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Rethinking the Columbian Exchange: Transoceanic Pathogen Circulation in the Age of Sail and Steam

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Rethinking the Columbian Exchange:
Transoceanic Pathogen Circulation in the Age of Sail and Steam

A thesis submitted in partial satisfaction
of the requirements for the degree
Master of Science in Biology

by

Elizabeth Naomi Blackmore

2023

ABSTRACT OF THE THESIS

Rethinking the Columbian Exchange:
Transoceanic Pathogen Circulation in the Age of Sail and Steam

by

Elizabeth Naomi Blackmore
Master of Science in Biology
University of California, Los Angeles, 2023
Professor James O. Lloyd-Smith, Chair

In the centuries following Christopher Columbus's 1492 journey to the Americas, transoceanic voyages opened unprecedented pathways in global pathogen circulation – commonly termed the "Columbian Exchange". Yet no biological transfer is a single, discrete event. We use theoretical modeling to quantify historical risk of shipborne pathogen introduction, exploring the respective contributions of journey time, ship size, ship susceptibility, transmission intensity, density dependence, and pathogen biology. We contextualize our results using arrivals data from San Francisco Harbor, 1850–1852, and from a selection of historically significant voyages, 1492–1918. We offer numerical estimates of introduction risk across historically-realistic journey times and ship population sizes, and show that steam travel and shipping regimes which involved frequent, large-scale movement of people both substantially increased risk of transoceanic pathogen circulation.

The thesis of Elizabeth Naomi Blackmore is approved.

Daniel T. Blumstein

Benjamin L. Madley

James O. Lloyd-Smith, Committee Chair

University of California, Los Angeles

2023

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Chapter One is a version of a manuscript currently in preparation: Elizabeth Blackmore and James O. Lloyd-Smith, 'Transoceanic Pathogen Circulation in the Age of Sail and Steam'. EB and JLS jointly conceived the project, developed methodology, and conducted formal analysis. EB curated data, conducted investigations, and prepared visualisations under JLS's supervision. EB wrote the original draft. EB and JLS both contributed to reviewing and editing.

Rethinking the Columbian Exchange: Transoceanic Pathogen Circulation in the Age of Sail and Steam

1.1 Introduction

In the centuries following Christopher Columbus's 1492 journey to the Americas, transoceanic voyages opened unprecedented pathways in global pathogen circulation. Fifty years ago, environmental historian and geographer Alfred Crosby coined the term "Columbian Exchange" to describe the ecological shifts that followed: a pivotal, irreversible transfer of plants, pathogens, and people between the "Old" world and the "New" [1]. Yet while "Columbian Exchange" narratives rightly characterize modern shipping as an important ecological force, no biological transfer is a single, discrete event. The Columbian Exchange was and is a centuries-long process.

Since the first publication of Crosby's work in 1972, a broad range of ecological scholarship has demonstrated the significance of human mobility and contact structures for pathogen ecology, evolution, and epidemiology [2]–[7]. Human mobility patterns affect the evolution, ecology, and epidemiology of influenza [8]–[10], measles [11], [12], Covid-19 [13], [14], cholera, [15], [16], malaria [17] and a host of other diseases. This raises a historical question. How quickly, and how uniformly, did transoceanic shipping create global pathogen ecosystems — and with what consequences along the way pathogen ecology, pathogen evolution, and for human health?

We contend that transoceanic pathogen transfer was slower, less uniform, and less inevitable than traditional "Columbian Exchange" narratives imply. In particular, we argue that acute respiratory pathogens such as smallpox, measles, and influenza rarely survived

early colonial voyages. Between short generation times, lengthy periods at sea, intense shipboard transmission, and crowded shipboard environments, these "crowd diseases" could rapidly exhaust all susceptible people on board long before a vessel reached port — leaving no pathogen to introduce.

The idea that onboard pathogens often went extinct among passengers and crew before a ship's arrival is qualitatively intuitive. Historians have made the point repeatedly [18]–[20]. However, a quantitative and theoretical investigation of shipboard outbreak duration can offer sharper insight into the historical contours of transoceanic pathogen circulation.

Previously, historical geographers Andrew Cliff and Peter Haggett have argued that technological innovation between the eighteenth and twentieth centuries — specifically, the emergence of steam travel — boosted global pathogen circulation by substantially reducing ship journey times [21]. Meanwhile, Paterson et al. have used stochastic epidemiological modelling to assess the plausibility of pre-1850 measles transfer from the United Kingdom to Australia, arguing that shorter journey times and rising ship population susceptibility (in particular, greater numbers of child emigrants) were both necessary for pathogen introduction across this route [22].

We offer a more general assessment of the risks of shipborne pathogen introduction using the toolkit of contemporary theoretical ecology. We present a stochastic SEIR (Susceptible, Exposed, Infected, Recovered) model which quantifies the probability that an outbreak will last a given duration across a range of shipboard conditions. We investigate the respective contributions of pathogen natural history, onboard transmission intensity and density dependence, ship population size, and ship susceptibility rate to overall outbreak duration. Finally, we use port data from Gold Rush-era San Francisco, California, 1850-1852, to explore the potential real-world implications of journey length, ship size, and natural history on transoceanic pathogen circulation.

Our results indicate that shipborne pathogen introductions were neither trivial nor inevitable. Ships were not simply pathogen vectors: they were populations. As with all

populations, the dynamics of pathogen extinction and survival were complex and highly contingent on population size, population composition, and population interaction. Thus, the history of transoceanic disease introduction is a story both of fundamental pathogen biology, and of human economics, technology, and behavior. Theoretical modelling can reveal how these forces interacted to shape global disease transmission.

1.2 Results

Transoceanic pathogen introduction requires an outbreak of infectious disease which lasts at least as long as a ship's journey time. To investigate the basic dynamics of shipboard outbreak duration, we simulate outbreak length in a fully susceptible population ($N = 100$) using a hypothetical pathogen which has characteristics typical of acute respiratory viruses (mean incubation and infectiousness periods of 5 days each) (Figure 1).

Historical accounts suggest transmission on board ships was substantially more intense than transmission in typical terrestrial environments (Appendix 1). Thus, we explore a broad range of transmission intensities. These are summarized by the epidemiological parameter R_0, or the average number of infections that a single person will produce in a fully-susceptible population.

We observe three outbreak duration regimes, all of which depend heavily on transmission intensity. Under strongly subcritical transmission ($R_0 \lesssim 0.8$), the majority of simulations result in zero transmission beyond the index case (Figure 1A). These "single-generation" outbreaks last only as long as the course of infection in a single individual, in this case an average of 10 days.

Under strongly supercritical transmission ($R_0 \gtrsim 5$), outbreaks are large and almost universally reach or exceed the threshold for ship herd immunity, $\frac{S}{N} < \frac{1}{R_0}$ (Figure 1B). The result is outbreak durations that reliably fall within a 35-55 day range, decreasing steadily as R_0 approaches 100. Occasionally, simulations under strongly supercritical R_0 also gen-

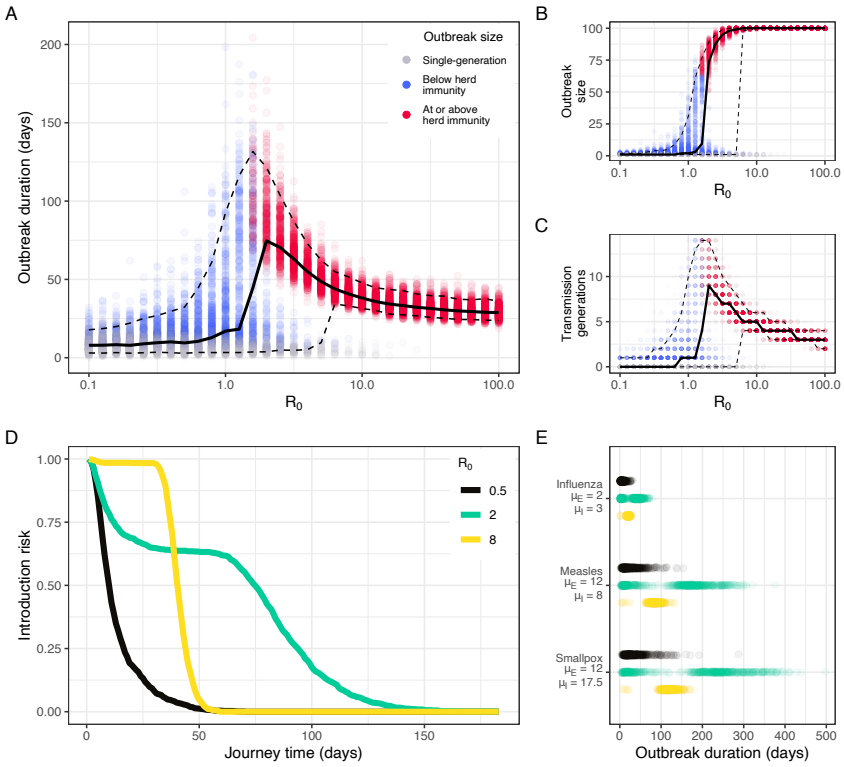

Figure 1.1: **Basic Dynamics.** (**A**) Outbreak duration, (**B**) outbreak size and (**C**) number of transmission generations by R_0, assuming a fully-susceptible population of $N = 100$ and a theoretical pathogen with $\mu_E = \mu_I = 5$ days and $k_E = k_I = 3$. Solid black lines show median outbreak duration, outbreak size, and number of generations. Top and bottom dashed lines respectively show 95$^{\text{th}}$ and 5$^{\text{th}}$ percentile outbreak duration, outbreak size, and number of generations. (**D**) Probability of at least one person in state E or state I ("introduction risk") for any given journey time by R_0, using the same population and pathogen parameters. (**E**) Outbreak length distribution by R_0 in a fully susceptible population of $N = 100$ for influenza, measles and smallpox, using epidemiological parameters detailed in Table A1.

erate short single- or two-generation outbreaks (Figures 1A, 1C). These are consistent with the occurrence of minor outbreaks due to random extinction in stochastic systems [23], [24].

