

## Article

# Research on Mathematical Modeling of Infectious Disease Spread on Cruise Ships

Guojin Wang<sup>1</sup>  and Wei Yao<sup>2,\*</sup> <sup>1</sup> School of Management, Fudan University, 220 Handan Road, Shanghai 200433, China; wanggj@fudan.edu.cn<sup>2</sup> Shanghai Key Laboratory of Acupuncture Mechanism and Acupoint Function, Shanghai Pudong Hospital, College of Biomedical Engineering, Fudan University, 220 Handan Road, Shanghai 200433, China

\* Correspondence: weiyao@fudan.edu.cn

## Abstract

Cruise ships, characterized by high density, enclosed environments and shared facilities, have become amplifiers for infectious disease outbreaks; however, predicting transmission in such settings remains challenging due to small initial case numbers. In this study, a mathematical modeling framework—including compartmental, small-world (SW), and scale-free (SF) network models—was developed to analyze the dynamics of hantavirus and norovirus transmission on recent cruise ship outbreaks. The results show that when the initial number of exposed or infected individuals is extremely small (e.g., 1–2 persons), disease spread is dominated by stochasticity, causing substantial variation between individual simulations and mean model predictions. Notably, SF networks exhibited lower transmission risk under these conditions, contradicting the theoretical expectation that when the population is large, SF networks facilitate the spread of disease. The compartmental model, while consistent with average simulation outcomes, failed to reliably predict any single outbreak event. In conclusion, for closed environments such as cruise ships with very few initial cases, stochastic variability is essential, and single-outbreak outcomes should be understood as highly contingent rather than deterministically predictable.

**Keywords:** cruise ships; infectious disease spread; compartmental models; small-world networks; scale-free networks; stochastic

## 1. Introduction

Cruise ships, often hailed as “floating sea resorts,” have become one of the fastest-growing segments of the global tourism industry in recent years. According to statistics from the Cruise Lines International Association (CLIA), the total number of ocean cruise passengers worldwide reached 34.6 million in 2024, marking a 9.3% increase from the previous year and a 16.7% rise compared to the pre-pandemic levels of 2019. This figure is expected to reach 39.5 million by 2028 [1,2]. However, behind this rapid growth, the unique physical environment and operational model of cruise ships also render them “amplifiers” for the spread of infectious diseases. A cruise ship represents a typical setting where people gather in a high-density, enclosed environment for an extended period of time. A modern cruise ship typically carries hundreds or even thousands of passengers and crew members, who live together in a confined space for periods ranging from several days to several weeks, sharing public facilities such as restaurants, theaters, bars, swimming pools, elevators, and corridors [3,4]. These environmental characteristics—including high population density, strong spatial connectivity, shared air and water systems, and frequent



Academic Editor: Carlo Bianca

Received: 10 June 2026

Revised: 8 July 2026

Accepted: 13 July 2026

Published: 17 July 2026

**Copyright:** © 2026 by the authors.

Licensee MDPI, Basel, Switzerland.

This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC BY\)](https://creativecommons.org/licenses/by/4.0/) license.

movement of people—create ideal conditions for the transmission of respiratory viruses (such as influenza and SARS-CoV-2), gastrointestinal pathogens (such as norovirus), and waterborne pathogens (such as Legionella) [5].

