Biological Consequences of European Expansion, 1450–1800

Volume XXVI

An Expanding World
The European Impact on World History
1450-1800

Edited by **Kenneth F. Kiple and Stephen V. Beck**

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An Expanding World
Volume 26

Biological Consequences of the European Expansion, 1450–1800

AN EXPANDING WORLD
The European Impact on World History, 1450–1800

General Editor: A.J.R. Russell-Wood

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General Editor's Preface

A.J.R. Russell-Wood

An Expanding World: The European Impact on World History, 1450–1800 is designed to meet two objectives: first, each volume covers a specific aspect of the European initiative and reaction across time and space; second, the series represents a superb overview and compendium of knowledge and is an invaluable reference source on the European presence beyond Europe in the early modern period, interaction with non-Europeans, and experiences of peoples of other continents, religions, and races in relation to Europe and Europeans. The series reflects revisionist interpretations and new approaches to what has been called 'the expansion of Europe' and whose historiography traditionally bore the hallmarks of a narrowly Eurocentric perspective, focus on the achievements of individual nations, and characterization of the European presence as one of dominance, conquest, and control. Fragmentation characterized much of this literature: fragmentation by national groups, by geography, and by chronology.

The volumes of *An Expanding World* seek to transcend nationalist histories and to examine on the global stage rather than in discrete regions important selected facets of the European presence overseas. One result has been to bring to the fore the multicontinental, multi-oceanic and multinational dimension of the European activities. A further outcome is compensatory in the emphasis placed on the cross-cultural context of European activities and on how collaboration and cooperation between peoples transcended real or perceived boundaries of religion, nationality, race, and language and were no less important aspects of the European experience in Africa, Asia, the Americas, and Australia than the highly publicized confrontational, bellicose, and exploitative dimensions. Recent scholarship has not only led to greater understanding of peoples, cultures, and institutions of Africa, Asia, the Americas, and Australasia with whom Europeans interacted and the complexity of such interactions and transactions, but also of relations between Europeans of different nationalities and religious persuasions.

The initial five volumes reflect the changing historiography and set the stage for volumes encompassing the broad themes of technology and science, trade and commerce, exploitation as reflected in agriculture and the extractive industries and through systems of forced and coerced labour, government of empire, and society and culture in European colonies and settlements overseas. Final volumes examine the image of Europe and Europeans as 'the other' and the impact of the wider world on European *mentalités* and mores.

An international team of editors was selected to reflect a diversity of educational backgrounds, nationalities, and scholars at different stages of their professional careers. Few would claim to be 'world historians', but each is a

recognized authority in his or her field and has the demonstrated capacity to ask the significant questions and provide a conceptual framework for the selection of articles which combine analysis with interpretation. Editors were exhorted to place their specific subjects within a global context and over the *longue durée*. I have been delighted by the enthusiasm with which they took up this intellectual challenge, their courage in venturing beyond their immediate research fields to look over the fences into the gardens of their academic neighbours, and the collegiality which has led to a generous informal exchange of information. Editors were posed the daunting task of surveying a rich historical literature and selecting those essays which they regarded as significant contributions to an understanding of the specific field or representative of the historiography. They were asked to give priority to articles in scholarly journals; essays from conference volumes and *Festschriften* were acceptable; excluded (with some few exceptions) were excerpts from recent monographs or paperback volumes. After much discussion and agonizing, the decision was taken to incorporate essays only in English, French, and Spanish. This has led to the exclusion of the extensive scholarly literature in Danish, Dutch, German and Portuguese. The ramifications of these decisions and how these have had an impact on the representative quality of selections of articles have varied, depending on the theme, and have been addressed by editors in their introductions.

The introduction to each volume enables readers to assess the importance of the topic *per se* and place this in the broader context of European activities overseas. It acquaints readers with broad trends in the historiography and alerts them to controversies and conflicting interpretations. Editors clarify the conceptual framework for each volume and explain the rationale for the selection of articles and how they relate to each other. Introductions permit volume editors to assess the impact on their treatments of discrete topics of constraints of language, format, and chronology, assess the completeness of the journal literature, and address *lacunae*. A further charge to editors was to describe and evaluate the importance of change over time, explain differences attributable to differing geographical, cultural, institutional, and economic circumstances and suggest the potential for cross-cultural, comparative, and interdisciplinary approaches. The addition of notes and bibliographies enhances the scholarly value of the introductions and suggests avenues for further enquiry.

I should like to express my thanks to the volume editors for their willing participation, enthusiasm, sage counsel, invaluable suggestions, and good judgment. Evidence of the timeliness and importance of the series was illustrated by the decision, based on extensive consultation with the scholarly community, to expand a series, which had originally been projected not to exceed eight volumes, to more than thirty volumes. It was John Smedley's initiative which gave rise to discussions as to the viability and need for such a series and he has overseen the publishing, publicity, and marketing of *An Expanding World*. As

General Editor, my task was greatly facilitated by the assistance of Dr Mark Steele who was initially responsible for the 'operations' component of the series as it got under way, latterly this assistance has been provided by staff at Variorum.

The Department of History,
The Johns Hopkins University

Introduction

Kenneth F. Kiple and Stephen V. Beck

Medical statistics have shown, in treating on the different races of mankind the dangers of changing one's position on the globe...

G. Pouchet (1864)[1]

Wherever the European has trod, death seems to pursue the aboriginal.

Charles Darwin (1836)[2]

This volume deals with some of the biological, and especially the medical and demographic consequences of the expansion of Europe. But its essays concentrate heavily on the American theatre of that expansion for the very good reason that the catastrophic die off of the American Indians after 1492 has proved to be a compelling topic of research for historians, and much has been written about it.[3]

In Africa, by contrast, it was the Europeans who were susceptible to disease, and there was relatively little they could do about the problem until the nineteenth century.[4] In Asia, although the Europeans brought syphilis to that part of the world, there was no dramatic melting away of populations. In fact, historically Europe came out a decided second-best in disease exchanges with Asia. The latter was the source of bubonic plague beginning in the fourteenth century, and Asiatic

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[1] G. Pouchet, *The Plurality of the Human Race*, 2nd edn., tr. and ed. H.J. Beavau (London, 1864), p. 92.

[2] Charles Darwin, *The Voyages of the Beagle* (New York, 1909), p. 459.

[3] It was during the 1940s that modern historians and other scholars began to take notice of the role of disease in the European conquest of the Americas. Two early studies are P.M. Ashburn's *The Ranks of Death: A Medical History of the Conquest of America* (New York, 1947), and Sherburne F. Cook's *The Conflict between the California Indian and White Civilization* (Berkeley, 1943). Cook and his colleague, Woodrow W. Borah, continued to work in this and related fields throughout their careers. More recent discussions of the subject are provided in David E. Stannard's *American Holocaust: The Conquest of the New World* (New York, 1992), and Alfred W. Crosby's *The Columbian Exchange: Biological and Cultural Consequences of 1492* (Westport, Conn., 1972). Broader discussions of the history of biological and epidemiological exchanges among peoples may be found in Crosby's *Ecological Imperialism: The Biological Expansion of Europe, 900–1900* (New York, 1986), and William H. McNeill's *Plagues and Peoples* (New York, 1977).

[4] K.G. Davies's 'The Living and the Dead: White Mortality in West Africa, 1684–1732', in eds., Stanley L. Engerman and Eugene D. Genovese, *Race and Slavery in the Western Hemisphere: Quantitative Studies* (Princeton, 1975), pp. 83–98, and Philip D. Curtin's *Death by Migration* (New York, 1989), illustrate the high 'relocation cost' to Europeans, meaning the price in disease and death, of moving into Africa and other tropical areas of the world before the later nineteenth century. As Curtin's findings indicate, after that time, medical advances and a better overall understanding of man's relationship with his environment combined to reduce this cost.

cholera in the nineteenth century – both plagues whose careers bracket our time period but fall outside of it.[5]

Thus it is the case that, outside of the Americas, it was only the new worlds of the Pacific and Oceania that experienced demographic disaster in the face of the biological Pandora's box opened by the Europeans as they knitted together the globe.

Although it was written in the Bible that 'God hath made of one blood all nations to dwell on the face of the earth' (St Paul, Acts 17:26), the suspicion must have arisen, as the European expansion got under way, that God had not intended the 'nations' in question to change their geographical position on that earth. The technology that carried the expedition of Vasco da Gama around the Cape and across the Indian Ocean to reach Calicut in May of 1498 had made it possible for humans to spend long months at sea. But during the lengthy outbound voyage, and again during the return to Lisbon, a mysterious disease broke out among the seamen which caused hands and feet to swell, gums to grow over the teeth, old wounds to open, and men to die.[6]

The disease was scurvy – brought on by months of vitamin C deprivation. It had doubtless been seen before by Europeans, but only rarely, under circumstances such as those produced by prolonged sieges. Yet prolonged sea voyages without fresh foods provided an even better milieu for the blossoming of scurvy and from this point forward, as Europeans increasingly took to the oceans of the world for exploration, for conquest, and for the defense of the spoils of conquest, the disease became an implacable scourge of seamen, killing more than a million of them by the nineteenth century – many more than those killed by all the naval battles, storms, and shipwrecks of the intervening centuries put together.[7]

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[5] Philip Ziegler's *The Black Death* (New York, 1969) is still a good introduction to the history of bubonic plague in Europe. For cholera, see Dhiman Barua and William Burrows, eds., *Cholera* (Philadelphia, 1974), and Reinhard S. Speck, 'Cholera,' in ed. Kenneth F. Kiple, *The Cambridge World History of Human Disease* (new York, 1993), pp. 642–9. McNeill, in *Plagues and Peoples*, discusses these pandemics as well. In connection with such major disease exchanges, it is worth noting here McNeill's concept of regional 'disease pools' which may differ greatly from one another. Thus, over time, various peoples have developed varying 'pools' of immunities to disease, which may prove inadequate in the face of new or repeated introductions of diseases from disparate regions. But despite occasional catastrophes resulting from disease exchanges among the regions of the Old World, the frequency and longevity of contacts among them ensured that their peoples had many immunities in common, which were not shared by the more isolated peoples of the New Worlds.

[6] Da Gama's description of the disease is quoted in Kenneth J. Carpenter, *The History of Scurvy and Vitamin C* (Cambridge, 1986), pp. 1–2.

[7] This discussion is based on Carpenter, *History of Scurvy*, pp. 1–76, and R.E. Hughes, 'Scurvy', in ed. Kenneth F. Kiple, *The Cambridge World History of Food and Nutrition* (forthcoming).

Meanwhile, other evidence indicating the perils of venturing to remote lands had been unfolding in Europe. In their ventures down and around the African coastline, the Portuguese had contracted *falciparum* malaria, the most dangerous of the malaria types, and brought it back to Iberia, whereupon it practically depopulated much of the Tagus valley.[8] But an even more dramatic outcome of venturing far from home began to emerge as the fifteenth century came to a close. It seemed that Columbus had scarcely returned from his first voyage to the Americans when another, apparently new, disease burst upon the Europeans in their own homelands. It may be that syphilis had been around Europe in a less malignant form for eons. But it would seem that venereal syphilis, at least in epidemic form, made its European debut in Spain, or Italy, or France in 1493 or 1494 before it erupted in late 1495 among the soldiers of both France and Spain during the Italian Wars.[9]

Because that epidemic began during the French siege of Naples, syphilis was known initially as the 'disease of Naples'. Yet after the French retreat, the disbanding of their multinational army saw the rapid spread of syphilis across Europe, whereupon it became known as the 'French disease' by everyone except, of course, the French. Early in the sixteenth century, however, a Spanish physician suggested that the sailors of Columbus had contracted the disease in the Caribbean during his first voyage and brought it back to Europe, thus giving rise to the Columbian theory of the origin of epidemic venereal syphilis.[10]

There is no question that syphilis in Europe acted like a new disease among inexperienced peoples. It followed a classic pattern of raging among them with extraordinary virulence during much of the sixteenth century, only to become increasingly less malignant afterwards – so much so that by the eighteenth century it was routinely confused with gonorrhea. But despite its apparently exotic nature there were contemporary physicians who stated flatly that they had treated syphilis prior to 1492, albeit in a less severe form.[11]

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[8] Ralph Linton, *The Tree of Culture* (New York, 1955), p. 27.