Values of R_0 near criticality ($0.8 \lesssim R_0 \lesssim 5$), produce some of the longest outbreaks, with outbreak durations that peak around $R_0 \approx 2$. These are made possible by extended, multigenerational transmission (Figure 1C). Yet while near-critical conditions give rise to some of the longest outbreaks, they also result in the widest range of outbreak durations. An R_0 of 1 may result in outbreaks as long as 141 days, but median outbreak duration under these conditions is just 14 days.

Transmission intensity modulates a pathogen's overall introduction risk — here defined as the net probability that at least one passenger is carrying the pathogen (i.e. in state E or state I) upon arrival at any given journey length. Under strongly subcritical transmission (e.g. $R_0 = 0.5$), introduction risk decays rapidly with journey time, with 50% probability of introduction at 10 days and 25% probability at 17 days (Figure 1D). Under strongly supercritical transmission ($R_0 = 8$), pathogen introduction risk is sigmoidal: introduction is near certain ($> 90\%$) for journeys of 34 days or less, then falls rapidly for journey times exceeding this threshold. Introduction is least predictable for weakly supercritical values of R_0 ($R_0 = 2$). Here, introduction risk falls rapidly across the first two weeks of a voyage, but presents an extremely long tail: half of pathogens survive an 80-day voyage, while one in ten survive to 117 days. Thus, the relative introduction risk of weakly and strongly supercritical transmission depends on journey length. Strongly supercritical transmission is significantly more likely to result in pathogen introduction for journeys of 34 days or less, since intense transmission carries minimal risk of early extinction. But across journeys of 50 days or more, pathogen introduction is most likely under weakly supercritical transmission.

For real pathogens, introduction thresholds are governed by pathogen-specific natural history, above all by the durations of a pathogen's latent and infectious periods (Figure 1E). We explore outbreak length using consensus latent and infectious periods for influenza, measles, and smallpox at subcritical, near-critical, and strongly super-critical R_0 (Table

A1). The results demonstrate that relative introduction risk can be broadly inferred from pathogen natural history, even in the absence of shipboard R_0 estimates. At any R_0, smallpox typically survives longer on board a ship than measles, which in turn typically survives longer than influenza. Natural history also indicates some general introduction thresholds, which hold regardless of transmission intensity. For example, influenza introduction is extremely unlikely for journeys lasting longer than 100 days, regardless of R_0.

1.2.1 Incorporating Population Size and Susceptibility

Next, we expand our analysis beyond the unlikely scenario of one $N = 100$ ship with 100% population-level susceptibility to consider the combined effects of a ship's population size N, and initial proportion susceptible, $\frac{S(0)}{N}$, on ship outbreak duration.

In populations with some initial immunity to infection (e.g. where $\frac{S(0)}{N} < 1$), transmission intensity is most meaningfully measured as a pathogen's "effective" reproduction number, R_e. Because population immunity levels change over the course of an outbreak, this is commonly expressed as a function of time, i.e. $R_e(t)$. $R_e(t)$ is a linear function of a pathogen's basic reproduction number, R_0. Hence, $R_e(t) = \frac{S(t)}{N} R_0$, with critical transmission occurring at the threshold $R_e(t) = 1$. We consider effective shipboard transmission at $t = 0$, where $R_e(0) = \frac{S(0)}{N} R_0$

First, we vary the total number of people who are initially susceptible, $S(0)$, while holding $R_e(0)$ constant. We do so by fixing $N = 1001$ and back-calculating R_0 to maintain the same effective rate of transmission.

We observe a log-linear relationship between initial susceptible population size and outbreak duration at critical and supercritical values of $R_e(0)$ (Figure 2A). At $R_e(0) = 1$, increasing $S(0)$ has little influence on median outbreak duration but substantially increases 95th percentile outbreak duration. At $R_e(0) = 2$, increasing $S(0)$ increases both median and 95th percentile outbreak times. Finally, at $R_e(0) = 8$, increasing $S(0)$ dependably increases

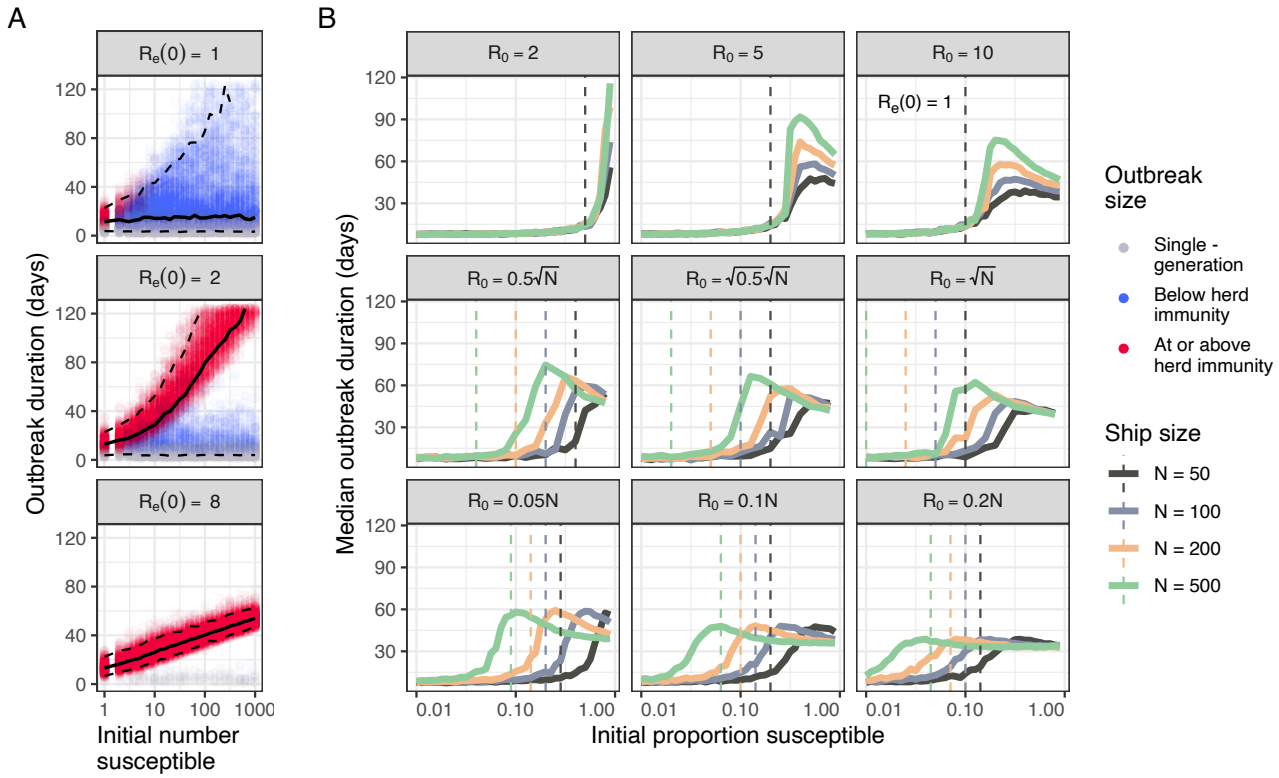

Figure 1.2: **Effects of Population Size and Susceptibility.** (**A**) Outbreak duration by initial susceptible population ($S(0)$), effective reproduction number ($R_e(0)$) and final epidemic size. We fix $N = 1001$ and back-calculate R_0 for each value of $S(0)$ to maintain constant $R_e(0)$. As above, we base simulations on a theoretical pathogen where $\mu_E = \mu_I = 5$ days. Solid black lines show median outbreak duration; top and bottom dashed lines show 95$^{\text{th}}$ and 5$^{\text{th}}$ percentile durations. Colors match those used in Figures 1A-1C and show single-generation outbreaks, outbreaks that reach herd immunity, and outbreaks that end before reaching herd immunity. (**B**) Median outbreak duration by density dependence, transmission intensity, ship size (N) and initial proportion susceptible ($S(0)/N$). Dashed vertical lines show $R_e(0) = 1$ by ship size. *Top row:* classical frequency dependence ($R_0 = \mu_I \beta_{fd}$), with β_{fd} values of 0.04 (left), 1 (center) and 2 (right) and $\mu_I = 5$. Dashed black vertical lines show $R_e(0) = 1$ for all N values. *Middle row:* intermediate density dependence ($R_0 = \mu_I(\beta_{fd}\beta_{dd}N)^{0.5}$), fixing $\beta_{fd} = 1$ and with β_{dd} of 0.01 (left), 0.02 (center) and 0.04 (right). *Bottom row:* classical density-dependent transmission ($R_0 = \beta_{dd}N$) with with β_{dd} of 0.01 (left), 0.02 (center) and 0.04 (right).

median, 5th percentile, and 95th percentile outbreak times.