Outbreaks of infectious diseases on cruise ships not only threaten the health of individuals on board but may also spread to land-based communities through disembarking passengers and crew, thereby triggering cross-regional public health events [6]. Historically, outbreaks on cruise ships have been frequent. For instance, the COVID-19 outbreak on the cruise ship *Diamond Princess* in 2020 became a landmark event that drew global attention, with 619 passengers and crew members confirmed infected. The disease spread rapidly on board and subsequently transmitted across multiple countries [7]. Norovirus is the most common pathogen on cruise ships. The Vessel Sanitation Program (VSP) of the U.S. Centers for Disease Control and Prevention (CDC) recorded hundreds of acute gastroenteritis outbreaks between 2006 and 2019 [8]. Between March and May 2026, the global cruise industry experienced a new cluster of norovirus outbreaks, with multiple large cruise ships successively reporting mass infection events. This series of outbreaks once again highlighted the vulnerability of cruise ships, as enclosed spaces, in the spread of infectious diseases. In March 2026, a norovirus outbreak occurred on *Star Princess* [9]. In May 2026, an outbreak of norovirus infection occurred on the *Caribbean Princess*, with 160 passengers and crew members showing symptoms of acute gastroenteritis [10]. Around the same time, the British cruise ship *Ambition* arrived in Bordeaux, France, where approximately 1700 people were restricted from disembarking due to a suspected gastrointestinal illness. A nonagenarian passenger died during the journey. Norovirus was confirmed via pathogen testing at Bordeaux University Hospital [11]. In addition, between April and May 2026, a clustered outbreak of hantavirus occurred on the Dutch polar cruise ship *MV Hondius*, caused by the Andes virus (ANDV). Previously, reports of human-to-human transmission of this virus were largely limited to long-term close contact settings within families or communities. However, this cruise ship outbreak marked the first time such a transmission pattern was observed in an enclosed, high-density population environment. As of 13 May, 11 cases had been reported, including 3 deaths [12].

Traditional epidemiological investigation methods often provide only retrospective descriptive analyses, making it difficult to predict the trajectory of an outbreak, assess the effectiveness of various intervention measures, or quantify the scale of asymptomatic infections. In this context, mathematical modeling has emerged as an indispensable tool. In recent years, significant progress has been made in mathematical modeling research on the transmission of infectious diseases on cruise ships. For instance, Rocklöv et al. (2020) used data from *Diamond Princess* to estimate the transmission potential of the COVID-19 virus and evaluate the effectiveness of quarantine measures [7]. Batista et al. (2020) analyzed the role of asymptomatic infection in the transmission of COVID-19 on cruise ships [13]. Emery et al. (2020) further quantified the contribution of asymptomatic infected individuals to COVID-19 transmission [14]. De Bellis et al. (2025) reconstructed norovirus transmission chains and used a branching process model to find that the first 10% of infected individuals were responsible for 57% of secondary cases [15]. Diagnostic delay was identified as a key driver of superspreading. The study also evaluated the effectiveness of different quarantine durations. Regarding the ANDV outbreak, Cui (2026) developed a discrete-time stochastic Susceptible–Exposed–Infectious–Recovered–Deceased (SEIRD) model and applied the Ensemble Adjustment Kalman Filter (EAKF) to infer parameters from WHO-reported data [16]. For the first time, the basic reproduction number ( $R_0 = 2.76$ ) of the Andes virus in a cruise ship setting was estimated, and the presence of a large number of covertly exposed individuals was revealed.

Beyond mathematical and epidemiological descriptions, the transmission dynamics of pathogens in confined environments are ultimately governed by a complex array of biological processes that determine microbial survival, persistence, and infectivity. For enteric viruses such as norovirus, environmental stability—influenced by factors including temperature, relative humidity, surface porosity, and organic matter loading—plays a critical role in fomite-mediated transmission, as viral particles can remain infectious on contaminated surfaces for days to weeks under favorable conditions [17,18]. Andes virus is the most well-documented hantavirus with confirmed human-to-human transmission [19]. Such transmission typically requires prolonged, close contact and is thought to occur primarily via respiratory droplets; however, its transmission efficiency is far lower than that of respiratory viruses such as SARS-CoV-2 [20]. Moreover, key individual-level factors—including the infectious dose required to establish infection, the kinetics of viral shedding (both presymptomatic and asymptomatic), and host immune competence—together determine the effective transmission potential within a closed population. While the present mathematical framework does not explicitly model these biological mechanisms at the molecular or physiological scale, the parameterization of transmission rates, environmental decay coefficients, and exposure risks should be interpreted as lumped parameters that encapsulate the net effects of these underlying microbial ecological processes. Acknowledging this biological foundation not only provides a more complete scientific narrative but also helps define the regimes within which the assumptions of compartmental and network models remain physiologically defensible.

