NO PLAGUE IN THE LAND? INFECTIOUS DISEASES AND THEIR IMPLICATIONS FOR THE PRE-COLUMBIAN-TRANSOCEANIC-CONTACTS CONTROVERSY[1]

by

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Summary

The supposed epidemiological "virgin-soil" character of the 1492 New World with respect to most serious Old World infectious diseases has caused many to reject the possibility of significant pre-Columbian contacts by sea between the hemispheres. However, most Old World diseases' putative absence in the Americas may be a result of: 1) a lack of those diseases in contact source areas at the times involved; 2) sailors' immunity to acute infections owing to childhood exposure; 3) on-board elimination of any such infection, owing to voyage lengths' exceeding lengths of incubation times plus contagiousness time; and 4) lack of concentrated American populations large enough to support endemism, so that any introduced acute infections would soon have died out. Thus, the *seeming* pre-Columbian non-presence in the New World of most Old World communicable diseases does not demonstrate that important pre-1492 contacts did not occur. Contagious diseases that apparently *were* shared between the hemispheres included syphilis, tuberculosis, and typhus. Although not at present demonstrable, there is some reason to suppose that one or more of these shared maladies may have been transferred across the oceans well before 1492. Key words: disease, epidemiology, transoceanic contacts, Americas.

Whilst my physicians by their love are grown
Cosmographers, and I their map. . . .

—John Donne, 1635

THE EFFECTS of post-1492-introduced Old World diseases upon the immunity-lacking native populations of the Americas were devastating. Introduction of livestock diseases also had indirect negative demographic effects on humans, by causing declines in some native game-animal populations, e.g., bighorn sheep (Kemper 2000:93, 97-98). Beyond human demography, however, apparent presences and absences of specific diseases raise issues that relate to the highly contested question of contacts across the oceans before the time of Christopher Columbus (Jett 1983). Geographers Martin R. Lewis and Kären E. Wigen (1997:142-43) expressed the common informed view:

> the earth's largest oceans posed the greatest challenges to regular communication in the premodern era. . . . The paucity of transoceanic contacts before 1500 created dramatic ecological as well as cultural differences between Afro-Eurasia and the rest of the inhabited globe. Among other things, it was the resulting vulnerability of the Americas . . . to the supercontinent's virulent pathogens that made European conquest there especially swift and ruthless.

It is the intention of the present article to assess aspects of the history of communicable diseases and their interhemispheric exchange, from the point of view of their implications for the transoceanic-contacts issue.

I. MICROBIAL DISEASES AND A "VIRGIN-SOIL" HEMISPHERE

The Post-Columbian Demographic Collapse in the New World

The Old World gave rise to a much greater number of serious microbial diseases than did the New. This reflects the larger size of the Afro-Eurasian land mass and earlier agricultural intensification, population densification, and urbanization there, as well as, importantly, the close association in the Eastern Hemisphere between people and their several species of domestic and commensal animals plus the presence, in the tropics, of closely related primates, a number of important human diseases having begun as animal maladies (McNeill 1976:51-53; Kiple 1993:36; Diamond 1997:196-97). In the Bering Strait region of the Arctic, mainland Asia and America are only some 87 kilometers (54 miles) apart and intercontinental human contact was continuous; nevertheless, this region acted as a "cold screen" that filtered out from entry into the Americas many of the disease organisms of Eurasia adapted to less severe climates. In addition, insect and annelid vectors for warmer-climate pathogenic diseases were unable to pass through the Arctic, excepting those vectors that lived continuously in the warm microclimates of their hosts' bodies (Newman 1976:668).

THE PRE-Columbian populations of the Americas have been considered, then, "virgin soil" with respect to most important Eastern Hemisphere microbial pathogens. Historian of epidemics Suzanne Austin Alchon (2003:31) wrote, "American populations . . . remained in isolation from a variety of acute infectious diseases still unknown in the Western Hemisphere." Archaeodemographer Henry Dobyns (1980:275) hyperbolically asserted that "Native Americans truly inhabited a pre-Columbian earthly paradise free of the diseases evolved in the Old World." It is certainly the case that when one of the early post-Columbian European contributions to the natives of the West Indies—communicable Old World diseases such as smallpox, measles, and plague—arrived, almost none of the Indians possessed any noticeable inherent immunity to most of them, unlike Europeans, who, after sickening to a lesser degree, more often than not recovered from many of these maladies,

owing to a degree of natural immunity. The populations of Europe had been exposed to typhoid fever, *Variola minor* smallpox, chickenpox, mumps, and other infectious diseases, and, in warmer areas, various tropical diseases, for generations; and, accordingly, in areas of endemism (self-sustaining disease presence) the most susceptible individuals had been weeded out over the generations and a certain, if incomplete, innate, genetically inherited immunity obtained (varying greatly in degree according to the nature of the disease), causing symptoms to be relatively mild (e.g., Curtin 1997:140-41).[2]

But, as geographer W. George Lovell (1992:426) put it, "Whoever watched as Columbus came ashore . . . witnessed the beginning of a conquest that would cause the greatest destruction of lives in history," mainly from disease. By 1548, for instance, the circa-quarter-million native Taíno inhabitants of 1492 Hispaniola had been reduced to 500 souls, followed by full extinction (Cook 1997:44). As the post-Columbian occupation and colonization continued, with immigration of both Europeans and African slaves, these introduced diseases had horrendous impacts on aboriginals everywhere in the hemisphere, and the latter expired in droves. This sometimes-literal decimation[3] often spread well ahead of direct European contact, "softening up" the indigenes for easy military defeat or domination. It was disease far more than force of superior arms that allowed the takeover of the hemisphere by the Spanish, Portuguese, French, and British (Dobyns 1983:9, 11, 24; Lovell 1992; Dennevan 1992; Kiple 1993:539, 1010).

Disease Susceptibility and the Pre-Columbian-Contacts Question

THIS SUPREME susceptibility of the Native Americans of circa A.D. 1500 to foreign infectious diseases has struck many scholars as irrefutable evidence of lack of important previous contact with the Eastern Hemisphere other than in Alaska and, in late pre-Columbian times, in Newfoundland. Had such contact occurred, it has been argued, the New World natives would already have been exposed to such diseases, which would have remained manifest in the aboriginal populations; the latter would also necessarily have evolved some significant degree of resistance to the maladies. Here is how one physician and medical historian put it:

> If Indian ancestors had encountered these European diseases, hundreds o[r] thousands of years before Columbus arrived, then the susceptible Indians would have died at that time causing only those with greater immunity to survive and produce children. But this did not occur because the Indians remained isolated. (Settipane 1995:3)

YET, AS ALICE Kehoe (1998:21) observed, "there has not been serious testing of the hypothesis that any transoceanic contact would have devastated the Americas as the post-Columbian invasions did." Therefore, a primary question to be asked regarding the issue of possible early transoceanic contacts is: Is there any way that such contacts could have occurred without the introduction and continuing presence of these insidious Old World maladies and the consequent development of native innate immunities? It is odd that this question has never before been addressed in any depth. Isolationists have seemingly believed that the implications are so obvious that further investigation is not necessary, while diffusionists have apparently felt that the evidence of contact is there and that, therefore, however it happened, acute infectious disease must have been excluded, the precise reasons being irrelevant or undiscoverable.

At the outset, it should be said that there are many instances in American archaeology of demographic crashes and abrupt

disappearances of cultures. One of the best known is the Classic Maya collapse of about A.D. 900, but the circa A.D. 1300 Anasazi abandonment of large areas of the American Southwest as well as other depopulations have occasionally engendered hypotheses of introduced diseases' playing a role (e.g., Cockburn 1971:52). Nevertheless, these notions are unproven at the moment, many alternative possible explanations have been offered (e.g., drought and political collapse), and the geographic patterns of depopulation do not always appear to be expectable ones for epidemic (Jett 1983:380).