Next, we vary N as well as $S(0)$. This opens the question of what relationship, if any, we should expect between N, S and R_0 on board the unique environment of a historical ship. Records from the time indicate that many vessels suffered from inadequate ventilation and extreme rates of crowding (Appendix 1). On land, these conditions generally give rise to "density-dependent" patterns of transmission, where contact rates scale linearly with population size ($R_0 \propto N$). However, ships were also famously structured and compartmentalized environments, which typically align with assumptions of "frequency-dependent" transmission (Appendix 1); here contact rates are assumed to remain constant, regardless of total population size ($R_0 \perp\!\!\!\perp N$).

In practice, we expect that effective density dependence varied substantially according to ship layout and construction, social norms, and pathogen-side biology. Thus we consider three density dependence scenarios: classical density dependence ($R_0 \propto N$), classical frequency dependence ($R_0 \perp\!\!\!\perp N$), and an intermediate mode of transmission ($R_0 \propto N^{0.5}$).

Under each scenario, we explore the effect of initial ship susceptibility, $\frac{S(0)}{N}$, on median outbreak duration across several total population sizes, N. In all circumstances, larger and more susceptible populations present greater risks of pathogen introduction across any given journey (Figure 2B). Yet they do so in different ways, and for different reasons.

Under classical frequency dependence, $R_0 = \mu_I \beta_{fd}$, where μ_I represents the average duration of an individual's infectious period and where β_{fd} represents the average number of onward infections that a single infected person would generate in a fully susceptible population. Critical transmission, $R_e(0) = 1$, occurs at the constant threshold $\frac{S(0)}{N} = (\mu_I \beta_{fd})^{-1}$. The threshold value of $\frac{S(0)}{N}$ required for $R_e(0) = 1$ is independent of total ship population, N. However, for any given $\frac{S(0)}{N}$, ships with greater N must have a proportionally greater number of susceptibles, $S(0)$. Since ships with greater $S(0)$ experience longer outbreaks at supercritical $R_e(0)$ (Figure 2A), ships with greater total populations display longer median outbreak times at $R_e(0) > 1$ (Figure 2B, top row).

Under classical density dependence, $R_0 = \mu_I \beta_{dd} N$, where β_{dd} represents the average proportion of a given population, N, that a single infected person would infect per day in a fully-susceptible population. Critical transmission occurs at the threshold $\frac{S(0)}{N} = (\mu_I \beta_{dd} N)^{-1}$. Multiplying both sides by a factor of N reveals that critical transmission depends solely on initial susceptible population size: $S(0) = (\mu_I \beta_{dd})^{-1}$. When N is large, this threshold for $S(0)$ represents a smaller fraction of the total population. But, in contrast to frequency-dependent transmission, $S(0)$ is constant at any given $R_e(0)$, and so peak outbreak duration does not vary across ships of different sizes. Rather, larger ship populations give rise to near-critical and supercritical transmission at lower thresholds of $\frac{S(0)}{N}$ (Figure 2B, bottom row).

Under intermediate transmission, $R_0 = \mu_I (\beta_{fd} \beta_{dd} N)^{0.5}$. Critical transmission occurs at the threshold $\frac{S(0)}{N} = \mu_I^{-1} (\beta_{fd} \beta_{dd} N)^{-0.5}$, and at the total susceptibility level $S(0) = N^{0.5} \mu_I^{-1} (\beta_{fd} \beta_{dd})^{-0.5}$. Under this model, larger ship populations reach critical transmission at slightly lower initial rates of susceptibility, have a critical population size that scales sublinearly with N, and hence display slightly higher outbreak length for any given $R_e(0)$ (Figure 2B, middle row).

Finally, it is worth noting that regardless of density dependence, ships with a higher rate of contact (represented either by β_{fd} or by β_{dd}) require lower initial susceptibility for critical transmission. Thus, more crowded ships require fewer susceptible people to achieve supercritical transmission, regardless of total ship size.

Thus, even in the absence of detailed reconstructions of ship transmission patterns, we can conclude that ships with larger, more crowded populations presented greater risks of pathogen introduction — be this by increasing total persistence times, decreasing the susceptibility fraction required for critical transmission, or both. In practice, the risk associated with larger ship populations was almost certainly boosted further by an increased chance of carrying at least one infected person on departure. We do not account for this difference, instead conditioning on the assumption that all ships depart with a single infected individual.

But in situations with low infection prevalence at the point of origin, this elevated chance of having at least one infected person on board at the time of departure would have increased a large ship's risk of pathogen introduction substantially. Ships with larger populations were both more likely to depart with infection on board *and* more likely to sustain this infection outbreak until arrival.

1.2.2 Historical Applications

Voyage characteristics such as journey time, population size, and population susceptibility varied substantially across time periods, transit routes, and ship technologies. We explore some of this variation – and its implications – using port arrivals data for Gold Rush-era San Francisco, 1850-1852, originally collected by historian and genealogist Louis J. Rasmussen (Figure 3)[25]–[27]. By the mid-nineteenth century, acute respiratory infections such as smallpox and measles had only recently begun to arrive across the Pacific basin. California saw its first region-wide outbreaks of smallpox and measles in 1806 and 1838, respectively [28]–[30]. Smallpox was first introduced to Australia in 1788 but did not see second introduction until 1829, while measles appears to have arrived for the first time in 1850 [22], [31]. Several Pacific islands saw initial introductions well into the late nineteenth century, including Hawai'i (Smallpox, 1853); Easter Island (Smallpox, 1863); Fiji (Measles, 1875); and Tonga (Measles, 1893) [31].

During the years 1850-1852, passengers journeyed to San Francisco from East Asia, Australasia, South America, and Europe. In an era preceding reliable transcontinental rail, ocean travel also provided one of the fastest and safest routes from East Coast North America to the newly-established state of California [32]. Median sailing times ranged from 7 days (from Oregon Territory) to 190 days (from Liverpool, England), with considerable intra-route variation (Figure 3A, Figure 3B, Table A2). Longer-range sail voyages displayed an especially broad range of transit times. For example, sail journeys from New York City could be as long as 283 days (on the *Primoguet*) or as short as 89 days (on the *Flying Cloud* — reportedly

"the fastest [sail] voyage ever recorded") [26, pp. 45, 198].

For several reasons, steam travel represented a phase transition in transoceanic pathogen circulation. In most cases, the technology dramatically reduced journey times. * Median transit times from Panama were 63 days by sail but just 20 days by steam. Meanwhile, steam reduced median journey times from Oregon from 7 days to just 3 days (Table A2). These shorter journey times would have increased risk of shipborne pathogen introduction significantly.

Second, steam ships transported some of the greatest numbers of passengers (Figure 3C). Steamers from Panama carried a median of 196 passengers and as many as 1,050, compared with a median of 53 and a maximum of 287 by sail (Table A2). Oregon steamers carried a median of 28 passengers and as many as 157, compared with a median of 4 and a maximum of just 12 by sail. Finally, steam vessels from New York City carried a median of 111 passengers and a maximum of 743, compared to a median of 5 and a maximum of 160 by sail. The only sail route that could compete with steam travel on passenger numbers was the route from Hong Kong, which transported a median of 163 passengers and a maximum of 553. As demonstrated above, larger passenger numbers would have substantially increased ships' capacity to sustain infectious disease outbreaks until arrival.

Finally, steam travel also represented some of the most frequent voyages (Figure 3C). Particularly striking are the 103 steam journeys from Panama that arrived June 1850 – June 1852. More frequent journeys result in a greater cumulative probability of pathogen introduction across any given period.