[9] The following summary of the early history of syphilis and the debate over its origins is based on Crosby's discussion in *The Columbian Exchange*, pp. 122–64. For further details of the spread of the disease in Europe, and a survey of early writings on the subject, see J. Johnston Abraham, 'The Early History of Syphilis', *British Journal of Surgery* XXXII, no. 126 (1944), pp. 225–37.

[10] Crosby, in *The Columbian Exchange*, pp. 144–7, seemingly favours the Columbian theory, albeit with appropriate disclaimers, and Saul Jarcho briefly states a case for it in 'Some Observations on Disease in Prehistoric North America', *Bulletin of the History of Medicine* XXXVIII (1964), pp. 11–15. In addition, it has been speculated that some New World sculpture suggests the pre-Columbian presence of syphilis or a similar disease; see Abner I. Weisman, 'Syphilis: Was It Endemic in Pre-Columbian America or Was It Brought Here from Europe?', *Bulletin of the New York Academy of Medicine* XLII, no. 4 (1966), pp. 284–300.

[11] A case for the Old World origin of syphilis is presented in Charles Clayton Dennie's *The Gift of Columbus* (Kansas City, 1936), pp. 13–35 and *passim*. For another discussion of the

These two apparently irreconcilable theories of the origin of syphilis in Europe had the field to themselves until the twentieth century, with the Columbian theory clearly the front runner. But syphilis is only one of the treponemal diseases, and other theories emerged following the realization that the causative agents of all of them – pinta, non-venereal syphilis, yaws, and syphilis – were indistinguish-able under the microscope.[12]

Among these theories was the so-called 'unitarian' concept of the treponemata, first proposed by E.H. Hudson in 1946 and subsequently restated and defended a number of times. It held that all of the treponemal diseases, although differing in symptoms, are caused by the same organism which is transmitted differently in different climates. The venereal transmission of syphilis simply represented the adaptation of the treponemata to cooler climates with adult to adult transmission. In tropical climates where the pathogens of pinta and yaws could exist outside the body, they travelled from skin to skin (usually from child to child). In the case of non-venereal syphilis, in hot dry climates, the agents moved via mucosal tissue, as, for example, when children used the same eating utensils or when people kissed.[13]

The unitarian theory does not exclude the Columbian theory, but it has mostly been viewed as supporting the notion of an Old World development of syphilis. In an exploration of the problem of 'The Origin and Antiquity of Syphilis' (Chapter 1), Brenda J. Baker and George J. Armelagos exhaustively review these and other theories in the light of current historical and anthropological evidence. On the basis of skeletal evidence they conclude that non-venereal syphilis was in the Americas prior to 1492 and suggest this was the fountain of what became an Old World plague after that date. But how non-venereal syphilis in the New World became epidemic venereal syphilis in the Old still awaits full explanation.[14]

Yet even if it turns out that contact with the Americas did mean syphilis first for Europe, and then for the rest of the globe as wandering Europeans spread it

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disease's Old World origins, see C.J. Hackett, 'On the Origin of the Human Treponematoses (Pinta, Yaws, Endemic Syphilis, and Venereal Syphilis)', *Bulletin of the World Health Organization* XXIX (1963), 7–41.

[12] Crosby, *Columbian Exchange*, pp. 123, 141–4.

[13] E.H. Hudson, 'Treponematosis', in ed. Henry A. Christian, *The Oxford Textbook of Medicine*, 8 vols. (Oxford, 1949), Vol. 5, pp. 9–121. Hudson further postulated that the slave trade's forced migration of millions of Africans to other regions of the globe helped to spread *Treponema pallidum* beyond its putative homeland. In cooler, 'more civilized' environments, the importation of Africans carrying non-venereal treponemal infections may have created 'foci for the local propagation of venereal syphilis'. For this discussion, see Hudson, 'Treponematosis and African Slavery', *British Journal of Venereal Diseases* XL (1964), pp. 43–52.

[14] For further discussion of the history of syphilis, see Jon Arrizabalaga's 'Syphilis', and Kenneth F. Kiple's 'Syphilis, Nonvenereal', both in ed. Kenneth F. Kiple, *Cambridge World History of Human Disease*, 1025–1035.

about, this was small revenge for the pathogenic holocaust inadvertently unleashed on New World peoples by Old World pathogens.[15] A crossfire of debate continues over questions concerning the die off of the Native Americans after 1492, not least over questions having to do with the magnitude of that die off. But lively arguments have also taken place over how much blame Iberian procedures, policies, and just plain greed must share with epidemiological factors; and over the extent to which the first Americans were 'virgin soil' peoples for Old World diseases: and even over exactly who the first Americans were and how they became so 'liable' to the pathogens that reached them from Europe and from Africa.[16]

Firm answers have proven elusive. Most probably there were no fewer than 50 million 'Indians' in the Americas when Columbus first set food on Hispaniola and no more than 100 million.[17] Perhaps, as conventional wisdom would have

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[15] Early accounts of the matter may be found in Bartolome de las Casas, *Historia de las Indias*, 3 vols. (Mexico City, 1951); Gonzalo Fernandez de Oviedo, *Historia general y natural de las Indias, islas, y tierra firme del mar oceano ...*, 4 vols. (Madrid, 1851–5); and Antonio de Herrera y Tordesillas, *Historia general de los hechos de los Castellanos en las islas y tirra firme en el mar oceano*, 17 vols. (Buenos Aires, 1945).

[16] These debates are not confined to questions of factual detail or interpretational nuance; some of the most fundamental findings within the field have been challenged. For a recent provocative critique, see Francis J. Brooks, 'Revising the Conquest of Mexico: Smallpox, Sources, and Populations', *Journal of Interdisciplinary History* XXIV, no. 1 (1993), pp. 1–29. Brooks suggests that the accepted account of the role of disease in the Spanish conquest of Mexico is largely 'false, epidemiologically improbable, historiographically suspect, [and] logically dubious'. Such criticism performs the highly desirable function of helping to synthesize recent research, and of forcing scholars to remain aware of conclusions needing further refinement.

[17] Population estimates for the Western Hemisphere in 1492 have ranged from 8.4 million to well over 100 million persons. Douglas H. Ubelaker's, 'Prehistoric New World Population Size: Historical Review and Current Appraisal of North American Estimates', *American Journal of Physical Anthropology* LXV, no. 3, part II (1976), pp. 661–5, and David Henige's, 'Native American Population at Contact: Discursive Strategies and Standards of Proof in the Debate', *Latin American Population History Bulletin* (1992), pp. 2–23, provide a good overview of much of the scholarship in this area. Many, but not all, scholars have become inclined to accept higher estimates than formerly.

Various aspects of the population debate in recent decades may be followed by referring to essays by Francisco Guerra and M.C. Sanchez Tellez, 'Missionary Reports from Mexico (1550–1563): Estimates on Native Population Decline', *Latin American Population History Bulletin* XXV (1994), pp. 23–5; Michael E. Smith, 'Hernan Cortes on the Size of Aztec Cities', *Latin American Population History Bulletin* XXV (1994), pp. 25–7; Henry F. Dobyns, 'Building Stones and Paper: Evidence of Native American Historical Numbers', *Latin American Population History Bulletin* XXIV (1993), pp. 11–19; Woodrow Borah, Thomas Whitmore, Peter Gerhard, Francisco Guerra, and David Henige (Contributors), 'Debate: 16th-century Demographic Collapse', *Latin American Population History Bulletin* XXIII (1993), pp. 16–23; Thomas M. Whitmore, 'Sixteenth-Century Population Decline in the Basin of Mexico: A Systems Simulation', *Latin American Population History Bulletin* XX (1991), pp. 2–18; Henry F. Dobyns, Dean R. Snow and Kim M. Lanphear, and David Henige (Contributors), 'Commentary on Native American Demography', *Ethnohistory* XXXVI, no. 3 (1989), pp. 285–307; Marshall T. Newman, 'Aboriginal New World Epidemiology

it, the first Americans crossed in many waves from Siberia to Alaska via the land bridge of the Bering Straits, exposed during the last Ice Age. Or perhaps, as has recently been proposed based on genetic data, at least some arrived by sea from Polynesia.[18] But either way, they would have reached the New World before the Old began its Neolithic Revolution. The invention of agriculture, the domestication of animals, and the stimulus such activities provided for urban life, combined to form the Old World crucible for many of the most important epidemic diseases of humankind.[19]

This is not to suggest that the Americas were a disease-free Eden prior to 1492. Although they lagged substantially behind the Old World, New World peoples did mount their own Neolithic revolution that, in fostering sedentarism, doubtless encouraged the proliferation of water-borne diseases such as hepatitis and polio, as well as a variety of intestinal parasites. Present also were encephalitis, arthritis, pinta, and tuberculosis, as well as some uniquely American infections such as Chagas' Disease and American leishmaniasis. But until the Europeans and the Africans arrived, the first Americans had been spared a veritable gauntlet of epidemic diseases that, many have suggested, may have reduced their numbers by a staggering ninety percent.[20]

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and Medical Care, and the Impact of Old World Disease Imports', *American Journal of Physical Anthropology* LXV, no. 3, part II (1976), pp. 667–72; Wilbur R. Jacobs, 'The Trip of an Iceberg: Pre-Columbian Indian Demography and Some Implications for Revisionism', *William and Mary Quarterly* (3rd series) XXXI, no. 1 (1974), pp. 123–32; Woodrow Borah and Sherburne F. Cook, 'Conquest and Population: A Demographic Approach to Mexican History', *Proceedings of the American Philosophical Society* CXIII, no. 2 (1969), pp. 177–83; and Henry F. Dobyns, 'Estimating Aboriginal American Population: An Appraisal of Techniques with a New Hemispheric Estimate', *Current Anthropology* VII (1966), pp. 395–416.

In addition to the journal literature, a few books have made important contributions to an understanding of aspects of New World demographic history. The most recent is the second edition of William M. Denevan's *The Native Population of the Americas in 1492* (Madison, Wis., 1992). Another recent work, if more generalized, is editor David Hurst Thomas's *Columbian Consequences*, 3 vols. (Washington, 1989–1991). Henry F. Dobyns's *Their Number Become Thinned* (Knoxville, 1983) is a more in-depth study. A seminal, if controversial, study of the question is provided by Sherburne F. Cook and Woodrow Borah in *Essays in Population History: Mexico and the Caribbean*, 3 vols. (Berkeley, 1971–9).

[18] Geneticist Douglas C. Wallace's studies of mitochondrial DNA indicate that native Siberians lack a 'peculiar mutation' which some American Indians, Southeast Asians, and Polynesians share. See Jerry E. Bishop, 'Strands of Time: A Geneticist's Work on DNA Bears Fruit for Anthropologists', *The Wall Street Journal* (November 10, 1993), A1, A8.

[19] McNeill, *Plagues and Peoples*, pp. 31–68.