Ages of Infectious Diseases

LIKE ANYTHING else, diseases have histories in time and geographic space. In considering the question of introduced diseases, we need to take account of the fact that because disease organisms have quite short life cycles, rapid evolution of infectious diseases (bacterial, viral, and fungal) is possible—witness the sudden emergence (or recognition) of poliomyelitis in 1840,[4] Fort Bragg fever in 1942, o'nyong-nyong in 1959, Lassa fever, Lyme disease, toxic-shock syndrome, and Legionnaire's disease in the 1960s and 1970s (with Lyme being retrospectively identifiable by 1909); AIDS and new strains of hepatitis in the 1980s; and sin nombre hantavirus, Ebola, mad-cow disease (Levins et al. 1994; Diamond 1997:200, 205, 208), and West Nile, Hendra, and Nipah viruses in the 1990s; not to mention new strains of influenza and malaria. Therefore, we cannot assume that all or even most of the implicated post-Columbian maladies referred to above, at least in their later, more-virulent forms, were abroad in the Old World in early times. *Many of the proposed pre-Columbian contacts may have occurred before the emergence of at least most of these virulent diseases, or prior to their arrivals in the areas initiating overseas contacts.* Without documentation, it should not be assumed that any particular disease was present in any particular area or era.

To be sure, certain pestilences were anciently present in the Eastern Hemisphere; there exist in the ancient literature some quite recognizable descriptions of symptoms of presently known diseases. A number of epidemics were recorded during Classical times, although most are not definitively identifiable as to the specific diseases involved. However (despite the 430-B.C. smallpox epidemic of Athens and the 395-B.C. one of Syracuse [Austin 2003:23-25]), in general "The Mediterranean world in the centuries before the first emperor of Rome appears to have been an environment abundantly populated but relatively free of at least major epidemic disease" (Kiple 1993:508). Let us look, then, at what is known about the ages of the more prominent diseases concerned.

One of the major modern contagious maladies (water-borne) cholera, native to the Ganges Delta, is first recognizably mentioned in India at about 400 B.C. but, although devastating, as far as we know remained confined to that subcontinent for the next 2,000+ years, despite India's being at the heart of the vigorous Indian Ocean maritime trade network extending from East Africa to Southeast Asia, with connections beyond. "Most of this spread [of cholera] has occurred since 1847, when the modern history of the disease outside India begins" (Kiple 1993:414, 541, 642).

HUMAN malaria probably goes back to Neolithic Africa; it is attested in pre-dynastic Egypt circa 3200 B.C. (Miller et al. 1994). It appears to have arrived in Greece (or at least have become common) no later than the fifth century B.C., reaching Rome by the first century A.D., and it is also pre-Christian-Era in China and India. In contrast, influenza "is probably not a very old human disease. . . . there is no clear evidence of its spread among humans until Europe's Middle Ages, and no undeniable evidence until the fifteenth and sixteenth centuries"—although it does seem to have been in Japan by the ninth century

Table 1.

Earliest documentable dates for introduction of selected Old World diseases to the Americas (source: Dobyns 1983:11-23, 279-80; notes added).

Disease	Date	Place	Years since 1492
smallpox	ca. 1516[1]	Hispaniola	ca. 24
typhoid fever	1528	Gulf Coast	36
measles (prob.)	1531[2]	Sinaloa, Mexico	39
plague (poss.)	1545[3]	Mexico	53
influenza	1559[4]	Colombia	67
typhus (prob.)	1586[5]	Carolinas	94
diphtheria	1601[6]	Mexico	109
scarlet fever (prob.)	1637	St. Lawrence Valley	145
yellow fever	1647[7]	Barbados	155
cholera	1832	Quebec	340

[1]More likely, 1518-1519; possibly, 1511 in Yucatan (Cook and Lovell 1991:219; Reff 1991:99-100; Austin 2003:62), although unlikely, according to Crosby (1997:98).

[2]Diagnosis unsure (Cook and Lovell 1991:27-30). Measles were present in Guatemala in 1532 (Austin 2003:71, 73).

[3]Diagnosis unsure (Cook and Lovell 1991:27-30). In the case of the 1545 Mexican epidemic of hemorrhagic fever, which yielded ca. 80% Indian mortality and little Spanish, the symptoms do not quite match a known disease (Acuna-Soto, Romero, and Maguire 2000:733). Plague was possibly present in Nicaragua/Honduras and the Andes in 1531 (Austin 2003:73).

[4]Probably, 1493 in Hispaniola, from hogs (Cook and Lovell 1991:223; Bianchine and Russo 1995:17; Cook 1997:45; may be pre-Columbian, see endnote 5).

[5]1576, according to Kohn (1995:208); possibly in Mexico in 1545 (Austin 2003:69).

[6]Slight possibility of 1559, Mexico (Cook and Lovell 1991:35-36; Austin 2003:69).

[7]Diagnosis unsure (Cook and Lovell 1991:27-30). Possibly among Spaniards in Hispaniola in 1493 (Bianchine and Russo 1995:17).

and has been attributed to first-millennium-B.C. China. Old World 'flu is not definitely attested in the Americas until after the importation of African slaves. (Swine-introduced) influenza *may* be implicated in Hispaniola in 1493 (Kiple 1993:40,379,498-99,808; also Grmek 1989:279-81; Cook and Lovell 1991:223; Cook 1997:62; Bianchine and Russo 1995:17; Austin 2003:19-20), but some set 1559 as its first firm appearance in the hemisphere (see Table 1).[5]

ALTHOUGH old in the Middle East, (louse-borne) typhus's first severe western Mediterranean outbreak, resulting from an introduction from the Levant, did not occur (in Spain) until A.D. 1489-1490 and the disease did not become firmly established in Europe until the seventeenth century; the United States—continuously and intensively in touch with Europe—was not significantly affected until the early nineteenth century, although probable typhus appeared in the Carolinas in 1586 (Dobyns 1983) and may, in fact, be pre-Columbian in the New World (see Part II, below). (Food- or water-borne) typhoid fever is probably ancient in India and elsewhere (Austin 2003:17, 19) but is difficult to distinguish in the record.

(Flea-borne) bubonic plague, which may have originated in pre-Christian southern Asia (Austin 2003:19) or in North Africa (Lobell 2004:12), was absent in ancient Greece (Grmek 1989:89). The first pandemic of which we are aware involving plague—that of Justinian—began in Egypt in A.D. 540. Although plague re-erupted here and there for another two centuries and never died out afterward, it had long and mysterious periods of relative quiescence, not surviving in Europe except by reintroduction from the Middle East; the next thousand years saw only two great pandemics, the devastating Black Death of Europe (A.D. 1340-1390) that followed new stress on overland traffic with China, and the Great Plague of A.D. 1665-1666. Although some think that disease outbreaks in seventh- and thirteenth/fourteenth-century China may have been plague, "The first epidemic in China, which we have substantial reason to believe was plague, was that first striking Yunnan in 1792." It spread from there by sea: "along with Australia and Eastern Africa, North and South America were infected *for the first time*" (Kiple 1993:355, 630; emphasis added)—although Dobyns (1983:264) placed it in Mexico in 1545 and there is a possibility of its presence in Central America and the Andes in 1531 (Austin 2003:73).