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*For reasons that are unclear, four steam voyages originating in New York City had transit times longer than 200 days: the *SS New Orleans* (210 days), the *SS Sea Bird* (240 days), the *SS Goliah* (279 days) and the *SS Chesapeake* (364 days) [25, pp. 27, 83] [26, pp. 43, 182]. The *SS Sea Bird* ran aground on San Martine on its way to California and had to be repaired [26, p. 76]. Meanwhile, the extraordinarily long voyage of the *SS Chesapeake* astonished contemporary observers, with companies who had shipped goods on the vessel suing for damages on its arrival [33]

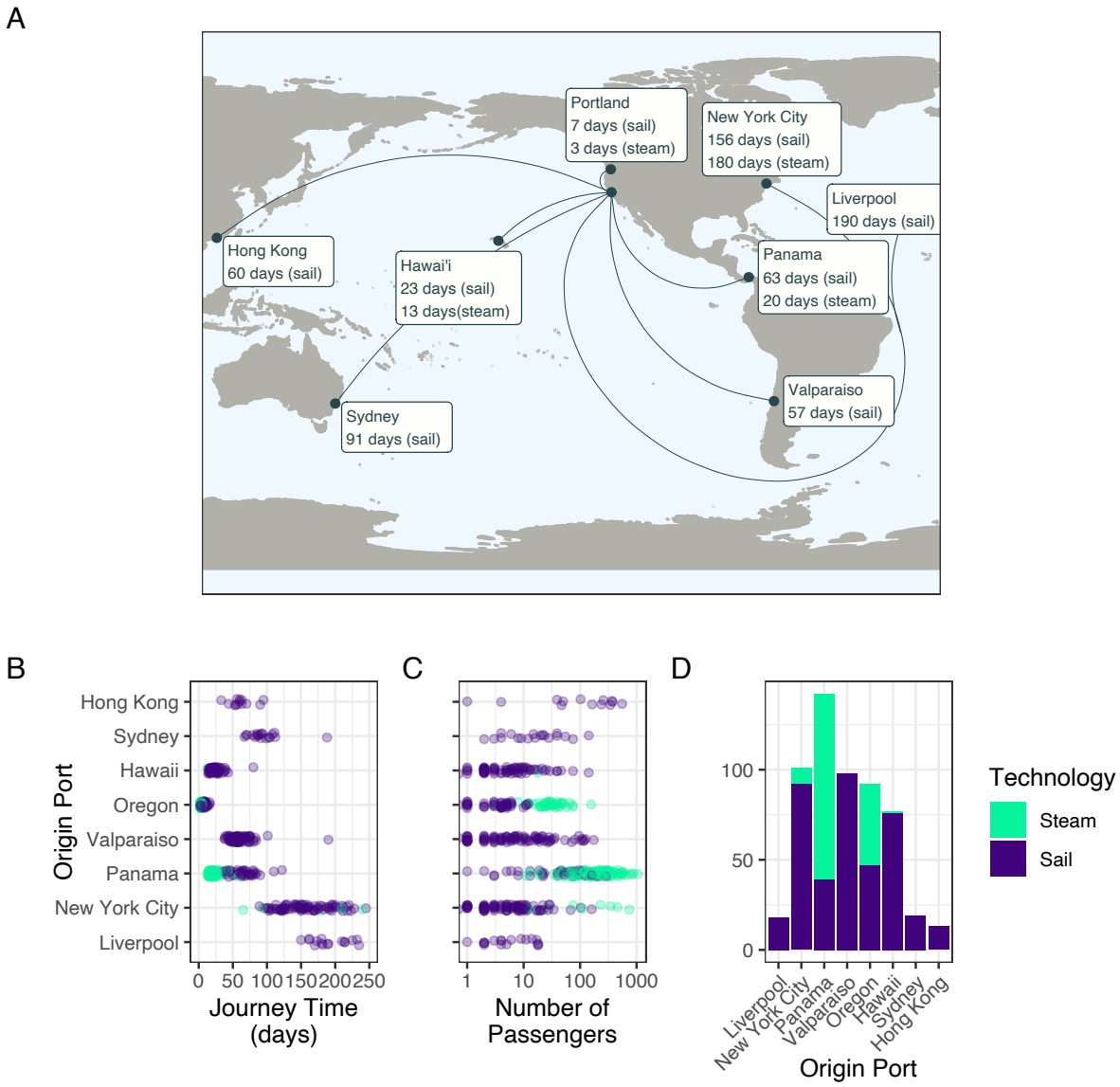

Figure 1.3: **San Francisco Arrivals, June 1850 – June 1852.** (**A**) Map of arrivals into San Franscisco Harbor, June 6$^{\text{th}}$ 1850 – June 19$^{\text{th}}$ 1852, with median journey times for sail voyages and, where these routes operated, for steam voyages. (**B**) Journey time, (**C**) passenger number and (**D**) number of voyages by origin port and by ship technology. Data from Louis J. Rasmussen, *San Francisco Passenger Lists* [25]–[27].

Taken together, the speed, passenger numbers, and frequency of steam travel all indicate that this technology carried a substantially elevated risk of pathogen introduction. Indeed, Rasmussen's notes offer frequent evidence of disease on steamers, in particular on the route from Panama. One ship travelling this route, the *SS Panama,* arrived in San Francisco on June 1, 1851 having experienced "a good deal of sickness among the passengers during the voyage, principally fever and dysentery". Eleven days later a second steamer, the *SS Gold Hunter* arrived with 163 passengers and one case of smallpox [26, pp. 153, 158].

To assess differences in pathogen introduction risk along each route in more detail, we simulate introduction risk for influenza, measles, and smallpox across the full range of ship populations and journey times represented in Rasmussen's San Francisco arrivals dataset (Figure 4A). Here, contours represent pathogen introduction risk by journey time and by total ship population, N, assuming 5% population-level susceptibility, $\beta_{dd} = 0.05$, and β_{fd} calculated according to standard literature values of R_0 (Table A1). We overplot San Francisco journey statistics to assess risk of pathogen transport across each route (Figure 4A). For visual clarity, we plot each route on a single panel. However, the observations below concern introduction risk for all pathogens across all routes travelled.

Influenza's low R_0 and extremely fast generation period present a very low risk of introduction into San Francisco from any origin ports except Oregon and perhaps Panama or Hawai'i. Even then, only the fastest voyages presented any substantial chance of pathogen transport. Had a person with influenza been present on board the *Columbia* steam ship (3 days, 74 passengers) at its time of departure from Oregon, we estimate a 74% risk of introduction into San Francisco (Table 1). By contrast, we estimate just a 1% risk for the Oregon *Tarquina* sail ship (7 days, 11 passengers), a 0.8% risk for the Panama *Columbus* steam ship (21 days, 225 passengers), and a negligible risk ($< 0.1\%$) for sail travel from Panama.

Measles, which has longer latent and infectious periods, presents a moderate introduction risk for all journeys $\lesssim 25$ days in duration (Figure 4A) – consistent with the single-generation outbreak duration range for this pathogen (Figure 1E). This $\lesssim$ 25-day range includes the

vast majority of journeys from Oregon (by steam or sail), Hawai'i (by steam or sail), and Panama (by steam). Additionally, we estimate substantially longer pathogen survival on board ships with large populations. Had the *Iowa* sail ship (377 people) departed Hong Kong with a measles patient on board, we estimate a 56% chance of introduction despite the ship's 54-day journey (Table 1). Similarly, had the *Golden Gate* steam ship (66 days, 458 passengers) departed New York City with one infected passenger, we estimate a 60% introduction risk.

Smallpox has substantially longer generation period than either measles or influenza ($\mu_E = 12$ days; $\mu_I = 17.5$ days). Consequently, journeys of $\lesssim 40$ days present a moderate introduction risk at any ship population size, expanding the range described for measles to include several sail ships originating in Panama and Valparaíso. As before, we estimate longer survival ranges on board ships with larger populations (Table 1). However since smallpox is less transmissible than measles, ships require larger population sizes to achieve a equivalent $R_e(0)$. Hence, in some cases highly-populated ships present a lower risk of introducing smallpox than they did measles: for instance, had the *Golden Gate* departed with one infected patient on board, we estimate a 45% risk of smallpox introduction (Table 1).

Finally, we use these analyses to inform the plausibility of ship-borne pathogen transfer across a wider range of historical contexts (Figure 4B). In particular, we explore hypothetical pathogen introduction risks from a selection of historical voyages, chosen to reflect the range of shipping routes, technologies and practices between the 15th and 20th centuries. These are: Christopher Columbus's First Voyage on the *Santa María* from Spain to the present-day Bahamas (1492); John Cabot's voyage on the *Matthew*, which sailed from England to a disputed location in northeastern North America, possibly present-day Newfoundland, Nova Scotia, or Maine (1497-98); the *Sea Venture* emigrant ship, which set out from England for Roanoke but which instead accidentally "discovered" present-day Bermuda; the *Mayflower* emigrant ship from England to Massachusetts (1620); the Portuguese slave trade ship *Diana*,