[20] Crosby, *Ecological Imperialism*, pp. 197–8; McNeill, *Plagues and Peoples*, pp. 176–8; Stannard, *American Holocaust*, X, pp. 53–4; Alfred W. Crosby, 'Virgin Soil Epidemics as a Factor in the Aboriginal Depopulation in America', *William and Mary Quarterly* (3rd Series) XXX, no. 2 (1976), pp. 289–99; Sherburne F. Cook, 'The Incidence and Significance of Disease Among the Aztecs and Related Tribes', *Hispanic American Historical Review* XXVI, no. 3 (1946), pp. 320–35; and Jane E. Buikstra, 'Diseases of the Pre-Columbian Americas', Marvin J. Allison,

For the Tainos of the Caribbean the slaughter began in 1493 when an epidemic swept Hispaniola and radiated out from there to other islands. Swine influenza has been put forward as a candidate, in part at least because the disease attacked the Europeans as well as the Indians and, as we are still periodically reminded, few, if any, develop a steadfast immunity to its rapidly mutating pathogens. Even Columbus became ill, but he recovered and there is little question that the disease, whatever it was, treated the Indians considerably more harshly than the Europeans.[21]

Assuming that the disease among the Indians was the result of Spanish migration, another candidate for this first American epidemic is typhus, a new disease for Europeans, which had slipped into Spain from Cyprus. During the recent war in Granada, Spain's final – and successful – effort to oust the Muslims from the Iberian Peninsula, typhus had killed five times more Spanish soldiers than the enemy. Even if typhus was not the first of the Old World illnesses to reach the New World, there is plenty of evidence to indicate its Caribbean presence during the early years of exploration and conquest.[22] In addition, given the many reports of sickness among the Tainos, it seems clear that numerous other illnesses were intruding as well, and Noble David Cook indicates something of the initial onslaught of European disease in the Americas by looking at 'disease and the Depopulation of Hispaniola, 1492–1518' (Chapter 2).

In shifting our focus from the Caribbean to the mainland, smallpox, which reached the Americas by at least 1518, has been credited with spearheading the conquest of Mexico for Cortez, and that of the Andean regions for the Pizarros.[23] Some of the smallpox, however, may have been measles, which was also a proven scourge of inexperienced peoples as the suddenly not so isolated Japanese had discovered centuries earlier. And in the wake of these diseases from Europe came

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'Chagas' Disease', and Marvin J. Allison, 'Leishmaniasis', all in ed. Kenneth F. Kiple, *Cambridge World History of Human Disease*, pp. 305–17, 636–8, 832–4.

[21] Francisco Guerra, 'The Earliest American Epidemic: The Influenza of 1493', *Social Science History* XII, no. 3 (1988), pp. 305–25. Influenza also may well have been among the early killers accompanying the conquerors of Central America; see F. Webster McBryde, 'Influenza in America during the Sixteenth Century (Guatemala: 1523, 1559–62, 1576)', *Bulletin of the History of Medicine* VIII, no. 2 (1940), pp. 296–302.

[22] Hans Zinsser, *Rats, Lice, and History* (Boston, 1963), pp. 241–6, 253–7; Kenneth F. Kiple, *The Caribbean Slave: A Biological History* (Cambridge, 1984), pp. 145–6, 173; Crosby, *Columbian Exchange*, p. 42.

[23] It has been suggested, however, that a *virulent* smallpox strain may not have been endemic in Europe at this time, 'raising questions ... about the provenance of the lethal strain and the genetic susceptibility of its isolated [New World] target population'. Ann G. Carmichael and Arthur M. Silverstein, 'Smallpox in Europe before the Seventeenth Century: Virulent Killer or Benign Disease?', *Journal of the History of Medicine and Allied Sciences* LXII, no. 2 (1987), pp. 147–68. This may also be held to throw some doubt on the identification of the pathogens involved in early American epidemics.

countless others – among them diphtheria, chicken pox, scarlet fever, whooping cough, typhoid, mumps, pneumonia, anthrax, even bubonic plague.[24] Donald Joralemon reviews the question of 'New World Depopulation and the Case of Disease' (Chapter 3), and makes that case without assigning responsibility or blame. In his words, 'The tragedy appears to be a necessary outcome of human interaction across biological boundaries'.[25]

Alfred Crosby would doubtless agree. His seminal essay, 'Conquistador y Pestilencia: The First New World Pandemic and the Fall of the Great Indian Empires' (Chapter 4), was crucial in educating historians about the vital role that smallpox played in the Spanish conquest of Mexico and Peru. In this essay, and in his subsequent book on *The Columbian Exchange: Biological and Cultural Consequences of 1492*, Crosby, perhaps wisely, refrained from attempting to measure quantitatively the demographic catastrophe such diseases wreaked on New World Indians, and one of the aims of Henry F. Dobyns, in a book entitled *Their Number Become Thinned*, was to determine the numerical magnitude of that tragedy in a portion of the Americas. This effort has established Dobyns as one of the 'high counters' in the words of David Henige.[26]

In this book, Dobyns was building on much of his earlier work, beginning with his controversial but ground-breaking essay on 'An Outline of Andean Epidemic History to 1720' (Chapter 5). As the reader will soon note, the article is not bound by its title, but ranges over much of Spain's American empire in assigning dates to the major epidemic events.

Unhappily, the pathogens that prevailed in Europe were not the only new microbes invading the Americas. If the diseases brought by the Iberians made conquest easy, they complicated colonization by destroying so many of those that had been counted upon to do the labour. But Spaniards in Hispaniola noticed that the few blacks among them were far hardier and much more disease-resistant than the natives. These first blacks were *ladinos*, born in Iberia, and thus with

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[24] Crosby, *Columbian Exchange*, p. 42ff.; McNeill, *Plagues and Peoples*, pp. 1–2; Dobyns, *Their Number Become Thinned*, p. 11; W. Wayne Farris, 'Diseases of the Premodern Period in Japan', Ann Ramenofsky, 'Diseases of the Americas, 1492–1700', and Kenneth F. Kiple, 'Disease Ecologies of the Caribbean', all in ed. Kenneth F. Kiple, *Cambridge World History Of Human Disease*, pp. 376–85, 317–28, 497–504. In addition, it seems probable that some of these epidemics swept across parts of North America ahead of the Spanish advance. See, for example, Daniel T. Reff, 'Old World Diseases and the Dynamics of Indian and Jesuit Relations in Northwestern New Spain, 1520–1660', in ed. N. Ross Crumrine and Phil C. Weigand *Ejidos and Regions of Refuge in Northwestern Mexico* (Tucson, 1987), pp. 85–94; and John C. Ewers, 'The Influence of Epidemics on the Indian Populations and Cultures of Texas', *Plains Anthropologist* XVIII (1973), pp. 104–115.

[25] Joraleman, p. 90 of this volume.

[26] Crosby, *Columbian Exchange*, p. 39; Dobyns, *Their Number Become Thinned*, p. 4 and *passim*; Henige, 'Native American Population at Contact', *passim*.

the same disease experience (and immunities) as their masters. But they were too few, and a slave trade directly from Africa was underway by 1518. Fortunately for their masters, the arriving slaves left a disease environment that included most of the illnesses that Europeans routinely faced and acquired immunities against. Unfortunately, however, they brought with them a few other maladies to which only they were resistant[27] – a matter that Philip Curtin discusses in his now classic look at 'Epidemiology and the Slave Trade' (Chapter 6).

Two of these diseases, *falciparum* malaria and yellow fever – which rank among the greatest tropical killers in human history – wrote an extraordinarily virulent chapter in the history of the Americas. The protozoa of *falciparum* malaria (the most lethal of all the malaria types) doubtless arrived in the blood of the first Africans to step ashore in the New World, and anopheline mosquitoes were on hand to greet the new arrivals and begin to spread the pathogens about. The virus of yellow fever, however, would have encountered far more difficulty in remaining alive during a transatlantic crossing, and most probably the favorite vector of yellow fever, the *Aedes aegypti* mosquito, was not native to the Americas and also had to be imported from Africa. Thus yellow fever's official debut in the Americas was delayed until 1647, although many suspect that it made some earlier appearances.[28]

These African diseases joined with those from Europe in further eradicating the Indians in low-lying areas to the point where, save for a handful, they completely disappeared from the Caribbean. A stark illustration of the virulence of these mosquito-borne plagues begins in the highlands of Mexico and the Andes where the mosquito vectors that transmitted them seldom reached. There, native populations, having only to face the pathogens carried by the Europeans, eventually developed immunities to defend against those germs, recovered from a downward demographic spiral and began to grow once again. The illustration is completed in those areas where populations faced waves of both European and African illnesses. In these instances there were no populations left alive to recover.[29]

Although much remains to be elucidated, the nature of black resistance to *falciparum* malaria and yellow fever is at least partially understood by modern medicine in immunological terms. In earlier times, however, it was thought of as 'racial' resistance, and as both diseases slaughtered whites but not blacks on

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[27] Kiple, *Caribbean Slave*, pp. 3–4, 8–13.

[28] Kenneth F. Kiple and Virginia H. King, *Another Dimension to the Black Diaspora: Diet, Disease, and Racism* (Cambridge, 1981), pp. 23, 31–7, 48–51; Kiple, *Caribbean Slave*, pp. 19–20, 161–8.

[29] Cook and Borah, *Essays in Population History*, Vol. 1, *passim*; Dobyns, *Their Number Become Thinned*, *passim*; John Hemming, *Red Gold: The Conquest of the Brazilian Indians* (Cambridge, Mass., 1978), p. 4; Kiple, *Caribbean Slave*, pp. 4, 9–11.

plantations and in battle alike, the African ironically became ever more valuable in the eyes of the European.[30] Francisco Guerra treats this and other themes in his examination of 'The Influence of Disease on Race, Logistics and Colonization in the Antilles' (Chapter 7).

It was a bitter irony that although they had the blacks to do the hard work for them in hot places, whites nonetheless had to be present to ensure that the work got done. And such a presence left them exposed to fevers that were lethal for whites but not for blacks. When yellow fever struck Barbados in 1647–8 and then swept the Caribbean, thousands of whites – perhaps 6,000 in Barbados alone – went to yellow fever graves dug by slaves that the disease had seemingly left untouched. For whites to move inland, however, away from the yellow fever of coastal cities meant courting the 'ague' – frequently *falciparum* malaria – and the kind of nasty death that it could provoke.[31] No wonder that there developed a 'Fear of Hot Climates in the Anglo-American Colonial Experience' (Chapter 8), which is discussed by Karen Ordahl Kupperman.

As this title indicates, such a fear was not limited to the West Indies. Rather, the range of the African diseases was such that yellow fever struck New York in 1668, Philadelphia and Charleston in 1690, and Boston in 1691. In the eighteenth century the disease became a regular summer visitor to cities of the eastern seaboard, and in the nineteenth century a scourge of the Gulf coast as well.[32]

Meanwhile, in less dramatic fashion, *falciparum* malaria had also become a part of the North American disease environment. The milder *vivax* malaria, which had migrated in the blood of the colonists from Europe, was an old curse for Englishmen. But toward the end of the seventeenth century a new kind of malaria had set upon settlers in South Carolina that was so 'extraordinarie sicklie that sickness quickly seased many of our numbers'. Such a sudden 'seasing' of lives, coinciding with the arrival of colonists from the West Indies on the one hand and the beginning of the African slave trade to North America on the other hand, seems like a trumpet-call announcing the arrival of *falciparum* malaria.[33] These same circumstances probably account for the establishment of the disease in the

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[30] Kenneth F. Kiple and Brian Higgins, 'Yellow Fever and the Africanization of the Caribbean', in eds. John W. Verano and Douglas H. Ubelaker, *Disease and Demography in the Americas* (Washington, 1992), *passim*; Kiple and King, *Another Dimension*, pp. 29–68.

[31] Kiple, *Caribbean Slave*, pp. 20, 163–5.

[32] K. David Patterson, 'Yellow Fever Epidemics and Mortality in the United States, 1693–1905', *Social Science and Medicine* XXXIV, no. 8 (1992), pp. 855–65; Donald B. Cooper and Kenneth F. Kiple, 'Yellow Fever', in ed. Kenneth F. Kiple, *Cambridge World History of Human Disease*, pp. 1100–1107.

[33] Wyndham Bolling Blanton, *Medicine in Virginia in the Eighteenth Century* (Richmond, 1931), pp. 54–5; John Duffy, *Epidemics in Colonial America* (Baton Rouge, 1953), p. 204; Peter H. Wood, *Black Majority: Negroes in Colonial South Carolina from 1670 through the Stono Rebellion* (New York, 1974), Chap. 3; Kiple and King, *Another Dimension*, p. 51.