Mumps was described by Hippocrates (fifth century B.C.) as well as for Roman times, and was recorded in Japan by the A.D. 900s. However, although measles (morbilli, rubeola) is thought to be ancient in China, its first clear description is that of the Persian physician Rhazes (Abu Bakr Muhammed Ibn Zakariya Al-Razi) circa A.D. 900 (Kiple 1997:80); it also appears to have been present in tenth-century Japan). "There is no reason to believe that the ancient inhabitants of the Mediterranean knew of measles and rubella ['German measles']" (Grmek 1989:336), and "Measles probably first appeared [only] during the Middle Ages in France," according to Cartwright (1972:131; also Dobyns 1983:9, 11, 24; Lovell 1992; Denevan 1992; Kiple 1993:539, 1010), not reaching Australia until 1854 (others feel that some of the ancient Roman and Chinese epidemics may have involved measles and that they were at least pretty clearly present by the seventh century. Measles is first attested in the New World only after about 39 years of post-1492 contact, becoming the biggest killer after smallpox. Yellow fever (probably of West African origin) is not well documented until the seventeenth century—first, in Brazil (Cartwright 1972:6-19, 83-84, 115, 142, 145, 157; Grmek 1983; Dobyns 1983:18; Reff 1991:108; Kiple 1993:279, 347, 355, 379, 410, 416, 541, 808, 873-74, 888, 1082, 1102; Settipane 1995:1; Austin 2003:17), although in 1526 Gonzalo Fernández de Oviedo indicated that it was

present among Spaniards in Santo Domingo in 1493 (Bianchine and Russo 1995:17).

RUBELLA was not described until 1619, although it may previously have been confused with measles and smallpox. The first clear mention of scarlet fever, "a relatively new disease," is from Italy in 1553, when it was differentiated from chickenpox, a disease of unknown but perhaps not tremendous age. According to some authorities, scarlet fever was absent in pre-1500 China, although others say that it was distinguished there from smallpox, chickenpox, and measles by the sixth century. Diphtheria was first recognized as such in 1882 and was considered a new disease in the early modern period, although a few epidemics in ancient Greece and in sixteenth- through eighteenth-century Europe involved diphtheria-like maladies and it is said to have appeared in Mexico in 1601 (barely possibly, 1559) and in Peru in 1614. Whooping cough (pertussis) "cannot with certainty be traced back further than the mid-sixteenth century and it was almost certainly unknown to the ancient world," although symptoms described in thirteenth-century Korea are consistent with whooping cough (Kiple 1993:1095; also, 395, 541, 682, 987, 991, 1093; also, Grmek 1989:335, 337; Dobyns 1983:11-23, 279-80; Cook and Lovell 1991:35-36; Austin 2003:20).

Many microbes have more than one strain, and, as indicated above, "Viruses and other microorganisms undergo significant genetic changes when exposed to a new host environment, changes often resulting in new and more virulent strains of microorganisms" (Reff 1991:124); in fact, bacteria are the "most evolutionarily active organisms on our planet" (Gould 1997b:14).

Smallpox has an interesting history in this connection. It could not have existed prior to population growth and nucleation during the Neolithic. The mummy of Pharaoh Ramses II (d. 1234? B.C.) and two others of the same millennium exhibit smallpox pustules (Sandison 1980:32; Austin 2003:17), and DNA tests have verified the disease's presence (JaRue S. Manning, personal communication, 1997). It *may* be implicated in Roman-period Mediterranean epidemics (not in Classical Greece, according to Grmek 1989:89) such as that of Antoninus (A.D. 165-189), and it is said to have been introduced into North China by the Huns about 250 B.C. It is documented in Europe in A.D. 581 and in Japan in the 700s. It is plainly described by the circa-A.D.-900 Baghdad-based Rhazes. But with regard to India, where the disease is thought to have originated by 1500 B.C., "Ralph Major . . . holds that . . . the first Indian description of *virulent* smallpox is not found until the sixteenth century," when the first description of an epidemic is recorded (Cartwright 1972:116, 118; emphasis added; also, Kiple 1993:355, 378, 390, 414, 539, 1009; Austin 2003:17, 20)—about the same time that it began its post-Columbian devastation of Native (and immigrant) Americans (it first arrived in Hispaniola no earlier than 1516 and most likely in 1518-1519—some 26 years after Columbus' first voyage; Dobyns 1983:11). Similarly, historian Philip Curtin pointed out that "There are two types of smallpox. *Variola minor*, less deadly and less likely to scar its victims, had been known in Europe since antiquity. In Columbus's day, infected Europeans suffered only a three- to ten-percent mortality rate. But *V. major* is first attested only around the mid-1500s" (L. 1992). "It did not become a major demographic check in Europe until the sixteenth and seventeenth centuries" (Kiple 1993:1010; also, Crosby 1986:199-200). American Indians' suffered a circa-95-percent mortality rate (Settipane 1995:2).

A VERY interesting fact concerning smallpox in India is that "Inoculation for smallpox seems to have been known to the Hindus from a very early age [first millennium B.C.]. . . . certain classes . . . are purported to have enjoyed a certain amount

of immunity from the disease. . . . since ancient times" (Kiple 1993:414). (Sailors are not, however, mentioned as one of these classes.) Inoculation of a different sort was also known in China, reportedly initially among Daoist (Taoist) hermits in southwestern Szechuan, where it was recorded in the tenth century; it may have been done as early as the second century A.D. and was widespread by the sixteenth. The practice is recorded in Egypt by the thirteenth century (Needham 1970:375; Temple 1986:135-37; Austin 2003:17, 20).

DISEASES may alter not only spontaneously in situ but may also change evolutionarily and/or in terms of manifestation, in response to changes in ecology, as these factors alter in an area over time or as when the organism is introduced into a new physical and/or sociodemographic environment (e.g., Curtin 1997:138). Plague is essentially a rodent disease; it becomes seriously troublesome to humans when host rodents live in close association with people. Most rodents shun humans, and transmission to the latter is normally accomplished only via fleas from infected rats, especially the black rat, *Rattus rattus*, a human commensal of less-severe Old World climates (there is also an uncommon but virulent pneumonic form of the disease that can develop, which spreads directly from person to person via breath and sputum). Because black rats infested ships as well as homes, plague was spread by sea as well as overland; yet, although it is possible that it was present earlier, it is not documented in North America north of Mexico before 1899. Plague, a bacterial infection, confers little acquired immunity on its sufferers, a fact facilitating its spread (Cartwright 1972:17-18, 29-32, 38, 51-53; Kiple 1993:526, 629). On the other hand, to the extent that with respect to human infection the rat is plague's (and typhus's) definitive host, the pre-Columbian absence of *Rattus rattus* in the New World could help explain why plague appears not to have been established there in pre-Columbian times—although that would still leave the question as to why the rat itself did not become naturalized then (Davies 1986:117-18), as it did post-Conquest (on smaller vessels, rats might have been eliminable—none was seen on the experimental *Brendan* voyage [Severin 1978:161], for example—and cats were routinely taken along to control rats). It is also the case that American rodents such as native rats, prairiedogs, and ground squirrels were potential reservoirs for plague, but these are not human commensals. The minimal clothing worn among most tropical American indigenes, being less favorable to harboring fleas than ample European or East Asian garb, could also have been an inhibiting factor. Too, the black rat may not have been as common in the Old World East, where the more aggressive but not human-companionable and less-susceptible brown rat (*R. norvegicus*) seems to have prevailed (ultimately spreading into Europe and largely displacing the black rat and thereby reducing the incidence of plague).