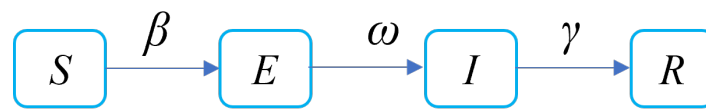
The aim of this study is to develop a mathematical modeling framework for the transmission of infectious diseases within the enclosed, high-density environment of cruise ships. This framework is used to systematically analyze the dynamic transmission characteristics of different models—namely compartmental models, small-world (SW) networks, and scale-free (SF) networks; examine two types of viruses recently transmitted on cruise ships—hantavirus and norovirus; and assess the impact of key parameters on pathogen spread. This study demonstrates that when the initial number of exposed or infected individuals is very small, disease transmission is governed by stochasticity, and the mean predictions from compartmental or network models cannot reliably forecast the outcome of a single outbreak. Under such conditions, scale-free networks may paradoxically exhibit lower transmission risk, contradicting the theoretical expectation that when the population is large, SF network facilitate disease spread.

## 2. Materials and Methods

### 2.1. Model

The Susceptible–Exposed–Infectious–Recovered (SEIR) model is one of the most used foundational frameworks in the mathematical modeling of virus transmission and is applicable to both hantavirus and norovirus spread. The model divides a population into four compartments—Susceptible ( $S$ ), Exposed ( $E$ ), Infectious ( $I$ ), and Recovered ( $R$ )—and has been widely adopted in infectious disease modeling on cruise ships. Given that hantavirus (particularly the Andes virus) is characterized by a high case fatality rate (approximately 36–50%), the standard SEIR model, in which the “Recovered ( $R$ )” compartment does not distinguish between survival recovery and death, requires extension. To address this, Cui expanded the model by introducing a Death ( $D$ ) compartment, establishing an SEIRD framework. Since neither recovered nor deceased individuals are infectious or susceptible to infection, their functional roles in the model are equivalent—except for their relevance when comparing against actual mortality data, used to estimate the death coefficient  $\delta$ . In subsequent studies, the classic SEIR model (Figure 1) was still employed, where  $R$  denotes removed individuals (i.e., Recovered + Deceased). Given the very low fatality rate of

norovirus, mortality may also be neglected. A cruise ship represents a closed system with no new individuals entering. In this model,  $\beta$  is the effective contact rate,  $\omega$  is the transfer rate from the exposed to the infected, and  $\gamma$  is the recovery rate.



**Figure 1.** The transfer diagram of the SEIR model.

The classic SEIR model assumes that individuals within each compartment are homogeneous and indistinguishable. As a result, the transitions of individuals between states (compartments) are described using differential equations (Equation (1)).

$$\begin{cases} \frac{dS(t)}{dt} = -\beta S(t)I(t) & (1a) \\ \frac{dE(t)}{dt} = \beta S(t)I(t) - \omega E(t) & (1b) \\ \frac{dI(t)}{dt} = \omega E(t) - \gamma I(t) & (1c) \\ \frac{dR(t)}{dt} = \gamma I(t) & (1d) \end{cases} \quad (1)$$

Homogeneous models can be used to analyze fundamental parameters such as the basic reproduction number ( $R_0$ ), but they fail to explain the “explosive transmission” phenomenon of norovirus. This limitation arises because individual heterogeneity within compartments means that a small number of infected individuals can cause a large number of transmissions. In contrast, complex network models—structures composed of nodes and edges—can incorporate node-specific parameters to reflect individual characteristics. In such models, disease transmission must occur along the edges connecting two nodes, unlike in compartmental models, which assume that an infected individual can transmit to any node. From a network perspective, complex network models can capture more features and thus more realistically represent the disease transmission process. Based on degree distribution, complex networks can be classified into homogeneous networks and heterogeneous networks. For homogeneous networks (where the degree distribution follows a normal or Poisson distribution), we denote the average degree of nodes as  $\langle k \rangle$ . The dynamic model for infectious disease transmission on such a network can be expressed as Equation (2).

$$\begin{cases} \frac{dS(t)}{dt} = -\langle k \rangle \beta S(t)I(t) & (2a) \\ \frac{dE(t)}{dt} = \langle k \rangle \beta S(t)I(t) - \omega E(t) & (2b) \\ \frac{dI(t)}{dt} = \omega E(t) - \gamma I(t) & (2c) \\ \frac{dR(t)}{dt} = \gamma I(t) & (2d) \end{cases} \quad (2)$$

The ordinary differential equations derived from mean-field theory for homogeneous networks are mathematically essentially the same as those of compartmental models. It can be observed that the two sets of equations differ only by the coefficient  $\langle k \rangle$ ; that is,  $\langle k \rangle \beta$  replaces the  $\beta$  epidemiological dynamics model. Therefore, conclusions drawn from epidemiological dynamics models can be directly applied to the results obtained from network models using mean-field theory.