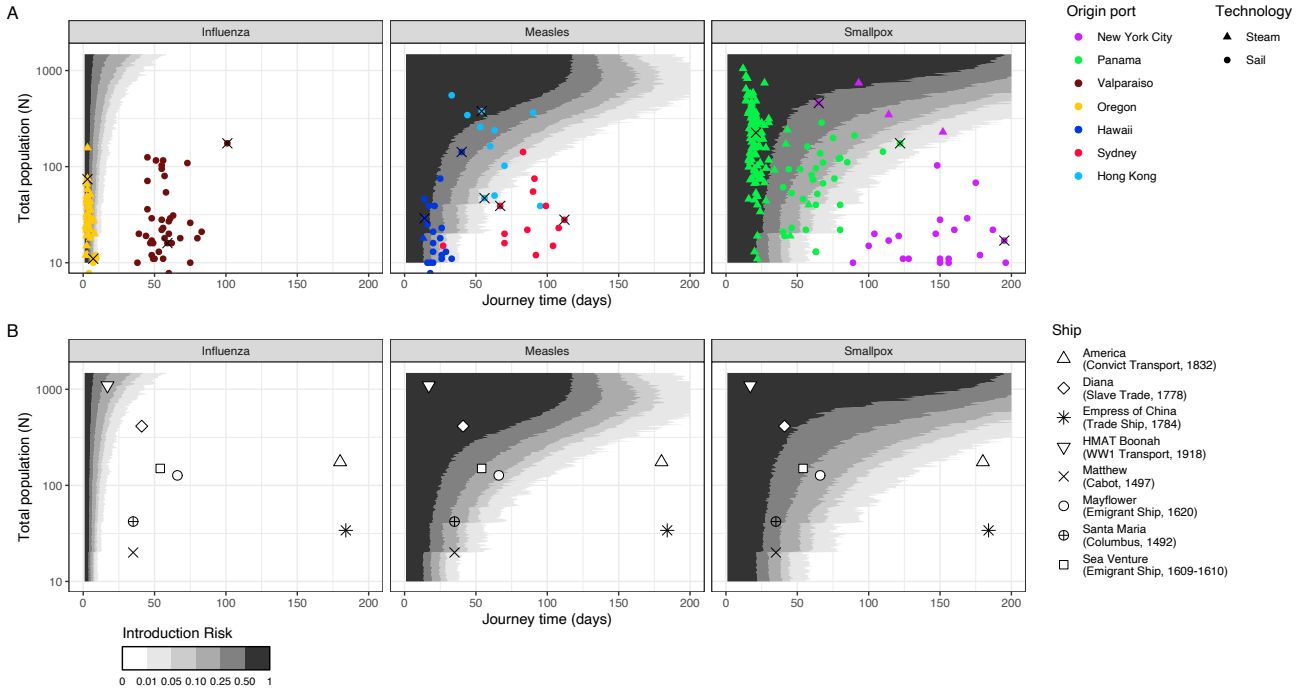

Figure 1.4: **Historical Applications.** Introduction risk for influenza, measles, and smallpox by journey time and by total ship population, N, assuming 5% initial population-level susceptibility, intermediate density dependence ($q = 0.5$), $\beta_{dd} = 0.05$, and μ_E, μ_I, and β_{fd} according to consensus natural history parameters (Table A1), with β_{fd} back-calculated as $1/\mu_I$ times a pathogen's typical R_0 on land. (**A**) overplots data on San Francisco Port arrivals, June 1850 – June 1852. Here, total population (N) represents only the passengers on board each ship, as crew data is not available. Introduction risk for all three pathogens are shown for selected voyages in Table 1; these voyages are indicated with black crosses. (**B**) overplots selected historical journeys, 1492–1918, chosen to be indicative of the broad trends in transoceanic shipping. N represents the combined totals of passengers and crew. Sources and further data are available in Table A3. Numerical introduction risk estimates for (**B**) are provided in Table 2.

which transported 413 crew and enslaved people from Îles de Los, off the coast of West Africa, to Curaçao, in the Caribbean (1778); the outward leg of the *Empress of China*, first United States trade ship to sail to China (New York to present-day Macao, 1783); the British convict ship *America*, which transported 175 British crew and convicts, likely from the United Kingdom to Australia (1832); and the Australian WW1 military transport ship *HMAT Boonah*, which carried 1,095 crew and troops from South Africa to Australia (1918) (Table A3).

For these analyses we likewise assume a 5% rate of susceptibility, although in practice we expect this rate to have varied significantly. For example, emigrant ships likely transported more children than other forms of shipping, who we would expect to exhibit greater pathogen susceptibility than older passengers.

Under these assumptions, early transatlantic voyages of exploration could plausibly have introduced measles or smallpox to their ports of arrival (Figure 4B, Table 2). We estimate a 21% chance of smallpox introduction and 35.5% chance of measles introduction had the *Santa María* (35 days, 42 people) departed with a case on board, and a 12% and 30% introduction risk respectively on the *Matthew* (35 days, 20 people). However introduction risk for both pathogens substantially increased substantially on the transatlantic slave trade ship *Diana*, which carried 448 enslaved people and crew: 64% risk for measles and 58% risk for smallpox, had one person been infected at the time of departure.

Meanwhile, the lengthy journey times of the *Empress of China* trade ship (190 days) and the *America* convict ship (180 days) suggest a compelling explanation for the substantially later introduction of smallpox and measles to these regions. Even with > 100 passengers, a voyage such as the *America*'s is outside the range of plausible influenza and measles introduction, and extremely unlikely (0.1% risk) to introduce smallpox.

Our analysis suggests that by far the greatest introduction risk of smallpox and measles – and the only plausible influenza introduction – comes from fast, highly-populated ships such as the WW1 troop ship HMAT *Boonah* (17 days, 1,095 passengers and crew). Had this

ship departed South Africa with an infected person on board, it would have had a 21% risk of introducting influenza and an 89% chance of introducing either measles or smallpox into its destination port in Australia. Thus, this combination of fast transit and extremely large passenger sizes substantially increased both the magnitude of introduction risk for moderately fast-burning pathogens (measles and smallpox) and expanded the range of potential introduction to include pathogens (e.g. influenza) with much faster life cycles.

1.3 Discussion

Canonically, transoceanic pathogen transfer has been a story of colonial exploration. Our analysis indicates that introductions of smallpox and, to a lesser extent, measles from early transatlantic voyages of discovery were plausible, though by no means guaranteed. Depending on weather, these journeys could last just 5-10 weeks — a reasonable time frame for these pathogens to persist on board a ship [34]. However, early introductions are not plausible for faster-burning pathogens such as influenza, nor for longer-running journeys beyond the Atlantic Ocean. Moreover, in the absence of high crew susceptibility, it is not clear how easily smaller ship populations such as the *Matthew* (with a crew of 18) or the *Santa María* (with a crew of 42), could have reached the supercritical values of $R_e(0)$ necessary for measles outbreaks longer than around 30 days and smallpox outbreak duration greater than around 50 days.

More recently, transoceanic pathogen transfer has become a story of technological innovation. Our work supports Cliff and Haggett [21]'s argument that steam technology transformed shipborne pathogen introduction risk by dramatically reducing journey times. Yet we contend that lower transit times were only part of the ecological transformations initiated by steam travel. In Gold Rush-era San Francisco, steam vessels carried the greatest risk of pathogen introduction not simply because they were fast, but because they were more reliable, made more frequent journeys, and carried a greater number of passengers. Steam

Table 1.1: Influenza, Smallpox, and Measles introduction risk across selected voyages into San Francisco, 1850–1852

						Pathogen introduction probability		
Route	Vessel	Date	Type	Duration	*N*	Influenza	Measles	Smallpox
New York City	*Elsinore*	8 Jun 1851	Sail	195 days	17	<0.001	<0.001	<0.001
New York City	*Golden Gate*	19 Nov 1851	Steam	66 days	458	<0.001	0.601	0.459
Valparaíso	*Aurora*	17 May 1851	Sail	59 days	16	<0.001	<0.001	0.006
Valparaíso	*Huntress*	2 May 1852	Sail	101 days	175	<0.001	0.032	0.083
Panama	Columbus	6 Aug 1850	Steam	21 days	225	0.008	0.679	0.741
Panama	*Sarah and Eliza*	29 Aug 1850	Sail	122 days	175	<0.001	0.007	0.030
Oregon	*Columbia*	30 Nov 1851	Steam	3 days	74	0.742	0.994	0.999
Oregon	*Tarquina*	28 Dec 1851	Sail	7 days	11	0.095	0.846	0.954
Hawai'i	*Edgar*	21 Aug 1851	Sail	40 days	142	<0.001	0.387	0.403
Hawai'i	*Baltimore*	27 Dec 1851	Sail	14 days	29	0.006	0.633	0.851
Sydney	*Cameo*	5 May 1851	Sail	112 days	28	<0.001	<0.001	<0.001
Sydney	*Walter Claxton*	12 Feb 1852	Sail	67 days	39	<0.001	0.002	0.025
Hong Kong	*George Pollock*	31 Jul 1850	Sail	56 days	47	<0.001	0.029	0.124
Hong Kong	*Iowa*	9 Jun 1852	Sail	54 days	377	<0.001	0.566	0.472

Table 1.2: Influenza, Measles, and Smallpox Introduction Risk across Selected Historical Voyages, 1492–1918

				Pathogen Introduction Risk		
Year	Vessel	Duration	N	Influenza	Measles	Smallpox
1492	*Santa María*	35 days	42	<0.001	0.210	0.353
1497-98	*Matthew*	35 days	20	<0.001	0.118	0.301
1609	*Sea Venture*	54 days	150	<0.001	0.247	0.292
1620	*Mayflower*	66 days	127	<0.001	0.115	0.187
1778	*Diana*	41 days	413	<0.001	0.638	0.582
1784	*Empress of China*	184 days	34	<0.001	<0.001	<0.001
1832	*America*	180 days	175	<0.001	<0.001	0.001
1918	HMAT *Boonah*	17 days	1095	0.212	0.890	0.889

travel was indeed a phase transition in global pathogen circulation – but this story is likely more complicated than previous tellings suggest.