Chesapeake Bay region at about the same time. Darrett B. Rutman and Anita H. Rutman probe the circumstances 'Of Agues and Fevers: Malaria in the Early Chesapeake' (Chapter 9), and compare the society, economy, and culture that developed there with that of nearby New England where the disaster was relatively rare.

But if African pathogens had moved north to loose themselves among the Europeans, so too had the diseases that, having previously conquered the Aztecs and the Incas, were now racing pell-mell through the bodies of the native North Americans. '[T]hey dye like rotten sheep ...' declared William Bradford, with no little satisfaction, whereas '... not one of the English was so much as sicke, or in the least measure tainted with this disease'.[34] This quotation was taken from John Duffy's survey of 'Smallpox and the Indians in the American Colonies' (Chapter 10), which, in tandem with Sherburne F. Cook's examination of 'The Significance of Disease in the Extinction of the New England Indians' (Chapter 11), speak volumes about the demise of the major Indian groups of North America beginning about a century after Cortez first set foot in Mexico.[35]

As in the Americas, European contact in the Pacific was spearheaded by the Spaniards. It began with Magellan's voyage, was extended by the Spanish trade route from Mexico to the Philippines, and expanded as Spanish explorers and missionaries fanned out among the islands. In the seventeenth century they were joined by Dutch traders, and in the eighteenth century by British explorers.[36] And as in the Americas, the end of isolation from the Eurasian civilizations, and thus from Eurasian pathogens, meant death and depopulation.

Yet because island communities were typically small, diseases were normally confined to an island or two with the result that, initially at least, there were no widespread epidemics of the spectacular sort that the New World had experienced.

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[34] Duffy, p. 237 of this volume.

[35] For further discussion of early North American disease exchanges, see Crosby, *Ecological Imperialism*, pp. 200–3; Reff, 'Old World Diseases and Dynamics of Indian and Jesuit Relations', *passim*; Ewers, 'Influence of Epidemics on Indian Populations', *passim*; Phillip L. Walker, Patricia Lambert, and Michael J. DeNiro, 'The Effect of European Contact on the Health of Alta California Indians', in Thomas, *Columbian Consequences*, Vol. 1, p. 64; Clark Spencer Larsen et al., 'Beyond Demographic Collapse: Biological Adaptation and Change in Native Populations of La Florida', in Thomas, *Columbian Consequences*, Vol. 2, pp. 409–28; Timothy K. Perttula, 'European Contact and Its Effects on Aboriginal Caddoan Populations between AD 1520 and AD 1680', in Thomas, *Columbian Consequences*, Vol. 3, pp. 501–518; Dean R. Snow and Kim M. Lanphear, 'European Contact and Indian Depopulation in the Northeast: The Timing of the First Epidemics', *Ethnohistory* XXXV, no. 1 (1988), pp. 15–33; and Robert Fortuine, 'The Health of the Eskimos, as Portrayed in the Earliest Written Accounts', *Bulletin of the History of Medicine* :LXV, no. 2 (1971), pp. 97–114.

[36] J.H. Parry, *The Age of Reconnaissance* (Berkeley, 1963), pp. 159–161, 194–201; Crosby, *Ecological Imperialism*, pp. 122–30. See also J.C. Beaglehole, *The Exploration of the Pacific* (London, 1934).

Moreover, because populations were too small to maintain them, the new diseases had to be continually reintroduced. Thus although influenza probably pummeled the islanders from the beginning of contact with the Europeans, the first report we have of the disease is from Tahiti in 1772. Similarly, syphilis (and gonorrhea) may have reached the Pacific with early explorers and traders, but reports of it only came with the voyages of James Cook in the late 1760s and again in the late 1770s. In the case of smallpox, its first recorded epidemic took place close to a century earlier (1688) in Guam, which was right on the route of the Manila galleons sailing from Mexico.[37] But after this we hear little about the disease for another century until it exploded among the aborigines of Australia and began the grim work of nearly halving their numbers as is shown in Judy Campbell's discussion of 'Smallpox in Aboriginal Australia, 1829–1831' (Chapter 12).[38]

And finally in the Pacific, Hawaii, which can probably serve in microcosmic fashion as a model of the demographic tragedy which befell all of that vast region of the world, is the focus of David E. Stannard's examination of 'Disease and Infertility: A New Look at the Demographic Collapse of Native Populations in the Wake of Western Contact' (Chapter 13).[39]

We close this volume by noting that the ecological disruption of much of the globe occasioned by the expansion of Europe went far beyond uniting New World peoples with Old World pathogens. As Alfred Crosby has so brilliantly portrayed the process in his study of *Ecological Imperialism*, Old World plants, animals, and insects were as aggressive as micro-organisms in colonizing new areas, and just as spectacularly successful in working their own ecological mischief.[40]

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[37] Leslie B. Marshall, 'Disease Ecologies of Australia and Oceania', in ed. Kenneth F. Kiple *Cambridge World History of Human Disease*, pp. 482–496.

[38] As Campbell suggests, however, it seems likely that at various times smallpox was introduced into Australia by Asians as well as by Europeans. For more on smallpox in Australia, see Campbell's later article, 'Smallpox in Aboriginal Australia, The Early 1830s', *Historical Studies* XXI, no. 84 (1985), pp. 336–358; N.G. Butlin, 'Macassans and Aboriginal Smallpox: The "1789" and "1829" Epidemics', *Historical Studies* XXI, no. 84 (1985), pp. 315–335; and Crosby's discussions of the initial Australian smallpox epidemic in *Ecological Imperialism*, pp. 205–207, 309–311.

[39] For more on Oceania's (and especially Hawaii's) experience with European diseases, see David E. Stannard, *Before the Horror: The Population of Hawai'i on the Eve of Western Contact* (Honolulu, 1989); Andrew F. Bushnell, '"The Horror" Reconsidered: An Evaluation of the Historical Evidence for Population Decline in Hawai'i, 1778–1803', *Pacific Studies* XVI, no. 3 (1993), pp. 115–161; James Watt, 'Medical Aspects and Consequences of Cook's Voyages', in ed. Robin Fisher and Hugh Johnston *Captain James Cook and His Times* (Seattle, 1979), pp. 129–157; William Shainline Middleton, 'Early Medical Experiences in Hawaii', *Bulletin of the History of Medicine* XLV, no. 5 (1971), pp. 444–460; and Robert C. Schmitt, 'The Okuu - Hawaii's Greatest Epidemic', *Hawaii Medical Journal* XXIX, no. 5 (1970), pp. 359–364.

[40] Crosby, *Ecological Imperialism*, pp. 145–194 and *passim*. On this point and what follows, see also Crosby's *Germs, Seeds, and Animals: Studies in Ecological History* (Armonk, N.Y., 1994).

E.L. Jones provides us with one example in his look at 'Creative Disruptions in American Agriculture, 1620–1820' (Chapter 14), with the disruptions in question brought on by the effort to transfer European agriculture to North America. Deforestation opened the way for Old World weeds, and fields and livestock attracted an astounding variety of pests and predators, all of which set about rearranging the continent and – although Jones does not mention it – likely making life quite literally as difficult for the Native Americans as the Eurasian pathogens that were ricocheting about.[41]

Yet if European plants did harm to the Americas, American plants proved marvelously useful for the Europeans. Maize and manioc sent to Africa triggered a population explosion that fueled the flow of slaves back to the homeland of the plants. And in Europe maize fed to livestock meant that more could be overwintered with more available protein the result, whereas the potato entered the human stomach directly.[42] Indeed, as William L. Langer points out, the potato may have been the root at the heart of 'Europe's Initial Population Explosion' (Chapter 15).

Clearly, in the exchange of plants and pathogens that characterized the expansion of Europe, the Europeans, at least in the relatively short run, were the only winners.

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[42] Crosby, *Germs, Seeds, and Animals*, pp. 92, 149–156, 172–173; Kiple, *Caribbean Slave*, p. 25; Kiple and King, *Another Dimension*, pp. 8–9.

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This volume is dedicated to
Tascha, Tina, and Coneè

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The Origin and Antiquity of Syphilis: Paleopathological Diagnosis and Interpretation

Brenda J. Baker and George J. Armelagos

Despite Thomas Gann's 1901 publication of "Recent Discoveries in Central America Proving the Pre-Columbian Existence of Syphilis in the New World," the controversy concerning the origin and antiquity of syphilis remains. As the Columbian quincentenary draws near, it is appropriate to reassess the documentary and skeletal evidence regarding the origin of syphilis and its dispersion throughout the world in the light of paleopathological diagnosis and interpretation. A review of the literature strongly suggests a New World origin of the treponemal infections. Whereas the evidence for pre-Columbian treponematosis in the Old World is documentary and equivocal, there is a vast array of skeletal evidence indicating the presence of a nonvenereal form of treponemal infection in the Americas prior to Columbus's arrival.

Hypotheses on the Origin of Syphilis

Three hypotheses have been advanced to explain the origin and subsequent spread of venereal syphilis throughout the world.

The Columbian hypothesis, proposed by Crosby (1969), Dennie (1962), Goff (1967), Harrison (1959), and others, is that syphilis originated in the Americas and was carried to Europe by Columbus's crew in 1493. Subsequently, a syphilis epidemic occurred in Europe about 1500. The rapid spread of syphilis throughout Europe at that time suggests the introduction of a virulent disease into a population that had not previously been exposed to it and had no immunity to it.

Proponents of the diametrically opposed pre-Columbian hypothesis (e.g., Hackett 1963, 1967; Holcomb 1934, 1935) assert that venereal syphilis was present in Europe prior to Columbus's voyage but was not distinguished from "leprosy." The alleged epidemic resulted from the recognition of syphilis as a separate disease in the 1490s. Cockburn (1961; 1963:153–59) provides an evolutionary framework for the pre-Columbian origin of syphilis in which geographical isolation led to speciation of *Treponema*. Throughout most of human history, treponemal infection (i.e., pinta, yaws, endemic syphilis, and venereal syphilis) was mild and chronic because populations were small. As population size increased, more acute infections were selected for and spread by direct skin-to-skin contact among children. By 1492, European living standards had improved to the point of differentially affecting the transmission of *Treponema* species. Those dependent upon skin contact were disadvantaged and replaced by a hardier strain that was sexually transmitted (Cockburn 1961:226). Thus in Cockburn's view the discovery of America and the appearance of venereal syphilis are not cause and effect; rather, both resulted from other social and economic events.

A third, unitarian hypothesis is that the agent of

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syphilis has evolved with human populations and was present in both the Old and the New World at the time of Columbus's discovery. Hudson (1963*a*, *b*, 1965*a*, *b*, 1968) maintains that pinta, yaws, endemic (nonvenereal) syphilis, and venereal syphilis are four syndromes of treponematosis, a single disease caused by *Treponema pallidum*, which evolved simultaneously with humans. The syndromes form a biological gradient in which various social and environmental factors produce different manifestations of treponematosis (Hudson 1965*a*). Although Hudson and Cockburn agree on the role of improved hygiene in the appearance of venereal syphilis, they disagree on several aspects of its etiology and epidemiology.

According to Hudson (1963*a*, 1965*a*), treponematosis originated during the Paleolithic period as a childhood disease (yaws) transmitted by skin-to-skin contact in the hot, humid climate of sub-Saharan Africa. The infection accompanied gatherer-hunters in their migrations throughout the world. As groups moved into drier zones bordering the tropics, the focus of treponemal activity retreated to the moist areas of the body (mouth, armpits, and crotch), as in endemic syphilis (Hudson 1965*a*:891). Treponematosis in the form of endemic syphilis was carried into the New World by the earliest migrants from the Old World. As the tropical zones of the Americas were populated, the climatic change caused the shift back to yaws (p. 893). The appearance of villages in the Neolithic period did not alter the nonvenereal nature of the infection; crowded, unsanitary conditions and increased frequency of child-to-child contact in village settings facilitated its spread (1963*a*:1042–43; 1965*a*:892–93).

