although over half of the population died in the nine-year period, this figure obscures the marked improvement in annual mortality. Had the pre-1625 mortality rate of 28.3 percent per year continued, Virginia in 1634 would have numbered 2,456 instead of 4,914. In effect, annual mortality was cut in half (to about 14.2 percent) during the early royal period (see table 5).
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in the saltwater zones. While I cannot prove this statement from the available evidence, the oligohaline zone appears to have been more deadly in the eighteenth century. The spatial pattern of mortality can be crudely reconstructed from a 1725–1726 parish census of births and burials (figure 2). For all four Virginia estuaries, the pattern of mortality hypothesized by the estuarine model for early Virginia remains recognizable a century later. The proportion of burials to births generally is least in the freshwater zone, peaks in the oligohaline zone, and drops slightly in the saltwater zone of the lower estuaries. Left bank (north side) proportions are usually higher than the corresponding right bank (south side). The map's subtleties and its several departures from the model—notably the higher-than-expected mortality levels in the upper Rappahannock, lower James, and the Potomac estuary generally—warrant more attention, but to do so would push us beyond the limited scope of this essay. More important is the map's suggestion that typhoid and dysentery caused some

71. The census records births and burials for the year beginning April 15, 1725. I have assumed census recording procedures were uniform among Virginia parishes, but undoubtedly these procedures varied markedly. More thorough studies of Virginia demography may reveal whether the parishes departing from the estuarine-disease model reflect actual differences or recording biases in the census. C.O. 5/1320, f. 74. Public Record Office. Parish boundaries are roughly accurate. The four Henrico County parishes are aggregated on the map. Charles Francis Cockerell, Parish Lines, Diocese of Virginia. Virginia State Library Publications, no. 28 (Richmond, Va., 1967); George Carrington Mason, Colonial Churches of Tidewater Virginia (Richmond, Va., 1945). The freshwater–saltwater transition zones are located according to Nichols, "Sediments of the James River," 171–179. Even Reucchini, Virginia Institute of Marine Science, personal communication; H. C. Whaley and T. C. Hopkins, Atlas of the Salinity and Temperature Distribution of Chesapeake Bay, Chesapeake Bay Institute, Johns Hopkins University, Graphical Summary Reports, nos. 1–2, Ref. 52–4, 63–1 (Baltimore, 1952, 1963); Chirnan, A Huguenot Exile in Virginia, 174.

72. This geographical pattern of mortality might be explained by other models, such as the Rutman's malarial endemicity. According to the Rutman's, malarial "morbidly limbs as endemicity rises, since a greater percentage of infectious bites by Anopheles leads to symptomatic malarial attacks. Yet the rate of morbidity will be balanced at some point by the rate of immunities in the population and then will begin to decline until, in a hyperendemic situation, morbidity is largely limited to children, non-immune newcomers to the community, and pregnant women." Put geographically this process of endemicity should move roughly with the frontier of settlement, i.e., old settled areas being hyperendemic, newly settled areas having low but rising morbidity, and middle-aged areas having very high morbidity. If I have reasoned correctly, the entire James River area, as the oldest settled area, should have the highest values, whereas the Rappahannock should show much lower values on our map, followed by very high values in the middle-aged tier of Gloucester, Middlesex, Lancaster, Westmoreland, and Northumberland counties, and low values elsewhere. I do not detect such a pattern, and accordingly favor the three-zone estuarine model of enteric diseases. Rutman and Rutman, "Agues and Fevers," WMQ, 3d Ser., XXXIII (1976), 37–39, 44–45.
eighteenth-century mortality, perhaps enough to account for the high rates of the oligohaline zone and the intermediate rates in the saltwater zone.

The demographic history of early Virginia is both sad and tragic. Sad because so many died; tragic because they died needlessly. Smith, Dale, and others knew that epidemics of typhoid, dysentery, and salt poisoning were recurrent; they knew that these epidemics were spawned by a contaminated water supply in the vicinity of Jamestown and the freshwater-saltwater transition on the James estuary. They knew that dispersing in the summer or shifting permanently into the freshwater zone were the only ways to save lives. And they knew that scattered settlements required the protection of an aggressive Indian policy. Smith and Dale saved lives, but their insights were abandoned with the arrival of new colonial leaders or a new company administration. Jamestown was reclaimed, mortality rose, and the painful environmental learning process began again at ground level. The Sandys administration never learned. The nexus of environment and mortality confounded and eluded them. They mistakenly believed that the seasoning process would eventually take hold and Virginia’s population would grow. But typhoid and dysentery were no respecters of flawed theories of immunity. From a demographic standpoint, the best thing that happened in early Virginia was the dissolution of the company with its fixation on Jamestown.