Mean-field theory assumes that every node has the same degree, equal to the average degree  $\langle k \rangle$  [21]. However, when the degree distribution of the network takes on relatively large values—as in the case of the BA (Barabási–Albert) scale-free network—the node degree distribution follows a power law and often exhibits a fat-tail effect [22]. In this case, treating every node as having the same degree would erase the scale-free network’s characteristics and fail to capture its essential features. Therefore, for heterogeneous

networks, the equations must explicitly take the node degree distribution into account (Equation (3)):

$$\begin{cases} \frac{dS_k(t)}{dt} = -k\beta S_k(t)\theta & (3a) \\ \frac{dE_k(t)}{dt} = k\beta S_k(t)\theta - \omega E_k & (3b) \\ \frac{dI_k(t)}{dt} = \omega E_k - \gamma I_k(t) & (3c) \\ \frac{dR_k(t)}{dt} = \gamma I_k(t) & (3d) \end{cases} \quad (3)$$

where the subscript  $k$  means the degree is  $k$ , and  $\theta$  denotes the possibility of any connected node is an infective node, assuming no correlation between node degrees. Therefore,

$$\theta = \sum_j \rho_j * P(j|k) = \frac{\sum_j jP(j)\rho_j}{\sum_s sP(s)} = \frac{1}{\langle k \rangle} \sum_j jP(j)\rho_j \quad (4)$$

where  $\rho_j = \frac{I_j}{N_j}$  is the proportion of infected individuals in nodes with degree  $j$ , and  $N_j$  is the total population with degree  $j$ , that is,  $N_j = S_j + E_j + I_j + R_j$ .  $P(j)$  is the degree distribution function,  $P(j) = \frac{N_j}{N}$ , where  $N$  is the total population.

The infected compartments are  $E$  and  $I$ . An equilibrium solution with  $E = I = 0$  has the form  $x_0 = (S_{k0}, 0, 0, 0)^T = (N_k, 0, 0, 0)^T$ ; therefore,

$$\theta \approx \frac{1}{\langle k \rangle} \sum_j \frac{jP(j)I_j}{N_j} = \frac{1}{\langle k \rangle} \sum_j \frac{j}{N} I_j \quad (5)$$

## 2.2. Reproduction Number $R_0$

In the classic SEIR model, the expression for the basic reproduction number  $R_0$  is [23]

$$R_0 = \frac{\beta}{\gamma} \quad (6)$$

In a homogeneous network, since a node has an average of  $\langle k \rangle$  neighbors and the probability that a neighbor is infected at time  $t$  is given by  $I(t)$ , the probability that a susceptible individual remains susceptible after one time step is determined by the probability of not being infected by any infected neighbor, that is,  $S(t) = -S(t)[1 - (1 - \beta)^{\langle k \rangle I(t)}]$ . When  $\langle k \rangle \beta I(t) \ll 1$ , using the Newton binomial expansion and omitting higher-order terms, the change rate of the susceptible nodes is  $\langle k \rangle \beta I(t)$ . Therefore, the basic reproduction number  $R_0$  is given by

$$R_{0\langle k \rangle} = \frac{\beta \langle k \rangle}{\gamma} \quad (7)$$

In heterogeneous networks,  $k\beta S_{k0}\theta = \frac{k\beta P(k)}{\langle k \rangle} \sum_j jI_j$  is related to the structure of the network. Suppose the infection rate is  $\frac{\beta k^2 P(k)}{\langle k \rangle} \approx \frac{\beta \langle k^2 \rangle}{\langle k \rangle}$  (the average excess infection rate of the network). Then,

$$R_{0k} = \frac{\beta k^2 P(k)}{\langle k \rangle \gamma} = \frac{\beta \langle k^2 \rangle}{\gamma \langle k \rangle} \quad (8)$$

where  $\langle k^2 \rangle$  is the second moment of the degree distribution. In the BA SF network, where  $P(k) = 2m^2 k^{-3}$ ,  $m = \frac{\langle k \rangle}{2}$  [22],

$$\frac{\beta \langle k^2 \rangle}{\gamma \langle k \rangle} = \frac{\beta \sum_{k=m}^{k_{\max}} k^2 P(k)}{\gamma \sum_{k=m}^{k_{\max}} k P(k)} = \frac{\beta \ln k_{\max} - \ln m}{\gamma \left( \frac{1}{m} - \frac{1}{k_{\max}} \right)} \quad (9)$$