Urbanization, beginning in Mesopotamia and Egypt by 4000 B.C., was accompanied by an improvement in personal and community hygiene (Hudson 1963*a*:1043). Although it seems counterintuitive for sanitation to have improved in cities, Hudson (1965*a*:895) points out that "hygienic barriers do not have to be very high to prevent the spread of touch-contact syphilis." Availability of water, washing and bathing with soap, separate sleeping quarters, and the like became adequate barriers to the proliferation of treponematosis by casual contact among children. As a result, individuals reached sexual maturity without prior exposure to it. Hence, "coitus . . . became the only personal contact of sufficient intimacy to permit transmission of treponemas," and adults disseminated the disease in a society in which there was "promiscuity and prostitution" (p. 895).

Hudson (1965*b*:738) indicates that both venereal and nonvenereal forms of treponematosis may be present within a narrow geographical area, for example, where a city characterized by venereal syphilis is surrounded by a rural area characterized by yaws. Despite identical climates, the higher hygienic level and different social customs in the city promote venereal transmission. Dissolution of urban life would result in a shift from venereal to nonvenereal forms of treponematosis (either endemic syphilis or yaws, depending upon the climate).

Syphilis and Leprosy

Because the pre-Columbian and unitarian hypotheses suggest that diseases such as yaws, endemic syphilis, venereal syphilis, and leprosy were confused from ancient times and grouped under the term "leprosy" (Holcomb 1935:277; 1940:177; Hudson 1965*a*:896), before examining the evidence it is necessary to discuss the differential diagnosis of these diseases.

Venereal syphilis has an incubation period of 10–90 days before the primary lesion appears in the anogenital region (Olansky 1981:299). Secondary lesions usually develop on the skin and mucous membranes. Prior to the advent of penicillin treatment, the prevalence of syphilis was about 5% in mostly urban adult populations (Steinbock 1976:110). Steinbock's survey of the clinical literature predating penicillin use indicates skeletal involvement in 10–20% of cases (cf. Hackett 1976:108, who cites a single study in which osseous lesions developed in only 1% of untreated patients). Since asymptomatic bone lesions often go undetected in early syphilis, skeletal involvement may be underestimated (Hansen et al. 1984; Steinbock 1976:109). Following Steinbock's (p. 110) arithmetic, however, one obtains a frequency of osseous involvement in 1 of every 100–200 individuals in a skeletal series representing an adult urban population (a prevalence of 0.5–1% in skeletal populations). Hackett (1976:108, 114) indicates that only 1 in 1,000 adults would develop syphilitic bone lesions.

Skeletal involvement in venereal syphilis most often affects the cranial vault, the nasal area, and the tibia. Together, these three locations comprise 70% of all tertiary syphilitic bone lesions (Ortner and Putschar 1985:182). The major diagnostic criterion of skeletal syphilis is the caries sicca sequence, described in detail by Hackett (1976:30–49), which results in the "worm-eaten" appearance of the outer table of the cranial vault, characterized by the formation of stellate scars. Caries sicca is usually accompanied by naso-palatine destruction. This destruction, more extensive and rapid than in leprosy, usually involves the nasal bones and is accompanied by healing and sclerosis (Hackett 1976:63–65; Ortner and Putschar 1985:192, 197; Steinbock 1976:145, 208). Where there is gross destruction of the naso-palatine region, there is often maxillary alveolar damage as well (Hackett 1976:65).

Postcranially, formation of subperiosteal bone begins in the metaphyses of the long bones, with the tibiae being most often involved. Inflammation of the entire periosteum initiates a subperiosteal response resulting in thickening and possible bone deformation (Steinbock 1976:115). Hackett (1976:79–90) proposes a sequence for nongummatous periostitis that ranges from finely striated nodes and expansions to grossly rugose expansions, which he tentatively considers diagnostic criteria of syphilis. Gummatous lesions—nodes/expansion with superficial cavitation—he regards as certainly diagnostic (pp. 93–97). Gumma formation may occur periosteally or in the medullary cavity, resulting in both proliferative

and degenerative changes. Syphilis lacks the smooth cloacae and the sequestrum and involucrum formation of pyogenic osteomyelitis (Hackett 1976:95; Steinbock 1976:137). Generally, the affected bone appears roughened and irregular because of thickening and increased density. The medullary cavity, particularly in the tibia, is greatly narrowed by cortical thickening (Steinbock 1976:117, 123). Hands and feet are rarely affected.

Pinta, yaws, endemic (nonvenereal) syphilis, and venereal syphilis have been thought to be caused by different species of *Treponema* (respectively, *T. carateum*, *T. pertenue*, and two subspecies of *T. pallidum*). The causative organisms of each disease, however, cannot be distinguished from each other by any known test. In electron microscope studies, the "species" of *Treponema* are morphologically identical (Hovind-Hougen 1983:5). Their antigenic structures differ only quantitatively (Hudson 1965*a*:886). DNA sequence homology analysis indicates that *T. pertenue* and the subspecies of *T. pallidum* are identical and "might be regarded as a single species" (Fieldsteel 1983:50). Partial cross-immunity exists between the treponemal syndromes (Cannefax, Norins, and Gillespie 1967:473–74). Clinically, yaws and endemic and venereal syphilis closely resemble each other in the prolonged course of the disease, with early and late manifestations. Primary yaws is similar to primary syphilis; secondary yaws resembles secondary syphilis, although the skin lesions of the former are often larger and more exuberant; and lesions of tertiary yaws, characterized by gummatous lesions of the skin, soft tissue, bones, and naso-palatine area, are indistinguishable from those of tertiary syphilis (Musher and Knox 1983:114–15). Where (as in all forms except pinta) bone lesions result from the treponemal syndromes, they are also indistinguishable from each other (Hackett 1976:113). Except for the dental stigmata and osteochondritis found only in congenital syphilis, the bone lesions found in one disease are identical to those found in the others (Steinbock 1976:139, 143). Steinbock stresses that the differences in skeletal involvement are merely quantitative. For example, in endemic syphilis and yaws, the cranial vault is infrequently affected in comparison with venereal syphilis, whereas tibial lesions are much more common.

Skeletal series in areas in which either endemic syphilis or yaws occurs are expected to reveal bone lesions in approximately 1–5% of the entire series (Steinbock 1976:139, 143).

Leprosy (now known as Hansen's disease) is a chronic infectious disease caused by the bacillus *Mycobacterium leprae*. The incubation period averages at least three to five years (World Health Organization 1980:16). A prevalence of about 0.5% (4.6 per 1,000) is found in modern Africa, where leprosy is endemic (p. 10). In clinical studies, skeletal manifestations occur in 15–68% of leprosarium patients (Chamberlain, Wayson, and Garland 1931, Esguerra-Gómez and Acosta 1948, Faget and Mayoral 1944, Murdock and Hutter 1932, Paterson 1961). Although leprosy is best known as a skin disease, its effects on the nervous and skeletal systems are well known. Skin changes usually consist of rough, dry macules, in which hypopigmentation may occur (Drutz 1981, World Health Organization 1980).

Skeletal manifestations of leprosy have been described in detail by Møller-Christensen (1967), Møller-Christensen and Faber (1952), Møller-Christensen and Inkster (1965), and Paterson (1959). The most reliable diagnostic criterion of leprosy is the occurrence of facies leprosa in the skull. This condition is characterized by atrophy of the anterior nasal spine, atrophy of the maxillary alveolar margin, mainly in the incisor region, and inflammatory changes of the superior surface of the hard palate. Facies leprosa has been identified in 60–82% of modern leprosy patients (Steinbock 1976:201). Postcranial changes accompanying facies leprosa include atrophy and resorption of the phalanges in the hands, beginning distally, and at the metatarsophalangeal joints in the feet. At a medieval Danish leper cemetery (St. George's Hospital, Naestved), 71.3% of 185 adequately preserved skeletons exhibited both facies leprosa and changes in the hands and feet (Weiss and Møller-Christensen 1971:262–63). Changes affecting only the hands and feet occurred in 26.5% and changes in the skull alone in 2.2% (Weiss and Møller-Christensen 1971:262–63). Examination of the hands and feet is therefore important in differentiating other diseases, such as syphilis, from leprosy. Subperiosteal bone deposits occur occasionally in the tibia and fibula in leprosy, but other long bones remain uninvolved. In contrast to the situation in syphilis, where extensive bone destruction is always accompanied by reactive new bone formation (Møller-Christensen 1952:106–7), bone resorption is not accompanied by proliferation.

Documentary Evidence

In the absence of extensive skeletal evidence for syphilis, medical historians have turned to ancient and medieval documents in an effort to establish the antiquity of syphilis in the Old World (Baker 1985). Supporters of the pre-Columbian and unitarian hypotheses argue that syphilis was confused with leprosy in the ancient literature and have sought passages purported to delineate the venereal communication of the disease. Columbianists discount such descriptions and point to accounts of a new disease of foreign origin at the close of the 15th century.

BIBLICAL REFERENCES TO "LEPROSY"

The Old Testament (written between the 8th and 2d centuries B.C.) is the most frequently cited text in reference to leprosy. The Hebrew word *tsara'at*, which is translated into Greek as *lepra*, "scaly," denotes ritual uncleanliness and probably refers to a wide range of dis-

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eases with dermatological manifestations (Cochrane 1959:viii; Hulse 1975; Møller-Christensen 1967:304–5). Lepromatous leprosy, originally described by the Alexandrian medical school about 300 B.C., is referred to as elephantiasis because of the thickening and corrugation of the skin (Dols 1979:315). As a result of inexact translation, the biblical term "leprosy" could, therefore, refer to syphilis.

Biblical passages suggesting syphilis have been reviewed at length by Willcox (1949; see also Hudson 1961:552–54 and Rosebury 1971:98–104). Moses describes punishment for disobedience as manifesting "emerods," scabs, itches that cannot be healed, madness, and blindness (Deuteronomy 28:27–28). Job (Job 16, 19, 30) suffered from a genital lesion, and boils covered his body; iritis is suspected from his failing sight and mucous patches from his corrupt breath. David's illness (Psalms 38:1–11) is also cited as a case of pre-Columbian syphilis masquerading as "leprosy." David suffered from shooting pains and odoriferous lesions, and his "loins are filled with a loathsome disease." Like Job, he had failing vision and recovered from his illness. David believed his condition to have resulted from sleeping with Bathsheba, who was "unclean" at the time (2 Samuel 2–5).

Leviticus 13 and 22:4 and Numbers 5:2 are among passages discussing the skin lesions of "leprosy" and the restrictions placed upon the "unclean" in great detail (see Brody 1974:108–14 for further explication of biblical references to leprosy). Depigmentation and discoloration characterize the lesions reported. The lengthy description in Leviticus is actually a list used by priests to differentiate among diseases that may or may not result in ritual impurity (Hulse 1975; Sussman 1967:211).

If the foregoing passages are references to venereal syphilis, one would also expect biblical descriptions of congenital syphilis. In Jeremiah 31:29, where "the fathers have eaten sour grapes and the children's teeth are set on edge," the dental condition is suggestive of Hutchinson's teeth, a sign of congenital syphilis (Willcox 1949:32). Willcox also points to Exodus 20:5, where "the iniquity of the fathers" is visited "upon the children to the third and fourth generation." Although syphilis can be inherited only by the second generation, Brown et al. (1970:2) find this passage significant because "syphilis is one of the few known communicable diseases that can be passed from one generation to another."

Miriam's "leprosy" is described in Numbers (12:9–15). A possible macerated syphilitic fetus is suggested by Aaron's statement, "Let her not be as one dead, of whom the flesh is half consumed when he cometh out of his mother's womb." Leviticus (21:16–20) states that "he who hath a flat nose," perhaps indicative of congenital saddle nose, was ostracized. Similarly, in 2 Samuel 12 it is said that the child conceived from the adulterous and unclean union of David and Bathsheba died seven days after birth. If David's subsequent disease was syphilis, the baby may also have been afflicted.