Diseases and Acquired Immunity

ALTHOUGH epidemics—including of plague—certainly often spread from port to port, one wonders whether, in the case of some maladies (mainly, viral ones) that convey post-infection immunity, repeated exposure did not lead to a kind of "curtain of immunity" along some coasts. With respect to smallpox, historian Alfred Crosby (1986:200) wrote, "Most adults [in Europe], especially in the cities and ports, had had it and were immune." Merchant sailors, who visited a variety of far-flung ports of call during their careers, would have been exposed to a variety of diseases early on, and would presumably have contracted—and, if surviving, acquired immunity to—more diseases than the average landlubber. It should also be noted that for millennia before the nineteenth-century development of germ theory, contagion—the passing of diseases between human beings—was recognized as a means of their spreading. For example, a

circa-1700-B.C. letter found at Mari, Syria, orders people to avoid all use of an ill person's possessions in order to avoid contagion (Sasson 2002:6.). The Romans posted guards at the gates of the city to control infection. In the Middle Ages, lepers were routinely driven off or isolated. The influential physician Avicenna of Bukhara (eleventh century A.D.) "noted the contagious nature of some diseases. . . . [This led to the introduction of quarantine as a means of limiting the spread of infectious diseases" (Tschanz 1997:31). Every medieval European port of note had its lazaretto, where visitors were confined prior to being permitted to land. With this in mind, we can suppose that a captain recruiting a crew would have rejected any obviously ill individuals., a captain recruiting a crew would no doubt have rejected any obviously ill individuals. He might even have kept the crew in isolation prior to the voyage in order to minimize the chance of infection on board.[6] *A disease-free crew may have been the rule rather than the exception*, at least with respect to most of the maladies under discussion.

ANOTHER consideration is what might be called the "time-filter factor." As Crosby (1986:197) put it, "Not only did very few people of any origin cross the great oceans, [so it is generally supposed,] but those that did must have been healthy or they would have died on the way, taking their pathogens with them." For most infectious disease organisms to remain viable, they must have a host. Those "that required transmission by intermediate host [such as a mosquito] would die once the territory of the necessary intermediate hosts was left behind" (Cockburn 1963; note, however, that some potential for mosquito-larvae survival existed, as in water butts). For directly infectious diseases, "a continuous chain of susceptible contacts is necessary to sustain transmission" (Kiple 1993:887). With the pneumonic form of plague, the death rate is 95 to 100 percent, and demise comes after only a day or two; the implication is that anyone initially infected with such plague would not survive a prolonged ocean voyage to pass on the infection upon landing (Seaver 1996:89). In the case of infectious diseases such as the poxes and mumps that impart eventual permanent immunity to their victims, if a voyage is relatively slow and especially if it involves a small craft carrying a small party, any disease organism is likely to have run its course during the crossing; thus, before journey's end, *all personnel would either be dead or disease-free and immune, and unable to transmit the sickness to residents at the landfall.*

Crosby (1997:96-97; echoed by Austin 2003:63) wrote,

> How can the evident absence of smallpox [in the pre-1518 Caribbean] be explained, if the American Indian was so susceptible, and if ships carrying Europeans and Africans from the pestilential Old World were constantly arriving in Santo Domingo? The answer lies in the nature of the disease. It is a deadly malady, but it lasts only a brief time in each patient. After an incubation period of twelve days or so, the patient suffers [symptoms lasting about two weeks]. . . . The whole process takes a month or less, and after that time the patient is either dead or immune. . . .
>
> Consider that, except for children, most Europeans and their slaves had had smallpox and were at least partially immune, and that few but adults sailed from Europe to America in the first decades after

> [Columbian] discovery. Consider that the voyage was one of several weeks, so that, even if an immigrant or sailor contracted smallpox on the day of embarkation, he would most likely be dead or rid of its virus before he arrived. . . . Consider that moist heat and strong sunlight, characteristic of a tropical sea voyage, are particularly deadly to the smallpox virus. The lack of any rapid means of crossing the Atlantic in the sixteenth century delayed the delivery of the Old World's worst gift to the New [by a quarter century].

AND SIMILAR statements can and have been made about others of the diseases in question (e.g., on measles: Cook 1997:62; on yellow fever, Kiple 1997:86).

Assuming 1) a complement of ten individuals (the number needed for a schooner-sized Roman vessel [White 1984:213]; some standard-sized Roman merchantmen had crews as small as five [Jurisic 2000:8]), one of these ten persons having contracted mumps on the eve of embarkation, and 2) a fully susceptible crew, a maximum latent (non-infectious) period of 18 days (other diseases have shorter periods—e.g., eight to twelve days for the poxes[7]; see Table 2), and a contagious period of eight days (longer for some maladies, e.g., up to a month or more for whooping cough; Anderson and May 1991:31; Kiple 1993:871, 986, 1093; Cook and Lovell 1991:218), a pathogen could, if passing from each ill individual only at the end of the possible term of contagiousness, survive for 260 days (nearly nine months; longer, if the crew was larger, shorter if smaller) and thus would endure through any crossing of reasonable length. However, such an absolutely worst-case scenario is so highly unlikely as to be hardly worth considering even with a fully susceptible crew, because such diseases are quickly transmitted among people in close quarters (Diamond 1997:202-03); it is far more probable that in the confines of a small ship any such disease would spread to all susceptible crew members within the initial four days of exposure, so that after one month or less everyone would be dead or immune and incapable of passing on the disease when they reached the other side of the sea.

Table 2.

Number of days of latency and infectiousness of selected Old World diseases. Source: Anderson and May 1991:31.

Name of the disease	Period of latency (non-infectiousness)	Period of infectiousness	Total duration of the disease
Chickenpox	8-12	10-11	18-23
Diphtheria	14-21	2-5	16-26
Measles	6-9	6-7	12-16
Mumps	12-18	4-8	16-26
Influenza	1-3	2-3	3-6
Rubella	7-14	11-12	18-26
Scarlet fever	1-2	14-21	15-23
Smallpox	8-11	2-3	10-14
Whooping cough	21-23	7-10	28-33

THIS TIME-filter factor would be more effective in the case of the vast Pacific than of the much narrower Atlantic. Early post-Columbian Mexico-Philippines crossing times ranged from Thomas Cavendish's 1587-88 traverse from Baja California in 56 days in 1564, to as much as 172 days (Lessa 1975:200-03). On the Atlantic side, in 1492 it took Columbus but 33 days to travel the circa 6,840 kilometers (4,250 miles) between the Canary Islands and the Bahamas (although he had taken six days to sail from Palos, Spain, to Las Palmas and tarried almost a month in the Canaries)[9]; in 1493, he shortened that time to 29 days, averaging 9.6 kilometers per hour (5.2 knots), and in 1502 to an astounding 21 days (the swiftest modern yachts can do it in a couple of weeks; Sandstrom 1983:184). The 1497 crossing of John Cabot (Giovanni Cabotto) from Bristol to Newfoundland lasted no longer than Columbus's first traverse, although for many years most crossings took about twice as long (Morison 1974:178; Parry 1981:220; Jacobson 1991:44:22). *Mayflower*, for example, took 64 days in 1620 (Chapman 1973:15). *Only when voyaging became routinely more rapid with the multiplication of masts and sails, and involved ever-larger ships and crews and, especially, complements of passengers (especially of European children and young, disease-susceptible African slaves*—see below*), might acute infectious maladies have had a good chance of still being abroad on shipboard at the time of landing.*

Even substantial numbers of personnel hardly guarantee disease transmission. The Norse settlement in Vínland included either 65 or 165 persons, and no serious sickness was reported; there is no indication of transmission of European disease to Native Americans at that time or during sporadic Norse contacts with northeastern North America over the next few centuries (McGhee 1984:12; Damas 1996:333). Perhaps even more striking is that there is no evidence that the circa-70,000 Norse settlers of Greenland passed diseases on to the indigenous Eskimo (Austin 2003:90; Seaver 1995). Columbus's 1492 expedition was disease-free. His 1493 expedition to the West Indies involved 17 ships and 1,500 colonists, yet no epidemics were initiated by personnel of this or other Columbian voyages (Crosby 1986:50; Cook 1997:45).[10] "During the first decade of the sixteenth century, more than two hundred ships crossed the Atlantic; on board were thousands of European settlers and their microorganisms. By 1510, some ten thousand Europeans resided on the island of Hispaniola. . . . During this same period, epidemic disease [plague and typhus or influenza] was rampant in southern Spain. . ." (Austin 2003:62). Yet, other than for what may have been swine-introduced 'flu (eight sows accompanied the 1493 voyage) there is no documentation of any of the diseases under discussion having been contracted by the native peoples during this time. Likewise, although it may have occurred we have no record of disease introduction to Brazil between Cabral's landfall in 1500 and the epidemic of 1552 (Austin 2003:85).