Moreover, steam travel was not the only revolutionary force in transoceanic pathogen transfer. Our study suggests that shipping practices involving large-scale movement of people substantially increased the risk of pathogen transfer, even under sail. In 1852, two sail ships from Hong Kong, the *Catalpa* and the *Iowa*, displayed similar transit times into San Francisco: 60 days and 54 days, respectively [25, p. 89] [26, p. 176]. Yet while the *Catalpa* carried "Chinese merchandise, rice, cordage, and assorted goods" – along with one solitary passenger – the *Iowa* brought "377 unidentified in steerage", likely, Chinese people bound for California's gold fields [35]. As our analyses show, the presence of 377 people on board would have substantially increased the *Iowa*'s capacity for sustained pathogen circulation — especially if, as seems likely, these emigrants suffered from crowded shipboard conditions, and were more vulnerable to infection. Such large-scale forms of human population movement could have transformed transoceanic pathogen circulation long before the steam travel revolution.

Our study raises several questions that require further consideration. One concerns the mechanics of shipboard transmission. Little is known concerning either the density dependence or the intensity of transmission on board historical vessels – although our results suggest that crowded ships with larger and more susceptible populations presented greater risks regardless of density dependence, and that rough introduction thresholds at any transmission intensity can be inferred from pathogen natural history (Figure 1, Figure 2). A related and more challenging question concerns the extent of population structure on board historical ships, and the degree to which this may have prolonged outbreak duration.

Second, more information is needed regarding pathogen dynamics in source populations. This matters both for inferring likely rates of population-level immunity, and for assessing the probability of at least one infected individual on board ship at the point of departure. Longitudinal mortality data exists for diseases such as smallpox and measles, especially in European and North American contexts with well-preserved time series [36], [37]. Reconstructing historical prevalence and immunity landscapes from these sources will be challenging. However, ultimately this data will be critical for reconstructing realistic rates of pathogen transfer in pre-20^{th} century contexts.

A related set of questions concern the contribution of partly-immune individuals to pathogen circulation within a given population. The ability of partly-immune people to be infected and transmit infection has long been recognized as an important force for population-level influenza [38] and smallpox [39] dynamics. Partial immunity also provides a compelling explanation for recent resurgences in mumps [40] and pertussis [41], [42]. Moreover, it is plausible that the contribution to transmission from partly-immune individuals was more significant on board a ship than it was on land, for example due to extended exposures or large infectious doses. This possibility – and the significance of partial immunity on outbreak duration more broadly – require further investigation.

Finally, our analysis specifically considers human-to-human respiratory transmission of infectious pathogens. Pathogens with significant food-, water- or fomite-borne transmis-

sion (e.g. cholera, Salmonella), with vector-borne transmission (e.g. malaria, yellow fever) with multi-species transmission (e.g. plague), and or infecting only non-human animals (e.g. rinderpest) will require modified modeling approaches and likely additional historical data. This issue is also pertinent to smallpox, for which the extent of fomite transmission is unclear. Recent research indicates that orthopoxviruses can remain viable on surfaces for weeks [43]. The World Health Organization's smallpox eradication campaign found that fomites ultimately caused only a small minority of outbreaks [39]. However, in the context of historical pathogen circulation even rare introductions can be impactful.

Our model offers a general assessment of outbreak duration in a closed population, which holds significance beyond historical systems. Pathogen persistence dynamics in discrete subpopulations are critical for understanding disease circulation in any metapopulation with slow rates of contact [44], [45]. Understanding epidemic duration in a closed population also carries present-day epidemiological importance — from anticipating outbreak length in isolated groups, to designing protocols for effective population-level quarantine and isolation.

Moreover, these findings carry important historical implications. Processes that involved frequent, large-scale movement of peoples could be highly significant in transoceanic pathogen circulation. In particular, historical developments such as the transatlantic slave trade, penal transport, voluntary emigration, and wartime troop movement potentially brought substantial transformations in transoceanic pathogen ecology, centuries before the present-day upheaval of air travel [8], [46]–[48]. This presents a rich opportunity for collaboration between ecologists and historians. How did global social, economic, technological, and military forces combine to shape the natural history of disease circulation — and with what consequences along the way for the world's people, places, and pathogens?

1.4 Materials and Methods

1.4.1 Model Description

We simulate shipboard outbreaks using a stochastic SEIR model (Appendix 3). We implement continuous-time stochastic simulations in R with the Gillespie Algorithm, using the Optimal Tau-leap method for computational efficiency [49]. All simulations assume a single index case in state E at the time of departure. We define outbreak duration as the time until both state E and state I contain zero individuals.

To achieve a more realistic depiction of the time course of infection, we make dwell times in state E and state I Erlang-distributed using the Linear Chain Trick [50]. For all simulations, we use shapes $k_E = k_I = 3$ and rates k_E/μ_E and k_I/μ_I for states E and I respectively. This technique gives a unimodal distribution with a long right hand tail, such that disease progression is relatively constrained in most individuals, but with some patients occasionally experiencing substantially longer periods of incubation or infectiousness [51]. We assume that state E is pre-symptomatic and that initially infected individuals could board ship at any point during this period, randomly assigning index cases across sub-states $E_1, E_2, ..., E_{k_E}$ at the point of departure.

Our model also tracks infection across pathogen generations. The I_n infectious individuals from generation n produce new exposed individuals E_{n+1}, which represent the $(n+1)^{\text{st}}$ generation of infections.

To account for uncertainty and variation in the density dependence of shipboard contact rates, our model does not assume either classical density dependence or classical frequency dependence. Instead, we model the shipboard transmission with the equation:

$$R_0 = \mu_I(\beta_{dd}N)^q(\beta_{fd})^{1-q}$$

R_0 is the pathogen's basic reproduction number on board a given ship. This represents the number of infections that an average infected person generates in a fully-susceptible popula-

tion over their average period of infectiousness, μ_I. The density dependence of transmission is adjusted with the parameter q, with $q = 1$ representing classical density-dependent transmission ($R_0 \propto N$), $q = 0$ representing classical frequency-dependent transmission ($R_0 \perp\!\!\!\perp N$), and $0 < q < 1$ representing intermediate density dependence ($R_0 \propto N^q$). The parameters β_{dd} and β_{fd} modulate the intensity of transmission under each density dependence pole — intuitively, the proportion (β_{dd}) and the raw number (β_{fd}) of people on board ship that a single infected individual will infect per day, on average, in a fully-susceptible population.

For these analyses we assume no formal relationship between β_{dd} and β_{fd}. Instead, we adjust each parameter explicitly to assess the effect of different combinations of q, β_{fd}, β_{dd} and N on outbreak duration. In cases where N is constant and where we do not explore the effect of density dependence, we set $q = 1$ and assume that $R_0 = \beta_{dd}N$; mathematically, this is equivalent to setting $q = 0$ with β_{fd} fixed at $\beta_{fd} = \beta_{dd}N$.

1.4.2 Historical Data

To provide real-life context on our theoretical results, we collected data on ship arrivals into the port of San Francisco between June 6 1850 and June 19 1852 from volumes I, II and III of genealogist and historian Louis. J. Rasmussen's reference book *San Francisco Ship Passenger Lists* [25]–[27]. We recorded the port of origin, the ship type, the journey time, and the number of passengers for ships originating from seven locations: Hawai'i; Hong Kong; Oregon Territory; New York City; Sydney, Australia; Valparaíso, Chile; and Liverpool, England. In the few cases where Rasmussen reports ships making multiple stops at one or more of these locations in the course of their voyage, we record both the journey time into San Francisco from a ship's port of origin and, where available, journey times into San Francisco from the intermediate port(s). We exclude ships where substantial numbers of people ($N > 10$) boarded during a voyage, as our model does not account for changes in population size subsequent to the initial point of departure.

For almost all ships, Rasmussen provides passenger numbers but not numbers of crew.

We assume that in most cases, crew (i) represented a small proportion of a ship's total population; (ii) were, as professional sailors, more likely to possess immunity to common maritime infections, and so represented an even smaller proportion of a ship's susceptible people. Thus, in the absence of crew size data, analyses considering population size of vessels arriving into San Francisco approximate N as the total number of passengers on board each ship.