OTHER ANCIENT REFERENCES SUGGESTIVE OF SYPHILIS

Several Greek and Roman physicians and historians including Hippocrates, Martial, Pliny, and Celsus described genital lesions following sexual activity (Brown et al. 1970:3; Hudson 1961; 1963*b*:646; Kampmeier 1984:22–23; Rosebury 1971:105–7). During the 1st century A.D., Celsus described hard and soft genital sores, reporting that the latter exuded a malodorous discharge (Hudson 1961:555). Galen (born in A.D. 131) differentiated dry ulcers from moist ulcerating tubercles, analogous to mucous patches (Kampmeier 1984:22). Such lesions were described by both Greeks and Romans as resembling mulberries or figs, which Hudson (1961) and others (see Kampmeier 1984:22) interpret as genital condylomata diagnostic of syphilis. Martial and Pliny (1st century A.D.) refer to *mentagra*, a term derived from the Latin *mentum*, "chin," from which *mentula*, "little chin," also originated. Hudson (1961:554–55; 1963*b*:646) points out that the latter term was euphemistic for the pubic area and concludes that the lesions and contagious nature of *mentagra* were venereal. Byzantine physicians of the 3d through 7th centuries documented several types of genital lesions that have been attributed to gonorrhea and syphilis (Kampmeier 1984:23). Finally, Hudson (1961:551), in his thorough etymological treatise, links the term *bubas* to Greek and Latin terms denoting "serpent." Diaz de Isla employed this term to describe syphilis in 1539, noting that it was previously used in Spain to describe "leprosy" and the Romans' *mentagra*.

The ancient literatures of India and China have also been cited as containing "unmistakable proofs" that genital lesions were associated with sexual activity (Hyde 1891:117). Kampmeier (1984:22) indicates that the Sanskrit Veda contains several references to genital disease, which some have interpreted as syphilis. Lu and Needham (1967) mention no disease resembling syphilis in ancient China, and Crosby (1969:219) quotes Wong and Wu (1936:218) as saying that no Chinese writer "has ever described syphilis as being mentioned in ancient literature." Wong and Wu, however, assert in the following sentence that these writers "did not know the connection between chancres and syphilides, for the former were mentioned as early as the 7th century A.D." As to whether these chancres are syphilitic, they indicate (p. 219) that "the original texts are too brief to enable us to form any definite conclusion." Clinical descriptions of leprosy in China and India from as early as 600 B.C. are, in contrast, quite clear (Browne 1970:641; Lu and Needham 1967:226, 236–37; Steinbock 1976:192).

MEDIEVAL "LEPROSY"

Medieval texts have also been studied for evidence that syphilis was included with other diseases under the term "leprosy." True leprosy was apparently unknown in the Mediterranean region prior to 300 B.C. Andersen

(1969:123) has proposed an introduction from India after Alexander the Great's campaign in 327–326 B.C. Celsus, Pliny the Elder, Galen, and Aretaeus were the first to describe the disease, which they called elephantiasis, in the first two centuries A.D. (Dols 1979:315; see also Patrick 1967:245). The generic term *lepra* was not applied to true leprosy until the 8th century A.D. (Steinbock 1976:192–93) in the translation of Arab medical texts into Latin (Richards 1977:9). Thus, a previously distinct and well-defined disease of no religious significance was blended with the biblical concept of impurity and acquired the stigma still attached to the word "leper" (Richards 1977:9–10). As a result, the medieval diagnosis of leprosy may have incorporated several afflictions, including true leprosy and syphilis.

The mode of transmission of medieval leprosy is confused. Bartholomeus Anglicus (ca. 1230–50) wrote that leprosy was caused by "intercourse with a woman after she had been with a leprous man, heredity, and feeding a child with the milk of a leprous nurse" (Rubin 1974:153; see also Gordon 1959:493–94). Theodoric of Cervia (1205–98) provides one of the more detailed descriptions of the disease within the prevailing humoral theory (7 of his 12 common signs of leprosy correspond to those found by modern diagnosticians), but he also insists that those "lying with a woman with whom a leper has lain" will be infected (Brody 1974:34–41). Numerous medieval scholars refer to "venereal leprosy," "hereditary leprosy," and "leper whore" and describe genital lesions (Brody 1974:54–56: Holcomb 1935:297–303; Hudson 1961:548; 1972:150–51; Kampmeier 1984:23–24). Leprosy is neither hereditary nor sexually transmitted. It does show a strong family incidence (4.4–12% of household contacts of lepromatous leprosy patients show signs of the disease within five years [World Health Organization 1980]), but both husband and wife are affected in less than 5% of couples (Richards 1977:xvi).

Leper hospitals were established throughout Europe prior to the Crusades (A.D. 1096–1221) in an effort to separate lepers from society. "Leprosy" reached its peak prevalence in Europe in the 11th through 13th centuries (Rubin 1974:151), coinciding with the Crusades. Hudson (1963*b*) has outlined the importance of concurrent pilgrimage to the Middle East in disseminating disease, which he contends included treponemal infection disguised as "leprosy." As supporting evidence he cites the use of "Saracen ointment," which contained mercury, by the returning lepers (1961:548; 1963*b*:648; see also Hackett 1967:163–64). Mercury has no effect on true leprosy but was the mainstay in treating syphilis until the early 20th century (Steinbock 1976:88).

Perhaps the most explicit description of medieval "leprosy" is found in Robert Henryson's poem "The Testament of Cresseid." Written in Scotland prior to 1492, the poem has been variously claimed as a delineation of venereal syphilis (Hudson 1972) and as a sensitive portrayal of an individual afflicted with leprosy (Richards 1977:6–8). The poem, a contemporary version of the myth of the Trojan lovers, Troilus and Cressida, depicts a fallen woman who acquired an "incurable disease" and died "a leper" (Hudson 1972:146). Cresseid's face became "o'erspread with black boils," her "clear voice" became "hoarse," "rough and raucous" (quoted in Richards 1977:6–7). Cresseid's condition resulted from her lustful life. She was confined to a leper house to prevent the spread of her infection to others. Her life as a leper is detailed, including the last will and testament required for entrance into the leper hospital, her wandering with cup and clapper, and her diet of "mouldy bread, perry, and cider sour" (quoted in Richards 1977:6–8). Henryson called Cresseid's disease leprosy, but Hudson (1972:149) suggests that because it is associated with immorality and sex it is venereal syphilis. Richards (1977:6) finds Henryson so compassionate in his portrayal of the "leper" that he must have had firsthand knowledge of leprosy and "of lives broken by it."

THE EPIDEMIC OF 1500

By 1500, a "new" disease, which we know as syphilis, was being described in Europe (see Crosby 1969, Dennie 1962, Holcomb 1934, Williams, Rice, and Renato Lacayo 1927). As syphilis became widely recognized and described, "leprosy" became less common. Historical events unrelated to the return of Columbus may explain this trend. For example, the invention of the printing press in the mid-15th century led to rapid diffusion of information. By 1566, 58 books had been published on the subject of syphilis. Kampmeier (1984:24) argues that the proliferation of such publications led to the widespread recognition of the disease at this time, making it appear as if it were a new disease of epidemic proportions. This dissemination of knowledge was accompanied by historical events that caused the displacement of people throughout Europe.

Papal proclamations in 1490 and 1505 abolished all leper houses (Holcomb 1935:282), allowing the dispersal of thousands with "leprosy." Holcomb notes (p. 278) that Matthew Paris, an English monk who died in 1259, records "in somewhat ambiguous terms" the existence of 19,000 leper houses in Europe. While this figure may be exaggerated, Gordon (1959:493) indicates that "France and Germany alone had nearly 10,000 leprosaria" in 1400, and Richards (1977:11) notes approximately 200 leper hospitals "in their thirteenth- and fourteenth-century heyday" in Britain. It should be cautioned, however, that most leper hospitals were ecclesiastical foundations that accommodated only about ten lepers and at least as many chaplains and sisters (Richards 1977:11). Richards (see also Creighton 1965 [1894]:86–100) concludes that the number of hospitals is not a reliable estimate of the number of lepers because of the propensity of the church to establish the institutions to garner perpetual charity. Whatever the motive for establishing the hospitals, they did house thousands throughout Europe. If the diseases were confused, it is possible that some of the inhabitants were syphilitic and

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therefore that when the hospitals were closed syphilis was dispersed.

Hudson (1964; 1968:11) claims that treponemal infection existed in venereal and nonvenereal forms in pre-Columbian Spain and Portugal because of the Moorish occupation and the importation of slaves from sub-Saharan Africa. When an estimated 160,000 to 400,000 Jews were expelled from Spain in 1492, they allegedly carried syphilis throughout Europe (Holcomb 1935:284). The expulsion of Jews and lepers coincided with the discovery of America and the apparent epidemic of syphilis.

In late 1494, Charles VIII of France conducted a campaign against Naples. The city fell in February of 1495 as a plague broke out among the mercenary troops. They subsequently disbanded, carrying their disease throughout Europe (Brown et al. 1970:5; Williams, Rice, and Renato Lacayo 1927:683). It is generally agreed that this disease was syphilis; the controversy concerns the time of arrival of Spanish troops purported to have contracted the disease from Columbian contacts and the issue of several edicts regarding the disease elsewhere in Europe. Holcomb (1934:419; see also Hudson 1968:5–6; 1972:152) claims that Charles's army left Naples on May 20, 1495, and the Spaniards did not arrive until June. Although Charles did not reach France until October 27, 1495, an edict had been issued by the Diet of Worms more than two months earlier (August 7, 1495), indicating that syphilis was already widespread in Germany (Gordon 1959:536; Holcomb 1935:289, 427; see also Harrison 1959:4). While this would seem to vindicate Charles VIII, Waugh (1982:92) has pointed to problems in dating events of the time due to the variety of calendars in use (e.g., Gregorian vs. French).

Further confusion in the dates of early edicts on syphilis has resulted from modern errors. Holcomb (1935:293) laments the penchant of some writers (Columbianists) for accepting ideas "without first assuring themselves of the correctness of the historical data that they introduce." Reliance upon Sudhoff's archival work, however, has led to wide acceptance of the 1495 date of the aforementioned edict of the Diet of Worms. Sudhoff later amended the date to August 8, 1496, and subsequent research by Haustein revealed that the text was actually drafted by the Diet of Lindau on January 12, 1497 (cited in Temkin 1966:32–33). Thus, it is possible that the passage regarding syphilis was a response to its dissemination by soldiers returning from Italy.

Holcomb's pre-Columbian thesis hinges largely upon an edict issued in Paris that bars those with *grosse verole* (syphilis) from the city. Holcomb (1934:416, 421; 1935:293) dates this edict to March 25, 1493, or ten days after Columbus returned to Spain from his first voyage. This would render it impossible for the disease to have been imported from America. In an attempt to verify this date, Harrison (1959:4–6) followed a series of errors in later compilations of ancient French laws (one of which was cited by Holcomb) and discovered that the ordinance with the text in question was actually issued on June 25, 1498 (verified by Haustein's research, cited in Temkin 1966:33). A thorough search of the French archives revealed that the earliest Parisian reference to the disease was contained in an edict promulgated by the Paris parliament on March 6, 1497 (Harrison 1959:4–6; see also Creighton 1965:[1894]:436), nearly four years after Columbus's crew returned from the first voyage. Holcomb (1934:428) misdates this edict as well, stating that it was issued on March 16, 1496.