DESPITE these limiting factors for the acute infections, malaria, which is documented for Classical times and earlier in Europe and which does not convey immunity, would not—being chronic and uneliminable from the body—have been filtered out during long voyages and, if present among the complement, would have been transmitted wherever its vectors, mild-climate *Anopheles* mosquitoes, were present. There have been a few murmurings about possible pre-Columbian malaria (and yellow fever) in the Americas (e.g., Hoeppli 1969:54; Thornton 1987:45-46), but current evidence is against its having been present there or in most of the Pacific (Wood 1975; Kiple 1993:483, 539). In fact, it seems not demonstrably to have been in post-Columbian North America before 1684 (Dobyns 1983:23, 27).

It should also be borne in mind that since some of the aforementioned diseases, such as

mosquito-borne yellow fever and malaria, are tropical/subtropical and require particular genera of insect hosts, their apparent absence in the pre-Columbian New World in no way argues against contacts between peoples of higher latitudes or from tropical/subtropical areas historically free of these diseases, such as the Pacific islands. Further, "Although it is now generally accepted that the anopheline mosquitoes that spread malaria were on hand in the New World, . . . the *Aedes aegyptae* vector of yellow fever [and dengue fever] most probably was not. Thus both vector and virus had to be imported and to reach human populations dense enough to support them" (Kiple 1993:499, 540; also, Verano and Ubelaker 1992:239).

PATHOLOGIST Francis E. Cuppage (1994:6) asserted, with regard to eighteenth-century European ships, "Tuberculosis, smallpox, malaria, and typhus took their toll." John Toohey (2000:86) mentioned syphilis, malaria, fluxes (dysentery), typhoid fever, tropical fevers, and various parasites but not smallpox, chickenpox, or measles. Interestingly, actual records of the British and American navies of around 1800 show that respiratory ailments, including colds, influenza, pneumonia, and tuberculosis, accounted for nearly half the illnesses on board. Malaria was also important, and dysentery and liver disorders, along with syphilis and gonorrhea, are also noted as common and rheumatism and the like occasional. In the warmer climes, additional health hazards mentioned included yellow fever, typhus, cholera, and perhaps plague. *Not* mentioned in general summaries were the acute infections mumps, measles, rubella, scarlet fever, smallpox, and chickenpox—i.e., most of the major killers of the colonial Americas (Estes 1995:45). These are "childhood diseases" in populations in which they are well established, and adults, having had the maladies in their early years, are typically immune (Cartwright 1972:114-16; Cook and Lovell 1991:218; Kiple 1993:524, 872, 986). Presumably, *most sailors and adult passengers had had these diseases prior to going to sea and were immune and so not infections.*

Cockburn (1971:52; also, Kohn 1995:207; Crosby 1997:99) cited Spanish chronicler Bernal Diaz's report that when Spaniards (under Pánfilo de Narváez) inadvertently introduced smallpox into New Spain (Mexico), with catastrophic effect, it happened via an infected *Negro* that they had brought with them; "The Spanish [themselves] were not affected, for they already had had the disease in childhood"; in Spain, it was rare that anyone had not contracted the infection before adulthood.

Immunity for measles settles at about 90% of the adult population, scarlet fever at some 86%, with susceptible individuals living mainly in isolated settings (Kiple 1995:872, 991, 1092). This means that for most communicable diseases that convey immunity, the average expectable number of non-immune individuals in a ship's crew of ten from a typical area of endemism would, *at most*, be one—who, if infected, would be dead or no longer infectious within a month at the maximum, too short a time for early ships to have completed the crossing. Noble Cook and George Lovell (1991:221) flatly stated, "it would be necessary to have a group of children on board a fleet to transfer measles across the Atlantic."

IN CONTRAST to non-colonial voyaging, colonialism involved bringing still-susceptible children along, as well, often, as young African slaves. Further, the contact established was essentially continuous, allowing for repeated reintroductions of diseases, thus repeatedly devastating even populations too small to support endemism (see below); and, ultimately, continuing colonization led to foreign-derived populations large enough to support endemism (not until the nineteenth century in North America for smallpox and measles; Kiple 1993:525). We should not make the

mistake of thinking that any putative pre-Columbian voyaging was for purposes of colonization on the European imperialist model, especially involving importation of slaves; there is no reason to suppose that it was, although small-scale colonizations of other types may well occasionally have occurred.

Population Size and Endemism

BECAUSE THEY have short periods of infectiousness and are passed directly from person to person and not via non-human vectors (Anderson and May 1991:21), acute infections "are diseases of the crowd and cannot have flourished in the primitive races of small, scattered settlements," the first such "herd diseases" probably having arisen with early Middle Eastern intensive cultivation and incipient urbanization (Cartwright 1972:114). Epidemiologist Aidan Cockburn (1971:50-51) "theorized that many infections require minimum host populations for permanent maintenance; if the sizes fall below the threshold levels, the infections die out." The concept of "critical community size" is now well recognized; it is the minimum magnitude of a substantially interacting population necessary to provide recruitment of susceptible persons at a rate high enough for the malady to persist (Davis et al. 2004:736). "To sustain themselves, [infectious diseases] need a human population that is sufficiently densely packed, that a numerous new crop of susceptible children is available for infection by the time the disease would otherwise be waning" (Diamond 1997:203). This is especially true of the acute community infections rubella, cholera, diphtheria, mumps, measles, smallpox, and chickenpox, which spread quickly but are of short duration in the body. Arctic population densities would have been much too small to maintain such diseases (achieve endemism). Sparse, scattered population may account for the fact that, like American Indians, most native Siberians had not evolved genetic immunities and were highly susceptible to diseases introduced and reintroduced in historic times by Russians, especially smallpox and measles, suffering population reductions of from 44 to 80 percent (Forsyth 1991:82-83); although pre-Russian-conquest contact between Siberia and neighboring populous regions was not intensive, no one suggests that there was none, and in fact there was a significant fur trade.

In 1846, 7,864 people (including 92 immune) on the Faeroe Islands were insufficient to support measles indefinitely (Cockburn 1963:81-82). In the Fiji Islands, there was a 70-year gap between outbreaks of introduced and reintroduced measles (Cook and Lovell 1991:29-30). Again, the illness was unknown in isolated Greenland until 1951, at which time the island had a population of about 30,000; and although the disease was thrice reintroduced there over the next two decades, infecting every susceptible individual, on each occasion it subsequently disappeared from the island. In modern Iceland, with a population of some 200,000, measles was repeatedly reintroduced, dying out between epidemics (Anderson and May 1991:84). Both smallpox and measles were repeatedly introduced to populous medieval Japan but did not become endemic (Kiple 1993:379). In fact, F. L. Black's (1966) study of disease records of several islands found breaks in the continuity of measles in all populations of less than 500,000. Cockburn (1971:50-51) estimated that it would require a coherent population of around a million to produce endemism for measles—a possibility in the pre-Columbian New World, if at all, probably only in highland Central Mexico (Crawford 1998:52). Physical anthropologist Marshall Newman (1976:668) went farther (and perhaps too far): "Old World 'crowd-type' diseases such as measles require a human reservoir of several million for maintenance. . . ." An empirical study of (interconnected) towns in England and Wales showed, for measles, a somewhat lower community endemic fadeout threshold, of

about 800,000 persons, and a real drop-off below about 315,000 (Keeling and Grenfell 1997). McNeil and McNeil (2003:80) also wrote of up to 300,000 interconnected persons being needed to provide sufficient susceptible hosts to sustain infection, and Anderson and May (1991:21, 82, 84-86) spoke of from a quarter to a half million.