In the generalized SF networks,  $k_{\max} = N - 1 \rightarrow N$  [24]. Therefore,

$$R_{0k} = \frac{\beta}{\gamma} m [\ln(N - 1) - \ln m] \xrightarrow{N \rightarrow \infty} \infty$$

### 3. Numerical Simulation

#### 3.1. Numerical Simulation of Andes Virus (ANDV)

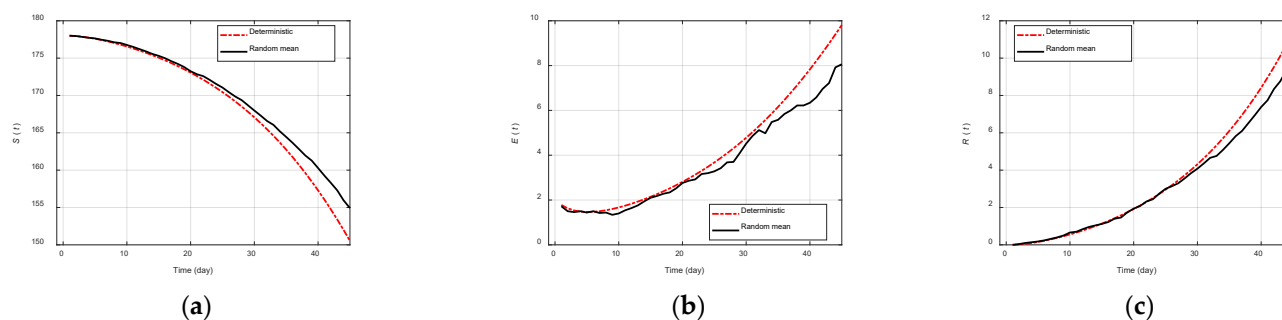
Cui referred to previous modeling studies on hantavirus, as well as publicly available reports from the World Health Organization (WHO) and the European Centre for Disease Prevention and Control (ECDC), to estimate the parameter values and ranges summarized in Table 1. At an early stage of the voyage, individuals showed no obvious symptoms; therefore, the initial number of infected individuals was assumed to be zero. According to the epidemic tracing data for ANDV, the total population  $N$  was about 180 (147 + 34). Since the first infected individual and his wife both died [25], we infer that both individuals had already been exposed prior to boarding the ship. Based on the edge weighting results obtained by Pung et al., using an exponential saturation function [26] and considering the relatively small total number of people on the cruise ship, we assume an average degree of 10.

**Table 1.** The parameters in the modeling process of ANDV.

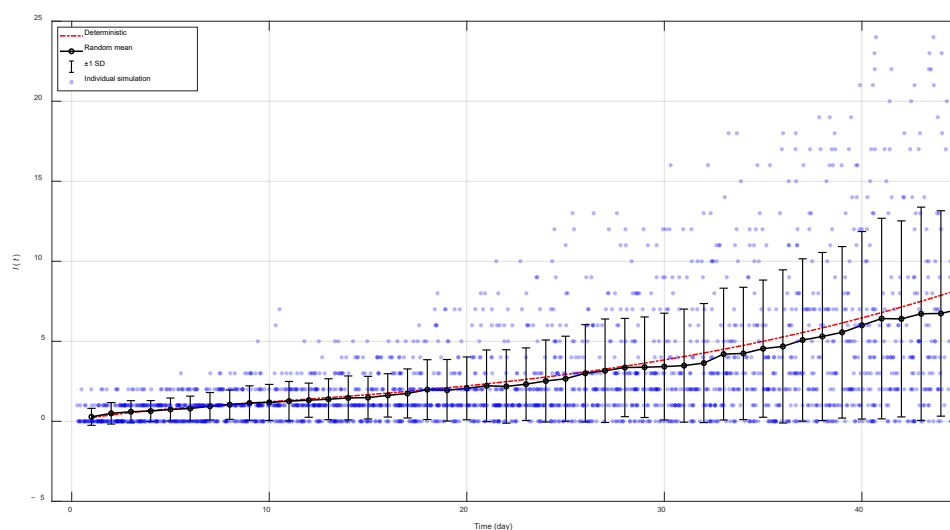
| Parameter                                          | Estimated Value | Range                                   |
|----------------------------------------------------|-----------------|-----------------------------------------|
| Transmission rate ( $\beta$ , days <sup>−1</sup> ) | 0.22 [19]       | [0.1, 0.5]                              |
| Latency period ( $1/\omega$ , days)                | 11.12 [27]      | [6, 12]                                 |
| Infectious period ( $1/\gamma$ , days)             | 5.52 [16]       | [7, 14]                                 |
| $\langle k \rangle$                                | 10 [26]         | Passenger [5.6–23.7]<br>Crew [4.4–13.5] |
| $N$                                                | 180             |                                         |
| $I(0)$                                             | 0               |                                         |
| $E(0)$                                             | 2               |                                         |