Several edicts ostracizing people infected with syphilis were issued elsewhere in Europe beginning in 1496. In that year, 12 such ordinances were passed at Nuremberg, and syphilitics were barred from the baths of Zurich and other municipalities throughout Switzerland and Germany (Holcomb 1934:428; Kampmeier 1984:24–25). Ten persons with "the Neapolitan disease" were expelled from Besançon, France, in April of 1496, while an edict at Lyon, dated August 12, 1497, required those with the disease to report within ten days or be apprehended (Harrison 1959:4, 6). Early reference to syphilis in Britain is from an ordinance of Aberdeen dated April 21, 1497, in which it is stated that "the infirmity came out of France" (Creighton 1965 [1894]:417). A proclamation issued in Edinburgh by James IV on September 22, 1497, requires those with "Grandgor" (syphilis) to go to the island of Inch Keith in the Firth of Forth, "there to remain until God provide for their health" (Creighton 1965 [1894]:417–18). Hospitals for the syphilitic such as St. Jobsgasthuis, founded in Utrecht in 1504 (Fuldauer, Bracht, and Perizonius 1984), were established throughout Europe by the beginning of the 16th century.

It would appear from the dates of European edicts that a new disease swept the continent within three or four years of the return of the first Columbian voyagers. The concurrence of these events has been challenged, however, by those who point out that edicts after 1493 closely resemble those previously issued to isolate lepers. For example, a Parisian edict of 1488 is directed against *les lepreux*, while those following the papal proclamation of 1490 refer to syphilis (Creighton 1965 [1894]:73; Holcomb 1934:416; 1935:282). Creighton questions the sudden reappearance of leprosy in the late 15th century, especially since the Paris edict is so close in date to those concerning syphilis. Thus, one is left to wonder whether these ordinances were issued as a consequence of the importation of syphilis or if the discovery of America was merely coincidental with the recognition and renaming of the disease as it was differentiated from "leprosy."

LATE 15TH- AND EARLY 16TH-CENTURY TREATISES ON SYPHILIS

Treatises on syphilis proliferated in the late 1490s and early 1500s (for reviews see, e.g., Crosby 1969, Dennie 1962, Holcomb 1934, Hudson 1961). Williams, Rice, and Renato Lacayo (1927) provide translations of large portions of early Spanish works, the most important of which is Ruy Diaz de Isla's "Treatise on the Serpentine

Malady, Which in Spain is Commonly Called Bubas, which was drawn up in the Hospital of All Saints in Lisbon," first printed in 1539. In it, he claims that the serpentine disease (syphilis) appeared in Barcelona in 1493, originated on the island of Española (Haiti), and was brought to Europe by Columbus's crew (p. 693). He goes on to say that in the following year Charles VIII entered Italy with "many Spaniards infected with this disease." Not knowing what it was, "the French called it the disease of Naples," and the Italians, "as they had never had acquaintance with a like disease, called it the French disease." This portion of Diaz de Isla's account is confirmed by Gonzalo Fernández Oviedo y Valdés (1478–1557): "Many times in Italy I did laugh, hearing the Italians say the French disease and the French calling it the disease of Naples; and in truth both would have hit on the right name if they had called it the disease from the Indies." Oviedo also verifies that among Charles VIII's army were Spaniards "touched with this disease," but he indicates that they did not join the French until 1496 (see Williams et al. 1927:687–89 and Crosby 1969:222). Much has been made of this discrepancy in dates (e.g., Holcomb [1935:292] uses it to dismiss Oviedo's entire account); Waugh's (1982:92) caution regarding the difficulty in dating such events must be borne in mind.

Critics of the 16th-century treatises, such as Holcomb (1934, 1935), point out that no mention is made of an American origin of syphilis for more than 30 years after the discovery of the New World. Earlier texts attribute the disease to divine wrath visited upon a sinful populace, astrologic convergences, and the weather; Oviedo's work is among the first to mention an American origin. His "Summaria of the Natural History of the Indies" was published in 1526 and is purported to have been written from memory. His larger work, "General and Natural History of the Indies," was first printed in 1535 (Holcomb 1934:406–7; Williams et al. 1927:687). In it he says that, while he is writing from memory, he is referring to "notes which were written at the time when the things described in them happened." Holcomb (1934:407) points out that taking such notes is not the usual activity of a teenager (Oviedo was only 15 when Columbus returned from his first voyage). Crosby (1969:222) maintains that Oviedo was quite friendly with the explorer's sons and cites a passage in which Oviedo asked several of his friends sailing with Columbus in 1493 (second voyage) to provide him with detailed reports (his affiliation with several crew members is also recorded in the translation by Williams et al. 1927:688).

The original manuscript of Diaz de Isla's account is dedicated to King Manuel of Portugal, who died in 1521 (Williams et al. 1927:695). In a paragraph omitted from the printed versions, he writes of an island "discovered and found by the Admiral Dom Cristoual Colon at present holding intercourse and communication with the Indies" (Williams et al. 1927:695). Since Columbus's last voyage culminated in his death in the New World in 1506, it seems that the manuscript must have been written prior to that event. Furthermore, Diaz de Isla states that "in the year 1504 there were given me in writing all the remedies that the Indians used for this disease," indicating that his belief in its American origin dated to within 11 years of its alleged importation. Thus, the manuscript, usually ascribed to the period 1510–20, would appear more likely to have been written in 1505 or 1506. Holcomb (1934:412–13), however, asserts that Diaz de Isla "frequently states he had 40 years' experience in the treatment of the disease" and therefore acceptance of such dates would place his treatment well before the discovery of America. Holcomb's observation is not apparent in the translation provided by Williams et al. (1927:694), in which Diaz de Isla writes only that he has had "long experience." It seems that a decade or more would qualify as such.

Several 16th-century tracts written by European scholars in the New World document the lifeways, languages, and mythologies of various native groups and refer to a disease much like syphilis among them. These documents have been employed to support the Columbian hypothesis in publications of the past century (e.g., Brühl 1890, Crosby 1969, Williams et al. 1927), while others (e.g., Holcomb 1934:417–18) attribute such references to the introduction of the disease by Europeans. The biography of Christopher Columbus, by his son Ferdinand, includes a 1495 manuscript by Fray Roman Pane recording an Arawak myth in which the hero, Guagagiona, "saw a woman . . . from whom he had great pleasure, and immediately he sought many lotions to cleanse himself, on account of being plagued with the disease that we call French," and afterwards went to a secluded place "where he recovered from his ulcers" (Williams et al. 1927:687; see also Brühl 1890:276 and Crosby 1969:221–22). Crosby, reminding the reader that folklore is very slow to change, finds it unlikely that the Arawaks would have altered their legend to give the hero a new disease, thus implying that the malady was extant among the natives long before the Europeans arrived. This is corroborated by Bartolomé de las Casas, who questioned the natives as to the origin of the disease and was told they had had it from time immemorial (Crosby 1969:222; Williams et al. 1927:690). Further examples of "syphilis" in native mythology, as well as differential burial treatment of those afflicted, were documented by Bernardino de Sahagún, who lived in Mexico from 1529 to 1590 (Brühl 1890:275–76; Williams et al. 1927:690–91).

Linguistic evidence compiled by Montejo y Robledo from 16th- and 17th-century dictionaries of native Mexican and Central and South American languages reveals indigenous terms for *bubas* and related European expressions (Williams et al. 1927:685–86). Brühl (1890:278–80) counters the view that these terms were invented after the arrival of Europeans by reviewing the ways in which names were assigned to previously unknown things—adopting the European word with little or no change or deriving the name from a conspicuous feature of the object. While the terms for previously unknown diseases

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described a prominent symptom, the words synonymous with European appellations for syphilis were "formed at the development of the respective languages" and, in many cases, associated with chieftains and gods (Brühl 1890:279).

Skeletal Evidence

The preceding review of ancient and medieval documentary sources reveals many ambiguities in disease description and the dating of events. The ensuing interpretations of these passages remain controversial. Skeletal evidence of pre-Columbian syphilis is subject to similar disagreement. As Williams (1932:780) states, "one must have proof that a bone is ancient and that it is syphilitic. It is owing to a difference of opinion as to what constitutes proof that the controversy continues." Unfortunately, many of the remains thought to be syphilitic (primarily those recovered prior to Williams's review) lack archaeological provenience and cannot, therefore, be assumed pre-Columbian. Further difficulties arise in interpreting many late 19th- and early 20th-century descriptions of syphilitic specimens. These reports often present descriptions of an isolated skeletal element. Since skeletal lesions resulting from yaws, endemic syphilis, and venereal syphilis are identical, speculation regarding the mode of transmission of the treponeme in a single individual is impossible (i.e., an isolated case of treponematosis cannot be assumed to have resulted from venereal transmission). Reliable conclusions regarding the prehistoric distribution of treponemal disease may, however, be drawn from skeletal evidence. The pattern of treponemal infection discerned in entire skeletal series, viewed in conjunction with social and climatological factors, may permit epidemiological inferences.

OLD WORLD REMAINS

Although numerous cases of alleged pre-Columbian Old World syphilis have been described in the literature of the past century, few have withstood reexamination. Once Parrot (1879) had aroused European interest in the paleopathological identification of syphilis, nearly every French anthropologist discovered syphilitic specimens (Sigerist 1951:56). Parrot, however, confused the manifestations of congenital syphilis and rickets, delineating a "rachitic period" of congenital syphilis for which "swelling . . . of the articular ends of the bones" and "cranial osteophytes" resulting in "the form of a cross" on the skull vault were diagnostic (1879:697–98). Thus he reported syphilis in prehistoric Ecuador, Peru, and France solely on the basis of cranial vaults exhibiting circumscribed areas of bone deposition (i.e., cranial bossing) that were more likely due to rickets, iron deficiency anemia, or congenital anemia (Steinbock 1976:101). Reliance on Parrot's diagnostic criteria underlies Wright's (1971) contention that syphilis is evident in Neanderthal remains in the form of cranial bossing, thinning and pitting of the occipital and parietals, and "the relative depression of the bridge of the nose" in both children and adults. Worn taurodont molars are suggested to resemble the mulberry molars of congenital syphilis. Bowing of the femur is attributed to syphilitic osteitis, also hypothesized to "account for Neanderthal long bones being so short and stout." Many of the lesions Wright describes are diagnostic of rickets, while the general skeletal variations he attributes to syphilis are the consequence of genetic and biomechanical differences between Neanderthal and modern populations.

The alleged skeletal evidence of pre-Columbian syphilis was thoroughly reviewed by Williams in 1932. Prior to investigating archaeological specimens, he examined the bones of over 500 modern individuals known to be syphilitic in order to establish diagnostic criteria. Various specimens described in the early literature as syphilitic had apparently been lost by the time of his research, and others were too incomplete for diagnosis to be attempted. In many of the remaining cases, the supposedly syphilitic lesions could be attributed to other causes. For example, several Egyptian cases had actually suffered postmortem damage by rodents or insects (pp. 802–3), and the lesions on Parrot's (1879:698) Peruvian crania were attributable to porotic hyperostosis (Williams 1929:852; 1932:971). Williams considered five cases of reputed Old World syphilis "suspicious." In the case of a tibia and fibula from Japanese shell middens, said to be more than 2,500 years old, he thought trauma or healed osteomyelitis with periostitis the cause of the lesions described (p. 802) and judged the antiquity of the remains questionable in any event (p. 974). For a Nubian femur and tibia dated to 1000 B.C., insofar as his examination of the published illustrations permitted, he found the diagnosis of syphilis plausible, although "other causes of periostitis would be equally probable" (pp. 803, 975). For the remaining "suspicious" instances, all from France—a tibia from Solutré, a humerus and ulna from the Marne Valley, and an ulna, femur, and femur fragment from the museum at Saint-Germain (pp. 805–9, 975)—he found the diagnosis of syphilis equivocal. The few possible instances of pre-Columbian syphilis consist of isolated long bones with inadequate archaeological provenience (Sigerist 1951:56; Williams 1932:974). Jeanselme, Pales, and others concur that the Old World evidence presented prior to 1930 is inconclusive or negative (Williams 1932:975–76; see also Sigerist 1951:56 and Steinbock 1976:97).