OWING TO these minimum host-population size requirements, with respect to high-density-dependent diseases 1) *a "virgin-soil" population like Mexico's is supposed to have been would have to have been far, far larger than one in say Europe (or than Mexico's in fact was*[11]*) to support endemism; because of the lack of immunity, as soon as an imported deadly disease hit, the population would quickly be reduced to levels below those necessary to sustain the disease*[12]; and 2) *overseas voyages originating in, and/or arriving at, uncrowded places, would have had a low likelihood of carrying persons infected with these diseases and/or to have resulted in endemism in the contacted areas.* In fact, had English connections with their North American colonies, or Spanish and Portuguese ones with theirs in South America, ceased after, say, fifty years, never to be resumed, *those few infectious diseases introduced might well soon have died out in those places, leaving no medical evidence that they had ever been present,* not to mention that they had devastated the indigenous populations. *Nor would much improvement in genetically inherited immunity have developed among American populations to protect against future reintroductions,* since only one or a very few cycles of epidemic would have played out as a selective force, rather than a continuing exposure over centuries as was the case with Old World populations large and dense enough to support endemism. This is illustrated by the case of the Hopi of Arizona, among whom even after repeated smallpox epidemics, traditionalists who contracted the disease but refused Western medicine in an 1898 epidemic, still suffered a 74-percent mortality rate (Dobyns 1983:13).[13]

While the above remarks apply to the short-duration acute crowd infections, Cockburn (1963:81) also observed that "Even pathogens that can live in their hosts like commensals for months find it difficult to survive if the population is too small."

Summary of Part I

The presence, prevalence, and effects of any communicable disease reflect a complex mix of historical, environmental, etiological, behavioral, nutritional, and demographic factors (including birthrates). The diseases that devastated aboriginal American populations following the Columbian colonization were, in several cases, not demonstrably in existence or in existence in their later virulent forms at the various times and/or places proposed as sources of pre-Columbian transoceanic influences (although the field of historical epidemiology is young and the possibility of new evidence being discovered always exists); specifically, as far as present data indicate we can probably safely eliminate the supposed pre-Columbian absences of at least influenza, cholera, rubella, yellow fever, diphtheria, whooping cough, scarlet fever, the virulent *Variola major* strain of smallpox, and possibly measles and chickenpox, as relevant to most or all proposed pre-Columbian transoceanic contacts. With regard to any acute infections remaining in consideration, these were mostly childhood diseases in their areas of endemism, and adult sailors and passengers would probably almost always already have acquired immunity and been incapable of being transmitters. In cases of slow voyages and small crews, any infectious diseases that did accompany the crew would likely have run their courses and have been eliminated prior to completion of the crossing. Finally, the acute crowd infectious diseases were maladies of areas with large and dense populations and could not have been

introduced when contacts did not originate in such areas. If but small native populations were encountered upon debarkation, the critical host-abundance threshold for pathogen invasion would not have been present (Anderson and May 1991:19, 86; Davis 2004:736). Even if such diseases *were* introduced and *were* decimating, recipient populations would normally or always have been too small (especially, following an epidemic) to support disease endemism and no diagnostic evidence of any epidemics would have survived, nor would significant inherited immunity have been attained.

MALARIA, and to a lesser extent, plague, do remain difficulties with respect to at least some possible contacts. Nevertheless, *the seeming lack, in the pre-Columbian Americas, of the Old World diseases discussed above cannot legitimately be used as a blanket exclusionary phenomenon with respect to proposals of early transoceanic encounters.*

Finally, we may observe that none of these diseases is documentable as even having been transmitted from Europeans or Africans to Native Americans during the initial quarter century following 1492 (Table 1). The first certain people-introduced acute infection, smallpox, did not reach the Caribbean until latest 1518 or later, and others did not demonstrably arrive until far later. If putative pre-Columbian absence of Old World diseases in the Americas "proves" that no pre-Columbian contacts occurred, then (ignoring possible swine-introduced influenza and unverified yellow fever) it also "proves" that the voyages and non-epidemiological impacts of Christopher Columbus, John Cabot, Pedro Álvares Cabral, Joaõ Fernandes, Alonso Hojeda, Amerigo Vespucci, Gaspar Corte-Real, Gonçalo Coelho, and perhaps Vasco Nuñez de Balboa and Juan Ponce de Leon, or any of Columbus's other pre-1518 successors, never took place. If, following Dobyns (1980), we (provisionally) accept 1545 as the first New World occurrence of plague and use this as a criterion, then the list is expanded to include Hernán Cortés, Ferdinand Magellan (Fernão de Magalhães), Francisco Pizarro, Francisco Vásquez de Coronado, Gerolamo da Verrazzano, Sebastian Cabot, Álvar Nuñez Cabeza de Vaca, Jacques Cartier, Diego de Almagro, Hernando de Soto, Francisco de Orellana, and so forth. Or, using yellow fever alone, first tentatively documented for 1647, we can similarly dismiss the reality of the sixteenth-century Spanish settling of the Caribbean, New Spain (Mexico), New Granada (northwestern South America), and Peru, as well as the English settling of Virginia in 1605 and Massachusetts in 1620! And since measles didn't strike Greenland until 1951 (Cockburn 1971:50-51), Eirík the Red and company—not to mention the later pre-1951 Danish occupation—must be mere figments of the imagination!

The point is, of course, not that these historically known expeditions and conquests did not occur, but that even massive transoceanic contact does not guarantee disease transmission.

II. INFECTIOUS DISEASES SHARED BETWEEN THE HEMISPHERES

WE HAVE discussed Old World diseases in the context of their apparent absences in the pre-Columbian New World. But were there any notable communicable diseases that clearly *were* shared between the hemispheres? The answer is a qualified "yes."

Syphilis

Sexually transmitted syphilis, a kind of spirochete-caused treponematosis, is one that has long been debated (Crosby 1972:122-47). One hoary theory is that since it is (supposedly) not proven to have existed in the Old World before 1500, it is one of the few important maladies of New World origin and was brought back to Europe by

Columbus's crew in 1493, as "the Indians' revenge," giving rise to more than one medical manifestation.[14] *Virulent* syphilis does appear to have first spread in Europe in the 1490s, later diffusing to Asia and Africa (Thornton 1987:45).

A SECOND theory rests upon the fact that yaws, a non-lethal, largely non-venereal, supposedly African infection, is a treponematosic disease that is bacteriologically indistinguishable, or virtually so, from syphilis; it is thought by many to manifest itself as yaws, spread by skin contact, among largely unwashed, unclothed peoples but as venereal syphilis among those (as Europeans) who are normally fully dressed and more cleanly. Yaws might, then, have been brought to Europe by slaves imported from Africa by the Portuguese, there, in a new environment, transforming to syphilis through adaptation or mutation (Cartwright 1972:58-62; Thornton 1987:45)—except that syphilis is recorded historically slightly earlier in Europe than the first documented African-slave importations (indubitably, though, a few Black Africans had reached Portugal earlier on).