Based on the parameters in Table 1, the compartment model described by Equation (1) is solved numerically using a discrete approximation. First, the equations are iterated strictly according to a first-order difference scheme, yielding the smooth SEIR curve of the deterministic model, shown as the red dashed line in Figure 2. The results obtained from the deterministic model may contain fractional values. This becomes problematic when the total population size is not large, because the number of individuals in each compartment must be a natural number, and the infection counts exhibit random fluctuations. To address this limitation, a Poisson function is introduced to generate random integers, and the deterministic results are recovered by averaging over multiple realizations (50 times), as shown by the black solid line in Figure 2. To ensure the reproducibility of the reported results while maintaining statistical reliability, we fixed the random seed (seed = 42) when generating the numerical outcomes.

Figure 3 presents the results of each stochastic simulation. When the number of infected individuals and the total population are sufficiently large, stochastic fluctuations tend to average out. However, in this model, both the population size and number of infections are small, leading to significant discrepancies between the outcomes of a single simulation and the solution of the deterministic model. Nevertheless, the average of multiple stochastic simulations converges to the deterministic solution after a sufficiently large number of repeated experiments.

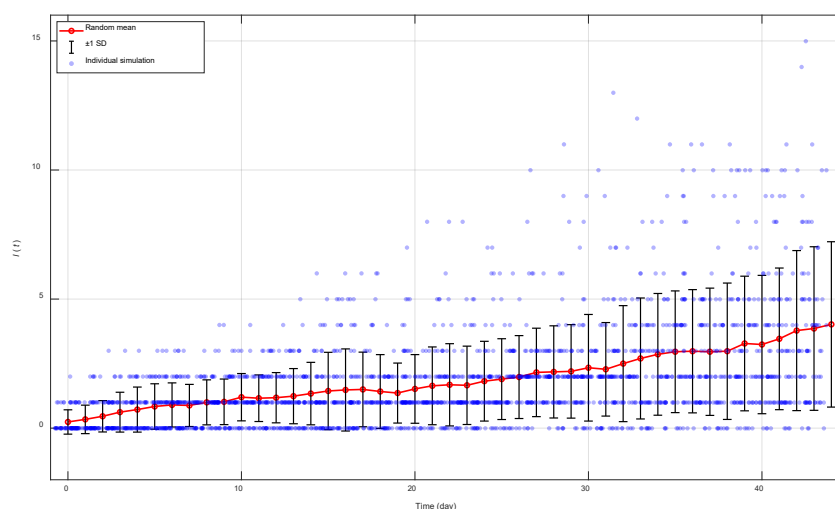


**Figure 2.** Numerical simulation results of ANDV in the compartment model. (a) Susceptible ( $S$ ), (b) Exposed ( $E$ ), (c) Infectious ( $I$ ).