Appendix

A.1 Transmission on board Historical Ships

A.1.1 Intensity

Multiple contemporary analyses indicate that transmission on board historical ships could be substantially more intense than transmission in typical land settings. Vynnycky et al. [52] estimated an R_0 of pandemic 1918 influenza of approximately 4-11 on board the troop ship His Majesty's Australian Transit (HMAT) *Boonah*, an R_0 of 5-17 on board the troop ship HMAT *Devon*, and an R_0 of 3.5-8 on board the troop ship HMAT *Medic*, which carried 1,095, 1,096, and 989 people respectively. By contrast, the same analysis estimated an R_0 of roughly 1.5-4 in American and Scandinavian towns and cities. Meanwhile, White and Pagano [53] calculated an R_0 of roughly 4.98 for the same epidemics on board HMAT *Boonah* and HMAT *Medic*, compared with an R_0 of 1.34-3.21 in Maryland communities.

Similar analyses are not available for earlier time periods. However qualitative descriptions likewise indicate that conditions on board pre-1918 ships were conducive to intense pathogen transmission. One 1801 newspaper report describes an emigrant ship from Ireland to New York City so crowded that "the space between decks, occupied by nearly 300 persons, became the receptacle of all excremental matters." [54] Half a century later, English author Frank Marryat recalled travelling from Panama to San Francisco on an unnamed vessel "so crowded with passengers, that it was not until it was ascertained that there was scarcely standing-room for those on board that she tripped her anchor." [55, p. 424] Longer excerpts from both texts, printed below, offer vivid depictions of the conditions that passengers suffered during these voyages.

In addition to crowding, pre-1918 ships also faced a substantial ventilation challenges. On sail ships, opening portholes risked losing heat and letting in water. Thus, below-deck spaces were sealed, resulting in notoriously poor air quality [56], [57]. The steam revolution somewhat improved onboard environments, both by providing a source of heat and in, some cases, powering active ventilation systems. Yet steam ventilation still failed – sometimes catastrophically. In 1848, seventy-three people died from suffocation on board the *Londonderry* emigrant steamer when hatches were closed during a storm [57, p. 185]. Six years later, fifty passengers out of a total of 204 died by suffocation in an unnamed ship carrying emigrants from Mauritius [58, p. 18]. "Under no circumstance can a ship of any kind be made as healthful or as comfortable as the house on shore," wrote United States Navy surgeon Albert L. Gihon in 1886. Gihon added: "It is, practically, a floating box sealed against the admission of water, and, of course, also of air." [59, pp. 767–768]

Even with the present-day benefits of modern hygiene, sophisticated air circulation, and more highly-regulated living conditions, infectious disease outbreaks are common on board contemporary cruise [60]–[62], cargo [63] and naval ships [64]. Attack rates are often high. On a 1996 United States guided missile cruiser, Earhart et al. [65] observed a 42% attack rate of H3N2 influenza across more than 500 sailors, despite 95% vaccination coverage. Brotherton et al. [66] reported a 37% attack rate of influenza-like illness on a cruise ship carrying over 1600 passengers and crew in the year 2000. On an unidentified Peruvian Navy ship in 2009, Vera et al. [67] reported a 49.1% attack rate of H1N1 pandemic influenza across 355 passenger, with greatest risk in cadets assigned high density living quarters.

A.1.2 Density Dependence

The extent of density dependence on board historical ships is unclear and unstudied. On the one hand, contemporary military studies have demonstrated a clear and intuitive link between population density and rates of respiratory infection. In a study of Fort Humphreys, Virginia in 1918, Brewer [68] documented higher rates of pandemic influenza in military

units training in more crowded environments. In 2008, Broderick et al. [69] showed that risk of febrile respiratory infection was higher in San Diego Marine Corps units with greater population sizes.

Some historical observers clearly thought of ship transmission in what we would today consider density-dependent terms. In a report on 1918 pandemic influenza on board British Royal Navy ships, surgeon-commander Sheldon F. Dudley argued that "infective material must become so dense and diffused as to saturate the ship. That is to say, everyone on board receives a dose of the specific agent sufficient to cause cause influenza." [70, p. 45] Dudley's report is excerpted below.

Yet density dependence does not appear to describe every instance of shipboard transmission. In an analysis of smallpox outbreaks on board vessels bound to Australia, 1850-1908, quarantine director J. H. Cumpston found that most shipboard smallpox transmission was limited to close contacts of infected patients, for example family members, close colleagues, or those sharing beds and cabins [71, pp. 114–115]. Such a pattern typically argues for "frequency-dependent" transmission, in which an infected individual on average transmits only to a fixed number of close contacts, regardless of a ship's total population size.

A.2 Qualitative Descriptions of Infection and Transmission on board Historical Ships, 1801-1921

Three written descriptions of disease transmission on board nineteenth- and early twentieth-century ships are excerpted below. We do not intend these extracts to give a comprehensive view of shipboard transmission, but rather to offer a qualitative view of possible transmission scenarios. All spelling and grammar is original.

A.2.1 "Another instance of pestilence engendered in a ship crowded with passengers from Ireland." [54, pp. 234–235]

The ship Nancy, Capt. John Herron, was charterd by a commercial house at Sligo, to carry passengers from that port to New-York. She sailed from Sligo on the 12th July, 1801, and arrived, after a passage of 77 days, at the port of New York, on the 27th of September following. This ship, of the burthen of 202 tons, received on board 417 passengers, and was navigated by nine seamen. The provisions, mere refuse, put up by government-contractors with the view of saving expense, were of the worst kind: and the water, which was also of bad quality, from the unexpected length of the voyage, became extremely scanty before the arrival of the ship.

In order to receive so great a number of passengers on board of this ship, temporary cabbins [sic] were built on the quarter-deck, which were filled with eighty persons. Three hundred were crowded into the space between decks.

It will excite no surprise that a vessel thus crowded became sickly soon after sailing from Sligo. Typhous fever and dysentery began to prevail, and destroyed the lives of a large proportion of the passengers.

In addition to the wretchedness of being confined in so small a space, these unhappy emigrants suffered all the evils which their habits of uncleanliness could produce. Their

bodies and clothes, covered and saturated with filth, exhuded [sic] poison all around them. Partly from the want of strength and assistance among the sick, and partly from the want of a sense of decency, the space between decks, occupied by nearly 300 persons, became the receptacle of all excremental matters, insomuch that they issued in streams from the scuppers. The filth on the upper deck was nearly over the shoes. The sides of the ship were daubed and incrusted [sic] with excrements; and even the rope for the support of such that wished to go on board were unfit to be handled. The stench was intolerably offensive.

In such condition arrived this unfortunate vessel at the place assigned for quarantine in the port of New York. Ninety persons had died on the passage; one hundred and eighty were sick. Scarcely a healthy countenance was to be seen on board of the ship; very few had escaped disease; and many had suffered from three to four relapses. About forty were taken ill after their arrival.

As soon as possible after their arrival the sick were brought ashore; stropped of their filthy and pestilential clothes; their bodies thoroughly washed and scoured with soap and water; and then wrapped up in clean blankets, and carried into the wards appointed for their reception in the Marine Hospital. The permanent buildings of the establishment were insufficient to receive so great a number; tents, and other temporary accommodations, were provided for the remainder. Separation, ventilation, and cleanliness, as soon as they could be brought into action, accomplished every thing that could be expected. And only twenty-six have died since their arrival at this port.

A.2.2 Frank Marryat's description of a voyage from Panama to San Francisco, 1851 [55, p. 424]

It seemed that we had brought the yellow fever with us to Panama, or rather it appeared at the time of our arrival, and it was now spreading with great rapidity. Cholera also broke out, and deaths from one or the other of these causes became very numerous.

The people being panic-struck, a great rush was made for the Californian boats, of which there happened, at this time, to be very few.

So soon as I was able to move, there was but one small screw steamer in port, and as the place was daily becoming more unhealthy, I secured, by great favour, a cabin in her.

Nothing could excuse the state in which this ship was put to sea, not even the panic; for she was not only ill-found in every respect, but was so crowded with passengers, that it was not until it was ascertained that there was scarcely standing-room for those on board that she tripped her anchor.

I had secured a dog-hole of a cabin, and was no sooner on board than my wife, worn out by fatigue and anxiety, was attacked by violent fever. There were two young doctors on board, but both were attacked shortly after we started. Then the epidemic (an aggravated intermittent fever) broke out among the passengers, who – crowded in the hold as thick as blacks in a slaver – gave way to fear, and could not be moved from the lower deck, and so lay weltering in their filth.

A.2.3 Sheldon F. Dudley's report of 1918 pandemic influenza on Royal Navy vessels, 1921 [70, pp. 45–46]

The density of susceptible persons in a ship must be very great as compared with an assemblage of susceptible persons on shore. If, for example, we contrast the sleeping accommodation in a ship with that of a big institution, we find that in the ship hammock hooks are less than 2 ft. apart, whereas institution bed centres are rarely less than 8ft. apart. Even when head to toe slinging is insisted on in a ship the men's heads must often be within 3ft. of each other. Now the volume of spray from a mouth at 3ft. is nearly twenty times that at 8ft. Therefore how much more readily will the man sleeping in a battleship's mess-deck get a requisite dose of infectious material than the man sleeping in an institution ashore? Again we also hear of many men in a ship not ill enough to go off duty, and, thus being

immobilized, wandering about among their fellows.