According to some thinking, however, the yaws, syphilis, and pinta organisms (the last, an uncommon American Indian treponematosic disease), although morphologically and serologically essentially identical, are each biochemically distinct, and syphilis evolved from pinta or its ancestor after that ancestor came to the hemisphere via Bering Strait human migrations (Grmek 1983:133-42). "Clearly, then, despite decades of debate . . . there is still no agreement on their place or places of origin, nor on their antiquity" (Kiple 1993:1055).

We may next ask what evidence there is relevant to the pre-Columbian presence of yaws/syphilis in the two hemispheres. To begun with, compared to others American Indians are relatively resistant to syphilis (Settipane 1995:3), suggesting centuries of exposure to treponematosic disease. Archaeological indication of the pre-Columbian presence of syphilis is of four kinds. The first is depictions of characteristic skin eruptions on certain ceramic figurines of humans from Nayarit, West Mexico, such dermal lesions having been adjudged by some to be syphilitic, although parasitic dermopathologies could conceivably be represented (Vérut 1973; Servain-Riviale 1995:69, 70, 77).

The second (and potentially more definitive) class of archaeological evidence is the lesions that syphilis leaves on bones. As recently as 1993 it could be written,

> [T]here is (at least thus far) a dearth of evidence in Old World skeletal remains that would testify to the presence of syphilis in Eurasia prior to 1493, although the presence of yaws and [non-venereal] endemic syphilis [or bejel] has occasionally been reported. In the New World, by contrast, there is a great deal of positive skeletal evidence of pre-Columbian treponematosis [especially, in Peru]. . . . [Y]aws, endemic nonvenereal syphilis, and syphilis are all possibilities for the American infection. . . . venereal syphilis seems the least promising, because of an apparent absence of congenital syphilis in the skeletal material. (Kiple 1993:537, 1054)

DESPITE SUCH statements to the contrary, quite a lot of skeletal evidence consistent with venereal syphilis has been forwarded for the Americas (e.g., Cybulski 1980). Specifically syphilis-caused bone lesions on some pre-Columbian skeletons are clear, "even judged very critically," and the disease is mentioned in Mesoamerican traditions of great age as well as in Colonial chronicles and may be depicted in a mural at Teotihuacan, Central Mexico (Hoeppli

1969:4,102-03, 207-08, Pl. 8). In California's Santa Barbara Channel region, "Although rare, cranial lesions considered pathognomonic of syphilis are present in both island and mainland skeletal collections," going back as far as 4,300 years (Walker 1998).

Two *possible* cases of syphilitic lesions from medieval northwestern Europe were reported in 1992 (Verano and Ubelaker 1992:12 and passim; also, Baker and Armelagos 1988:710-11, 717-18, 721). However, as of 1997 the latest and most extensive skeletal studies had indicated no clear pre-Columbian syphilis in the Old World but its definite presence in New Mexico, Florida, and Ecuador as much as 1,600 years ago; yaws was identified in Ohio, Illinois, and Virginia and was at least 6,000 years old (R[ose] 1997:24).

Only lately has unequivocal evidence of a pre-Columbian presence of venereal syphilis been unearthed in Europe: one skeleton from Norwich, England that shows clear signs of the disease was carbon-dated to between A.D. 1300 and 1450 (Malakoff 2000), well before Columbus's famous voyage—a date that, for a time, fueled speculation that the Norse rather than Columbus were responsible for the importation. However, other, older possible examples have been found since, suggesting syphilis's presence in antiquity.

THE THIRD tool for identification of treponemal infection is immunological. In fact, treponematosis (not necessarily in syphilitic form) was identified by a syphilis-antisera test in an 11,000-year-old bear skeleton from Indiana (Rothschild and Turnbull 1987), and an immunological test revealed treponematosis in a 1240 B.P. Woodland-period human skeleton in North America (Ortner, Tuross, and Stix 1992:347), but what kinds of treponematosis was not ascertainable.

THE FOURTH and final method of identifying the presence of syphilis in skeletons is DNA studies. Treponemal (although not necessarily venereal) DNA has been recovered from pre-Columbian burials in Chile (Verano 1998:54-56, 219). Current research on the genomes of the various diseases discussed above should eventually clarify much regarding which form(s) of treponematosis is (are) involved (Pennisi 1998:325). Yaws and syphilis DNA can now be distinguished, but methods of investigating possible presence in archaeological skeletons is only now being developed (George M. Weinstock, personal communication, 2004).

Tuberculosis

Next, we may mention the pulmonary infection tuberculosis (TB), a disease that goes back to Neolithic times among humans in the Mediterranean region and to the Bronze Age in East Asia. It s found among some nonhuman animals and is widely thought to have become a human disease through close contact among people, their bovine domesticates, and milk products, and to have spread particularly in association with the crowding found in emerging Neolithic towns.

For over a century, there has been some reason to believe that TB may also have been present in the pre-Columbian New World. Indeed, in 1973 good osteological paleopathological evidence was published for a circa-A.D.-800 mummy from Peru. Since that time, DNA and much more skeletal-lesion evidence has proven numerous instances. Dates of probable cases begin at 160 B.C., with convincing examples commencing at about A.D. 290 or a bit later. Known South American occurrences are mainly in the Central Andean region, with a a small number in Northwest South America. There are very few finds in Mesoamerica, but the North American Southwest and eastern woodlands provide quite a number of cases,

all of post-A.D.-900 date (Roberts and Buikstra 2003:187-213; Conlogue 2002; Morell 1994; Buikstra 1981).

THE IMPLICATIONS of these findings for long-distance overseas contacts are not entirely unambiguous. "[R]ecent studies have not found that temperature, humidity, or other climatic factors influence either one's risk of developing tuberculosis or its course once the disease is developed," so the Bering Strait presumably would not have operated as a cold screen (Kiple 1993:521, 1061-62). But whether Arctic human population density was sufficient to sustain TB is questionable (Austin 2003:44). Roberts and Buikstra (2003:184, 193-94) averred that "no cases of TB have been identified in hunting and gathering populations," although other authors have listed this disease as a leading cause of mortality in such societies (Austin 2003:45). One writer has even proposed a de novo emergence in the Americas, which would seem highly dubious on more than one ground, including—if one accepts a bovine origin—the New World absence of cattle and dairying.

A complication has recently arisen in this last connection, however. A form of TB closer to the human than to the bovine has been identified in an extinct North American bison at about 17,870 B.P., and genomics implies that human tuberculosis does not derive from Old World bovine TB (Ramenofsky, Wilbur, and Stone 2003:250).

Regarding human TB alone, present data support a scenario of earliest American TB appearance in Peru followed by sea-borne introduction to West Mexico, with subsequent movement to the Southwest and to eastern North America—movement opposite the pattern that a Bering Strait point of entry would produce. Initial introduction to South America by sea from Asia best fits the evidence available so far; TB is a disease that continues in the host, so that the time-filter factor would not eliminate it on prolonged voyages. Still, in light of the aforementioned tubercular bison, other possibilities may certainly be considered.

Typhus

Typhus, an Old World disease, leaves no physical markers in the archaeological record. Still, one writer stated that typhus (or a similar fever) was "a very common disease in ancient Peru," and anthropological geneticist Michael Crawford (1998:56) wrote, "There is evidence to suggest that typhus . . . was pre-columbian in origin" in the Americas. In fact, Austin Alchon (2003:54) added, "One can make a very strong case for the existence of both endemic (flea-borne) and epidemic (louse-borne) typhus in the New World before 1492." The disease is likely implicated in late pre-Columbian epidemics in the Central Andes, Mexico, and North America (Austin 2003:37, 55-56). Colonial Peruvian populations appear to have had some degree of resistance to the disease, and there are possible depictions of its symptoms in Aztec pictures. But, louse-transmitted "Typhus is widely known as a disease of cold climates" (Kiple 1993:538, 1080, 1082) and so could have passed the Bering Strait. At the same time, unlike victims of the acute infections typhus survivors can be carriers, facilitating carriage across oceans, which is certainly an alternative avenue for introduction.