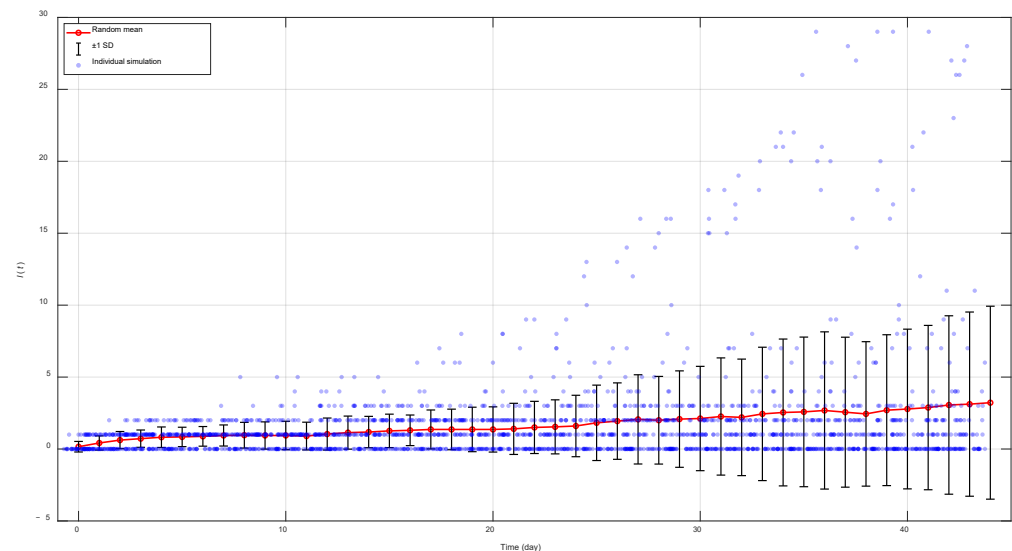


**Figure 3.** Statistical analysis of multiple numerical simulations of ANDV in compartmental models.

However, human contact is not uniformly mixed. Network models address the overly idealized assumption that “everyone interacts with everyone else” by incorporating the heterogeneity commonly observed in the real world. Figures 4 and 5 present the simulation results for a small-world network (representing a homogeneous network) and a scale-free network (representing a heterogeneous network), respectively.

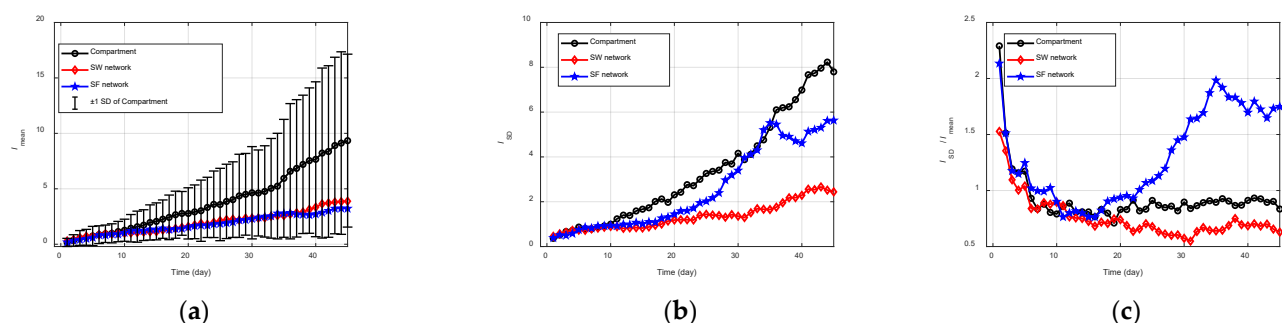


**Figure 4.** Statistical analysis of multiple numerical simulations of ANDV in small-world network.



**Figure 5.** Statistical analysis of multiple numerical simulations of ANDV in scale-free network.

Figure 6a compares the statistical results of numerical simulations from the three models. The mean infection outcomes of the scale-free network model and the small-world network model are both lower than that of the compartmental model. Figure 6a also shows that the variance of the compartmental model is relatively large. Consequently, although the three models differ in their mean values, the high dispersion in the simulation outcomes of the compartmental model, together with the pronounced fluctuations observed in individual simulation runs of all three models (Figures 3–5), means that the current statistical analysis is insufficient to demonstrate significant differences in transmission outcomes among the three models.



**Figure 6.** Comparison of simulation results of ANDV from three models: (a) mean of infected individuals  $I_{\text{mean}}$ , (b) standard deviation of the number of infected individuals  $I_{\text{SD}}$ , (c) relative standard deviation of the number of infected individuals  $I_{\text{SD}}/I_{\text{mean}}$ .