When we consider these points, and at the same time realize that a modern battleship, with its tiers of lumbered decks, its cramped accommodation, and its crew of often over 1,000 men, covers an area of less than one-fiftieth of a square mile, I do not think it possible to doubt the infective material must become so dense and diffused as to saturate the ship. That is to say, everyone on board receives a dose of the specific agent sufficient to cause influenza, unless he happens to be highly immune at that time to the strain of organism responsible for the epidemic. In a ship, the density of susceptible persons, the mass of infection, and the local migration are all so great that any diminution in one or more of these factors that may be produced by the use of sprays and gargles, by early isolation of cases and disinfection, is scarcely likely to diminish the rate of spread in a ship, once influenza has obtained a footing on board. And I think naval experience, where all these preventive measures have been vigorously employed, justifies this pessimism, as I have been unable to learn of any definite cases in which they did any good. In ships the outbreaks lasted ten days to three weeks; ashore the wave took about three months to pass over a locality. The longer wave period ashore was probably due to the lesser density of susceptible persons and infective sources, more time being required for the infection to hunt out all the susceptible individuals within its reach.

A.3 Model Equations

To achieve a more realistic depiction of the time course of infection, we make dwell times in state E and state I Erlang-distributed using the Linear Chain Trick [50]. This technique gives a unimodal distribution with a long right hand tail, such that disease progression is relatively constrained in most individuals, but with some patients occasionally experiencing substantially longer periods of incubation or infectiousness [51].

Individuals progress through k_E exposed states, $E_1, E_2, ..., E_{k_E}$, and through k_I infectious states, $I_1, I_2, ..., I_{k_I}$. We use $k_E = k_I = 3$ for all simulations.

The rate of progression from state E_e to state E_{e+1} and from state E_{k_E} to state I_1 is $\frac{k_E}{\mu_E}$, where μ_E represents the mean length of time that an individual spends in all exposed states. Similarly, the rate of progression from state I_i to state I_{i+1} and from state I_{k_I} to state R is is $\frac{k_I}{\mu_I}$, where μ_I represents the mean length of time that an individual spends in all infectious states.

We track infection across $g > 1$ transmission generations, where $E_{n,e}$ and $I_{n,i}$ denote n^{th}-generation individuals in states E_e and in states I_i respectively. The $\sum_{i=1}^{k_I} I_{n,i}$ infectious individuals from generation n produce new exposed individuals $E_{n+1,1}$, which represent the $(n+1)^{\text{st}}$ generation of infections.

The number of first-generation individuals, $n = 1$, is fixed at $t = 0$. For all simulations, we assume a single first-generation individual. We randomly assign this person to a state from $E_{1,1}, E_{1,2}, ...E_{1,k_E}$ at the time of departure, $t = 0$.

To account for uncertainty and variation in the density dependence of shipboard contact rates, our model does not assume either classical density dependence or classical frequency dependence. Instead, we model the shipboard transmission with the equations:

$$\frac{dS}{dt} = -\beta_{fd}^{1-q}(\beta_{dd}N)^q S \sum_{n=1}^{g}\sum_{i=1}^{k_I}\frac{I_{n,i}}{N} \qquad\qquad 0 \leq q \leq 1$$

Here, q represents the degree of density dependence on board the ship, with $q = 0$ repre-

senting classical frequency dependence, $q = 1$ representing classical density dependence, and $0 < q < 1$ representing intermediate modes of transmission. The parameters β_{dd} and β_{fd} modulate the intensity of transmission under each density dependence pole — intuitively, the proportion (β_{dd}) and the raw number (β_{fd}) of people on board ship that a single infected individual will infect per day, on average, in a fully-susceptible population. Since susceptible people can be infected by infectious people in any generation and at any stage of infection, $\frac{dS}{dt}$ is a product of the total number of infected people across all g transmission generations and all k_I infectious states.

The following equations show the deterministic analogue of our model. We implement continuous stochastic simulations in R using the Gillespie Algorithm, using the Optimal Tau-leap method for computational efficiency [49].

$$
\begin{aligned}
&\frac{dS}{dt} = -\beta_{fd}^{1-q}(\beta_{dd}N)^q S \sum_{m=1}^{g}\sum_{j=1}^{k_I}\frac{I_{m,j}}{N} && \\
&\frac{dE_{1,1}}{dt} = -\frac{k_E}{\mu_E}E_{1,1} && \\
&\frac{dE_{n,1}}{dt} = \beta_{fd}^{1-q}(\beta_{dd}N)^q S \sum_{j=1}^{k_I}\frac{I_{n-1,j}}{N} - \frac{k_E}{\mu_E}E_{n,1} && 2 \le n \le g \\
&\frac{dE_{n,e}}{dt} = \frac{k_E}{\mu_E}(E_{n,e-1} - E_{n,e}) && 1 < n \le g;\ 2 \le e \le k_E \\
&\frac{dI_{n,1}}{dt} = \frac{k_E}{\mu_E}E_{n,k_E} - \frac{k_I}{\mu_I}I_{n,1} && 1 \le n \le g \\
&\frac{dI_{n,i}}{dt} = \frac{k_I}{\mu_I}(I_{n,i-1} - I_{n,i}) && 1 \le n \le g;\ 1 < i \le k_I \\
&\frac{dR}{dt} = \frac{k_I}{\mu_I}\sum_{m=1}^{g} I_{m,k_I} &&
\end{aligned}
$$

A.4 Supplementary Tables

Table A.1: Natural History Parameters

Pathogen	Mean latent period, μ_E	Mean infectious period, μ_I	Typical Land R_0	$\beta_{fd}^{\dagger}$	Reference
Influenza	2 days	3 days	1.5	0.5	[72]–[74]
Measles	12 days	8 days	15	1.875	[72], [75]
Smallpox	12 days	17.5 days	7	0.4	[76]–[78]

† Calculated as $\beta_{fd} = \frac{R_0}{\mu_I}$

Table A.2: San Francisco Port Arrivals Statistics, 6 June 1850 to 19 1852

			Journey Time			Number of Passengers		
Origin	Type	n	Median	Range	Interdecile Range	Median	Range	Interdecile Range
Liverpool, England	Sail	18	189.5 days	150-300 days	162-245.5 days	4.5	1-18	2-18
New York City	Sail	92	155.5 days	89-283 days	113.1-219.8 days	5	0-160	1.1-20.0
	Steam	9	180 days	65-364 days	87.2-296 days	111	5-743	11.4-515.0
Valparaíso	Sail	98	57 days	38-190 days	45.0-74.3 days	7	0-175	1.0-41.4
Panama	Sail	39	63 days	36-122 days	43.4-83.4 days	53	1-287	4.6-164.6
	Steam	103	20 days	12-58 days	16.0-27.8 days	196	11-1050	49.8-487.2
Oregon	Sail	47	7 days	3-16 days	3.6-11.4 days	4	0-12	1.0-6.0
	Steam	45	3 days	2-8 days	2.5-5.0 days	28	3-157	12.0-55.2
Hawai'i	Sail	76	22.5 days	14-80 days	16.0-31.5 days	5	0-142	2.0-22.0
	Steam	1	13 days	–	–	18	–	–
Sydney	Sail	19	91 days	67-188 days	70.0-111.2 days	16	2-142	3.8-59.0
Hong Kong	Sail	13	60 days	33-95 days	45.8-86.0 days	163	1-553	11.0-374.6

Table A.3: Selected Historical Voyages, 1492-1918

Year	Vessel	Type	Purpose	Journey	Duration	Population	Reference
1492	*Santa María* [Columbus's First Voyage]	Sail	Exploration	San Sebastian de la Gomera, Canary Islands to San Salvador, present-day Bahamas	35 days	41-60 crew (estimates vary; we use the more recent estimate of 41)	[79]
1497-98	*Matthew* [Cabot's Second Voyage]	Sail	Exploration	Bristol, England to a disputed location in northeastern North America, possibly present-day Maine, Newfoundland, or Nova Scotia	35 days	20 crew	[80]
1609	*Sea Venture*	Sail	Emigration	Bristol, England to present-day Bermuda	54 days	150 passengers and crew	[81]
1620	*Mayflower*	Sail	Emigration	Leiden, Holland to near Cape Cod, Massachusetts	65 days	102 passengers, 20-30 crew (we assume 25 crew)	[82]
1784	*Empress of China*	Sail	Trade	New York City to Macao	184 days	34 crew	[76]
1778	*Diana*	Sail	Slave ship	Iles de Los, present-day Guinea to Curaçao	41 days	413 crew and enslaved people	[83]
1832	*America*	Sail	Convict transport	Unknown (likely United Kingdom to Australia)	180 days	175 crew and convicts	[22]
1918	HMAT *Boonah*	Steam	Troop ship	Durban, South Africa to Fremantle, Australia	17 days	164 crew, 931 troops	[84]

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