OVERALL CONCLUSIONS

WE HAVE arrived at several conclusions concerning communicable diseases and the transoceanic-contacts question. Firstly, we have seen that many putative source areas for transoceanic voyagers may have been free of some or all of the diseases implicated in the post-Columbian American "holocaust" and that, therefore, supposed absence of such diseases in the pre-Columbian Americas does not argue against contacts from those areas.

Secondly, transoceanic voyages likely usually involved small crews all or almost all of whose members would have acquired immunity to acute infections that they contracted and survived during childhood and unable to be carriers. Because of the limited durations of the acute infections, if any crewmember *was* ill at the time of embarkation, he would have been either dead or recovered during the normally long transoceanic passages, and incapable of introducing the disease. Even if certain diseases were nevertheless introduced from time to time, Old World acute infections would have been unable to maintain themselves for long in most or all of America owing to even existing dense populations' being insufficiently large to support endemism. For some illnesses, necessary disease vectors were absent in the New World as well.

ALTHOUGH syphilis, tuberculosis, and typhus do seem to have been in both hemispheres before Columbus, it cannot be shown that transfer was necessarily by sea; Bering Strait transfers remain a possibility, although an unlikely one, especially in the case of TB, whose earliest known New World occurrence was in South America.[15]

The infectious-disease picture thus hardly proves pre-Columbian contacts. However, an understanding of some basics about disease history, etiology, and ecology does lead to the conclusion that the alleged pre-Columbian absence in the Americas of many Eastern Hemisphere communicable maladies by no means *disproves*—or even justifies serious objections against—the occurrence of such contacts, and certain diseases shared between the hemispheres add some modest support to the notion of pre-Columbian contacts.

NOTES

1. Versions of this paper were presented at the 1998 annual meeting of the Association of American Geographers (Jett 1998) and at the 2001 joint annual meeting of the Institute for the Study of American Cultures and The Epigraphic Society. A somewhat condensed version will appear in my forthcoming *Crossing Ancient Oceans* (New York: Copernicus Books). I would like to acknowledge helpful comments by virologist JaRue S. Manning and three anonymous reviewers. The idea for the article was stimulated by a question from Terence Grieder.

2. A somewhat different perspective exists, however:

> In Europe, epidemics caused by smallpox, yellow fever, and influenza were extremely severe with high mortality. The mortality was somewhat higher in the New World because the effects were further exacerbated by starvation, slavery and physical exhaustion. Thus, it has been argued that Americans did not have any special sensitivity or susceptibility to imported Old World diseases. (Crawford 1998:51)

So far, there is specific support for this with respect only to measles (Bianchine and Russo 1995:13). It is the case, however, that Native American population decline was particularly dramatic in part because the Indians were impacted by a variety of exotic diseases within a mere century or two, sometimes simultaneously (Austin 2003:81-82).

3. WE MUST recognize, however, that demographic figures can be only very roughly approximated; for a severe methodological criticism of previous estimates, see demographic historian Cameron B. Wesson's (1998) *Numbers from Nowhere*. See, also, Austin 2003:47-72.

4. However, archaeology indicates that polio is pre-Christian in the Middle East (Austin 2003:17).

5. A good case can be made for pre-Columbian forms of influenza in the Americas. Its source could be ducks (Austin 2003:56-57).

6. This might at first seem unlikely before the post-1500 development of germ theory. In fact, "In the earliest medical literature [e.g., Aristotle,] there are vague expressions of the idea that invisible living creatures might be responsible for disease" (Anderson and May 1991:5). In any case, *contagion* theory does not depend on recognition of the microbial agency of contagion. There is the general "law of contact or contagion" proposed by James G. Frazer, in which humans have a tendency to feel that "having contact with someone or something . . . [has the] result [of] taking on the [good or bad] essence of that person or thing" (Simoons 1994:307). The Romans did post "gatemen to identify potential sources of infection in the city. . . . [although] the model of contagion was not widely accepted during antiquity and the Middle Ages, at least among the educated elite," the main exception having been with respect to lepers in Palestine and Europe, these being driven off or isolated (Kiple 1993:197; also, Austin 2003:26-27). The influential physician Avicenna of Bukhara (eleventh century A.D.) "noted the contagious nature of some diseases. . . . [This] led to the introduction of quarantine as a means of limiting the spread of infectious diseases" (Tschanz 1997:31). In 1479, "As the plague was rife in Venice, the port of departure, when the pilgrims left, they were not allowed to land during the voyage" (Ohler 1989:46).

7. A caution here is that an experiment revealed that smallpox microbes in scabs from pustules were viable after 540 days when stored in cotton at room temperature (Reff 1991:101-02). But although cotton garments would have been worn by many southern Asian voyagers, Europeans, Middle Easterners, and North Africans long had only linen and woolen clothing. Frequent salt-water wetting on board smaller craft as well as atmospheric-temperature swings and exposure to sunlight would no doubt rapidly render such organisms unviable.

8. Regarding chickenpox (and, presumably, similar diseases), "Few escape infection until adult life, and these usually live in isolated rural communities" (Kiple 1993:1092), not busy ports.

9. One of many false Columbus myths is that his 1492 voyage was unprecedentedly long and difficult but that the admiral's vision and determination prevailed against a frightened and mutinous crew; this is exemplified in lines from Joaquin Miller's nineteenth-century poem "Columbus":

> "Brave Admiral, say but one good word:
> What shall we do when hope is gone?"
> The words lept like a leaping sword:
> "Sail on! sail on! sail on! and on!" (Carman 1927:367)

Columbus did perceive his crew as becoming restive because the steady easterly trade winds engendered fear that no wind would be found to allow a return to Spain; this fear may have really existed or may simply have reflected Columbus's paranoid projection (Fernández-Armesto 1991:78-80).

10. The depopulation of Hispaniola began with what may have been influenza, carried not by humans but by swine aboard Columbus's ships on his second voyage (Kiple 1993:40, 498-99; Austin 2003:62).

11. "The population of the Central Mexican Symbiotic Region in 1519 can be calculated at between 2,600,000 and 3,100,000" (Sanders 1992:129). Mexico as a whole may

have held some 17,200,000 souls, the Andean region perhaps 14,000,000 (Austin 2003:163, 169).

12. The numbers of susceptibles would decline rapidly mainly because of the large numbers of disease deaths plus those caused by disruption of the food supply but also because all survivors of the infection would be immune (Anderson and May 1991:20, 81).

13. Cockburn (1963:76) gave an example in which TB mortality rates declined from about 90/1000 to .4/1000 among Saskatchewan Indians over some 70 years, and interpreted this as a consequence of selection for genetic immunity; however, it seems much more likely simply to reflect public health improvements among the Indians.

14. Wrote Girolamo Fracastoro, who gave the malady its name (in 1530), "If then by Traffick this plague was brought/How dearly, dearly was that Traffick bought!"

15. More information on these diseases will appear in John L. Sorenson and Carl L. Johannessen's forthcoming *Scientific Evidence for Pre-Columbian Transoceanic Voyages to and from the Americas*, Sino-Platonic Papers 113, CD-ROM ed., Department of Asian and Near Eastern Studies, University of Pennsylvania, Philadelphia, 2004).

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