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Body Lice, *Yersinia pestis* Orientalis, and Black Death

To the Editor: A scientific debate with public health implications wages: What caused the medieval European plague epidemics known as Black Death? Recent articles note inconsistencies between a rat flea-borne pandemic of *Yersinia pestis* (the bacterium that causes bubonic plague) and the documented characteristics of Black Death (1, among others). Ayyadurai et al. (2) acknowledged that a rat flea-only hypothesis does not fit Black Death observations, but they resolve theoretical transmission inconsistencies through a louse-borne hypothesis. Ayyadurai et al. base their surety of fact—that medieval “plagues” were caused by *Y. pestis* infection—on a 2007 study (3) in which 5 of 36 teeth of “plague” victims, none of which were dated to the Black Death era (1347–1351), contained biological evidence of *Y. pestis*. The 3 locations in that study were all port cities: 2 on the Mediterranean Sea and 1 on the Rhone River. As Duncan and Scott (4) note, bubonic plague most likely existed endemically near ship-borne trade, unlike the fast-moving epidemic fronts exhibited by medieval “plagues.” Moreover, Gilbert et al. (5) found no *Y. pestis* DNA in 61 skeletons from primarily nonport locations in England, France, and Denmark.

We do not dispute the authors' claim that *Y. pestis* might have been

present in some skeletons from port cities in France, or that body lice might, under certain circumstances, transmit the Orientalis biotype of *Y. pestis*; their work appears careful and considered. However, given the differences mentioned above and improved knowledge on the rapidity of virus mutation and worldwide transmission potential, we merely argue that the simplest explanation for medieval plagues has yet to be ruled out: that they may have resulted from a human-to-human transmitted virus. Adding complexity to an already complicated etiologic theory, and stating such as historical fact based on limited geography and sample size, does not seem congruent with Occam's razor.

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To the Editor: The letter of Ayyadurai et al. (1) reminded us of a little-known paper (2) on rats and Black Death by our colleague and mentor David E. Davis. He researched and wrote in his retirement after years of research and reflection on rat ecology and rodent-borne diseases (3,4). *Rattus rattus* is commonly recognized as the vertebrate host of flea-borne plague that swept through Europe in the 1300s, killing >50% of the population. Davis believed this explanation did not fit what he knew of the ecologic requirements of fleas and black rats. He studied reports of archeologic excavations and reviewed poems, medieval bestiaries, and paintings and concluded that these rats were scarce during the Black Death era.

His theory, based on historical information and investigative trips to Europe, was that invasive rats, if present, mostly occurred in low densities in port areas, not in rural inland areas. He noted that the expected rodent die-offs with bubonic plague were not associated with human epidemics and that rodent fleas would not have been active during winter to transmit plague. Flea-borne transmission from rodents usually causes a few deaths per household, but deaths of entire households commonly occurred in the medieval epidemics. Human-to-human transmission of pneumonic plague must have occurred, but as described by Ayyadurai et al., there was evidence of human bubonic plague, suggesting vector involvement. Davis did not present a viable reservoir/vector hypothesis for plague transmission; this and the later, well-known association of *R. rattus* and other rodents with plague throughout the world, may partially explain why his ideas received little attention. The finding that human body lice can be bubonic plague vectors suggests a mechanism for human-to-human transmission continuing during winter in inland areas and, as suggested by the authors, could also explain total deaths in households.

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In Response: Commenting on our recent demonstration that the human body louse was a likely vector of Black Death (the medieval European plague epidemics) (1), Welford and Bossak (2) point out that quantitative and qualitative inconsistencies in data for Black Death and modern plague argue against concluding that *Yersinia pestis* is the etiologic agent of Black Death (3). These authors acknowledge the paleomicrobiologic demonstration of *Y. pestis* in human remains collected at ports, yet they argue that such demonstration remains to be performed for human remains collected from inland burial sites (2).

Careful review of the literature indicates that 3 unrelated scientific teams have now demonstrated the

presence of *Y. pestis*-specific biomolecules in 14th–18th-century human remains in 11 sites in Europe. These locations include 7 nonport, inland sites ≤ 650 km from the coasts (4–6) in addition to 3 Justinian (nonport) locations (4,7). Therefore, the fact that *Y. pestis* was the etiologic agent of Black Death can no longer be disputed; the inconsistencies correctly noted by Welford and Bossak actually question the reservoir and the vector of *Y. pestis* during the Black Death and the following epidemics rather than its cause. McLean and Fall remind us that the cumulative work of their mentor, David E. Davis, suggested that black rat ectoparasites could not have been likely vectors of medieval plague in Europe, based on the facts that expected die-offs of rats were not reported and that the rodents' fleas would not have been active during winter in medieval Europe (8).

McLean and Fall acknowledge that our experimental data pave the way toward an alternative scenario of body louse-borne transmission of the Black Death. Such transmission of *Y. pestis* was observed by Blanc and Baltazard during a cluster of bubonic plague cases in households in Morocco during World War II (9). These authors demonstrated that the body louse could be infected when living on a septicemic patient, could stay alive for 7 days with infectious feces, and could transmit plague (9). Demonstration of *Y. pestis* in human lice collected from Black Death burials would be a step toward understanding the epidemiology of Black Death; this technically demanding approach has been successfully used to assess the transmission of typhus in soldiers of Napoleon's Grand Army buried in Vilnius, Lithuania (10).

In agreement with these observations and those reported by Welford and Bossak (3), our work clearly indicates that *Y. pestis* could be efficiently transmitted by the human louse (1), a potential vector of Black

Death because of its high prevalence in medieval Europe (11). Far from “adding complexity to an already complicated etiology theory,” as Welford and Bossak stated, it seems to us that the cumulative evidence provided by paleomicrobiologic demonstration and by our recent work (1) clarifies the epidemiology of the Black Death and the subsequent epidemics. Louse transmission of *Y. pestis* also explains inconsistencies rightly noted by Welford and Bossak and provides a reason for the current plague cases in poor areas of the world where poor hygiene is common. A search for alternative hypotheses, including the previous viral hypothesis for Black Death, may not be necessary (2).

The analogic reasoning based on observations of current infectious diseases cannot be applied to the medieval Black Death. Paleomicrobiologic evidence and historical data force us to change the paradigm and to question the established dogma about the epidemiology of plague. McLean and Fall remind us that, even in science, alternative hypotheses have trouble challenging dogma (8). Black Death is one of many areas at the intersection of microbiology and history for which many hypotheses have been proposed and none has received confirmation; these hypotheses have been repeated for so long that they became accepted as demonstrated truths.

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