Possible skeletal evidence accumulated since 1930 is sparse. Steinbock (1976:97) regards Siberian material consisting of several tibiae, a radius, and an ulna dated 1000–800 B.C. as the earliest indication of possible Old World syphilis. In addition, two tibiae dated 500–200 B.C. and three crania dated A.D. 100–700 are reported to show syphilitic lesions (cf. Hackett 1976:18, who indicates that the dates may be unacceptable). Evidence of pre-Columbian syphilis reported since 1930 in Europe is tantalizing but inconclusive. The skull of an adult female from Spitalfields Market in London presents the diagnostic stellate scars of caries sicca (Brothwell

1961:324–25; Morant and Hoadley 1931:222, pl. 3; Steinbock 1976:97). Historical records indicate that the site was part of the cemetery at the church of St. Mary Spittle, used A.D. 1197–1537 (Morant and Hoadley 1931:202). Brothwell (1961:324–25) finds it a "remarkable coincidence" that the woman succumbed to syphilis within 35 years of its supposed appearance in London, but the possibility cannot be dismissed. Similarly, excavations at the Helgeandsholmen cemetery in Stockholm, used from A.D. 1300 to 1531, have yielded syphilitic remains (Madrid 1986).

Hudson (1961:547–48) contends that "syphilitic skulls and other bones have been found in 'leper cemeteries' and doubtless many a European 'leper' lost his nose and his voice, or was covered with purulent crusts, as a result of treponemal infection." If he is correct, then excavations of cemeteries associated with medieval leprosaria should reveal skeletons of syphilitics in addition to lepers (his citations are to publications of 1868 and 1891, prior to the establishment of diagnostic criteria for syphilis and leprosy). Excavations at Danish leper hospitals and medieval churchyards and extensive examination of European skeletal collections reveal no evidence of pre-Columbian treponemal disease (e.g., Møller-Christensen 1952, 1967; Møller-Christensen and Faber 1952; Weiss and Møller-Christensen 1971).

Yaws and/or endemic syphilis have occasionally been reported in skeletal material from the Old World. An isolated skull from Iraq, dated prior to A.D. 500, exhibits a large crater-like depression on the mid-frontal and a smaller, slightly depressed area on the right side of the frontal bone that have been attributed to treponematosis (Guthe and Willcox 1954:fig. 2; Steinbock 1976:141). An elliptical area of porosity on the occipital of an eight-year-old child (INM 196) from the Chalcolithic site of Inamgaon in western India (dated 1000–700 B.C.) is interpreted as evidence of yaws (Lukacs and Walimbe 1984:123–24, fig. 7). This attribution is tenuous, however, since there is no other skeletal involvement and treponemal lesions are infrequent on the occipital.

Australia and the Pacific islands have yielded many examples of treponematosis in skeletal remains. Hackett (1976:109, 114) found treponemal changes in 1% of the 4,500 Australian Aboriginal crania he examined and argues that treponemal infection has probably existed in Australia "for some thousands of years." Unfortunately, no information regarding the antiquity of these remains is furnished, and it is uncertain if they predate European contact (see Steinbock 1976:141, 158). Two subadults from Tinian, in the Mariana Islands, display treponemal lesions thought to result from yaws (Stewart and Spoehr 1967[1952]). Pathological changes consist of a crater-like depression surrounded by an irregular zone of porosity on the frontal bone of one individual and similar lesions on the parietals. Parts of a femur, humerus, and radius from the same individual exhibit periostitis with cavitation. The incomplete tibia from the second subadult shows prominent thickening of the cortex along the anterior aspect (saber shin) accompanied by pitting and cavitation (Stewart and Spoehr 1967[1952]:311–17; see also Steinbock 1976:153, 158). The site is radiocarbon-dated to A.D. 854 ± 145, thus predating European contact by a considerable margin (Stewart and Spoehr 1967[1952]:311). Yaws has also been described in a pre-contact skeletal series from Tonga (Steinbock 1976:159).

NEW WORLD REMAINS

Interest in prehistoric skeletal evidence of syphilis developed in America at about the same time as in Europe. The earliest discussion is usually attributed to Jones (1876), although Williams (1932:931) cites an 1875 account, by R. J. Farquharson, of syphilitic lesions in skeletal remains from mounds near Davenport, Iowa. Jones's (1876:49, 65–67, 71–72, 85) detailed descriptions of skeletal lesions in ancient inhabitants of Tennessee and Kentucky support his conclusion that syphilis (i.e., a treponemal infection) was the cause of pathology observed in several individuals. Jones remarks (p. 66) that the tibiae are, in many cases, "thoroughly diseased, enlarged, and thickened, with the medullary cavity completely obliterated by the effects of inflammatory action, and with the surface eroded in many places." Skeletal involvement was not confined to the tibial shafts but included the cranium, clavicle, sternum, and other long bones. Significantly, Jones notes the symmetrical distribution of the skeletal lesions. The crania are described (p. 66) as exhibiting lesions "in which a network of periosteal deposit had been formed, and which had been perforated by ulcers, subsequently forming and assuming the annular type." Williams (1932:966) examined some of the skulls in the Jones collection and verified the presence of stellate scars in one specimen that he also attributed to syphilis. Soon after Jones's disclosure, claims of pre-Columbian syphilis in the Americas proliferated (e.g., Gann 1901, Lamb 1898, Langdon 1881, Orton 1905, Parrot 1879), although much of the purported evidence was deemed inconclusive by others (e.g., Hyde 1891; Putnam 1878:305; Whitney 1883). As with the European material, difficulty in differentiating disease processes, incomplete skeletons, and absence of archaeological context precluded reliable diagnoses in most cases. Even the most conservative, however, described skeletal lesions in ancient American remains that they admitted might have been due to syphilis (e.g., Hyde 1891:128; Whitney 1883:366). Williams (1932:976–77) considered reported cases of syphilis from several areas in North and South America "as nearly free from suspicion as any that can be found."

In the Southeastern United States, reported evidence of pre-Columbian treponematosis abounds. Following Jones's (1876) report, Lamb (1898) described syphilitic lesions in a skeleton excavated by Clarence B. Moore at Lighthouse Mound, in northeastern Florida. Moore (quoted in Bullen 1972:157) found the percentage of pathological specimens and degree of skeletal involvement in the 74 individuals recovered remarkable and indicated that "cranial nodes" were apparent. The skeleton examined by Lamb (1898:63–64) was not accom-

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panied by the skull but exhibited "lesions of osteoperiostitis, both hyperostotic and ulcerative," on the shafts of the long bones. Williams (1932:968) also examined this individual and agreed that the lesions "were in all probability syphilitic."

Bullen's (1972) survey of prehistoric skeletal material from Florida reveals considerable evidence suggestive of treponematosis. Enlarged long bones exhibiting encroachment upon the medullary cavity have been recovered from the Tick Island Archaic site, radiocarbon-dated to 3300 B.C. (p. 166). Burial 352 from Palmer Mound (FSM 97527) presents the most convincing case of pre-Columbian treponemal infection in Florida (pp. 138–50). The site belongs to the Weeden Island period and dates to A.D. 850. The nearlry complete skeleton of an adult female displays cranial caries sicca (see also Hackett 1976:110) and lesions on several long bones. The right humerus shows focal areas of destruction surrounded by diffuse osteitis and dense reparative bone, the radii and left fibula are slightly thickened with some periosteal new bone formation, and the left tibia is expanded and irregular. A radiograph of the left tibia reveals multiple lytic areas surrounded by sclerotic bone that Hackett (1976:110) has identified as superficial cavitation of nodes—diagnostic of syphilis. Additional remains from Palmer and several other prehistoric sites (mostly Weeden Island, A.D. 850–1350) exhibit treponemal lesions (Brothwell and Burleigh 1975:394; Bullen 1972:150–62; Iscan and Miller-Shaivitz 1985), indicating that the Palmer burial is by no means an isolated case.

Treponematosis has also been identified in a prehistoric skeletal series from Georgia. The remains of 265 individuals from Irene Mound (A.D. 1200–1450), near Savannah, reveal widespread inflammatory response with marked diaphyseal expansion in the lower legs and arms (Powell 1988c). Few cranial and naso-palatine lesions are noted, but in some cases focal lytic lesions of the skull vault are apparent. The demographic and anatomical patterning of skeletal lesions suggests endemicity rather than venereal transmission.

Syphilis is proposed as the cause of bone pathology in several specimens from northern Alabama, including a cranium in which the palate has been almost completely eroded and only the remodeled edges remain (Rabkin 1942:220–21, fig. 6). No specific provenience is provided for the syphilitic remains, but the sample includes material from as late as A.D. 1400–1600 (p. 218). Prehistoric pathological remains suggestive of syphilis at Moundville, Alabama, were first noted by Moore (1907:339–40). One skull that has received considerable attention in the literature (e.g., Bullen 1972:163–64; Hackett 1976:109–10; Haltom and Shands 1938; Williams 1936:785–86) displays extensive erosion and new bone formation on the frontal, resulting in the stellate scars characteristic of caries sicca. Hackett (1976:110) indicates that although the changes in this skull are not typical caries sicca, "in which the nodules are smaller and of more regular size," the diagnosis of treponematosis is "fully supported by the presence of similar changes in [crania found in] European medical museums and in Australian anthropological collections." Two tibiae from another individual present thickening due to osteoperiostitis, probably a result of treponemal infection (Williams 1936:786).

In an examination of over 500 individuals at Moundville, Powell (1988*a*) observed a "high prevalence of subperiosteal apposition on lower limb long bone shafts and moderate prevalence of cranial stellate lesions." Periostitis of major long bones is reported in 207 cases, of which 72% appear minor in extent and well-healed at death (Powell 1988*b*). The absence of the dental stigmata associated with congenital syphilis and the frequency of healed tibial lesions have led Powell (1988*a*,*b*) to attribute the observed pathology to a nonvenereal treponemal syndrome (yaws or endemic syphilis).

A possible case of treponematosis from the Late Woodland Hardin site in the North Carolina piedmont has been described by Reichs (1987). The skeleton exhibits destructive and proliferative changes resulting in node formation, expansion and cortical thickening of long bone shafts, medullary encroachment, and pathological fracture, with both cranial and postcranial involvement. Although the lesions are suggestive of treponematosis, Reichs recognizes that the overall pattern of pathology in this individual may be due to the synergistic effects of multiple diseases.

An apparent case of congenital syphilis in a six-to-seven-year-old child (U.S. National Museum of Natural History, Smithsonian Institution collection [hereafter NMNH], No. 379177) from Virginia dates prior to A.D. 1400 (Ortner and Putschar 1985:207–10). Abnormal reactive bone is evident in a frontal lesion, and the surface of the nasal aperture displays thickened, porous, periosteal bone. The extant deciduous incisors have hypoplastic defects so severe that, in three, the superior portion of the crown had broken off before death. The deciduous and first permanent molars are unaffected, as is an observable unerupted permanent incisor. Postcranial skeletal involvement is extensive. The shafts of both tibiae are thickened, with periosteal expansion occurring primarily on the anterior aspect. The other long bones also exhibit periosteal apposition and diaphyseal expansion, although to a lesser degree. A similar process is apparent in several metacarpals and metatarsals. Dactylitis is more common in yaws than in syphilis (Steinbock 1976:143); however, a congenital disease is indicated by the development of hypoplastic dental defects at about the seventh fetal month (Ortner and Putschar 1985:210). Thus, a pattern in which the bones with minimal overlying tissue are most severely affected, as is commonly observed in treponematosis, accompanied by congenital dental defects suggestive of Hutchinson's teeth is strong evidence of congenital syphilis. Although such dental stigmata are not pathognomonic of congenital syphilis, they are associated with characteristic bone lesions in about 50% of all cases, and the diagnosis in such instances is "very reliable" (Steinbock 1976:106).

The skeleton of a 25–35-year-old male (NMNH 385788) from a site radiocarbon-dated to A.D. 925 provides additional evidence of treponematosis in prehis-