Figure 6b presents the variances of the three models, revealing that the small-world network model has the smallest variance. And to better analyze dispersion, Figure 6c shows the relative standard deviation ( $I_{\text{SD}}/I_{\text{mean}}$ ). The scale-free network model exhibits a larger  $I_{\text{SD}}/I_{\text{mean}}$  than the other two, which can be attributed to the inherent heterogeneity of scale-free networks. Additionally, Figure 5 shows that some individual simulation runs of the scale-free network model deviate significantly from the mean.

### 3.2. Numerical Simulation of Norovirus

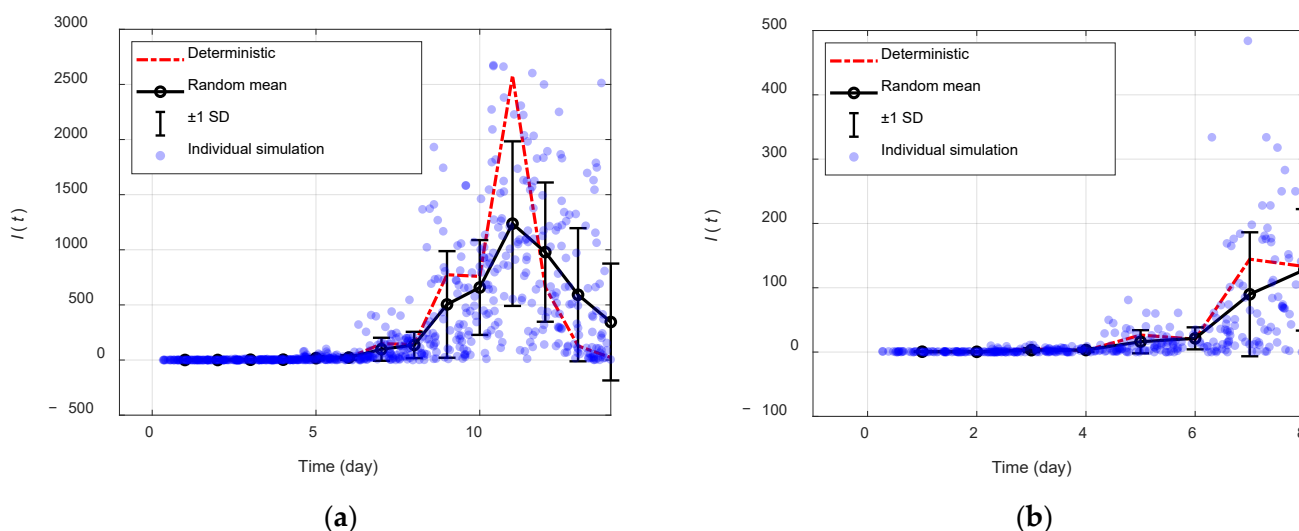
Norovirus is the primary pathogen responsible for acute gastroenteritis outbreaks on cruise ships. Towers et al. quantitatively analyzed the parameters of environmental transmission of norovirus in cruise ship settings using the mathematical models listed

in Table 2. Given the relatively large total number of passengers on cruise ships that experienced norovirus outbreaks this year, we assume an average degree of 20. Moreover, because norovirus has very short incubation and infectious periods, and only a very small number of exposed individuals are present at the time of boarding, we assume that there is initially only one exposed person.

**Table 2.** The parameters in the modeling process of norovirus.

| Parameter                              | Estimated Value | Range        |
|----------------------------------------|-----------------|--------------|
| Basic reproduction number ( $R_0$ )    | 7.2 [28]        | [6.1, 9.5]   |
| Latency period ( $1/\omega$ , days)    | 1.15 [28]       | [1.12, 1.18] |
| Infectious period ( $1/\gamma$ , days) | 1.15 [28]       | [1.05, 1.57] |
| $\langle k \rangle$                    | 20 [26]         |              |
| $N$ in the <i>Caribbean Princess</i>   | 4247 [10]       |              |
| $N$ in the <i>Ambition</i>             | 1747 [11]       |              |
| $I(0)$                                 | 0               |              |
| $E(0)$                                 | 1               |              |

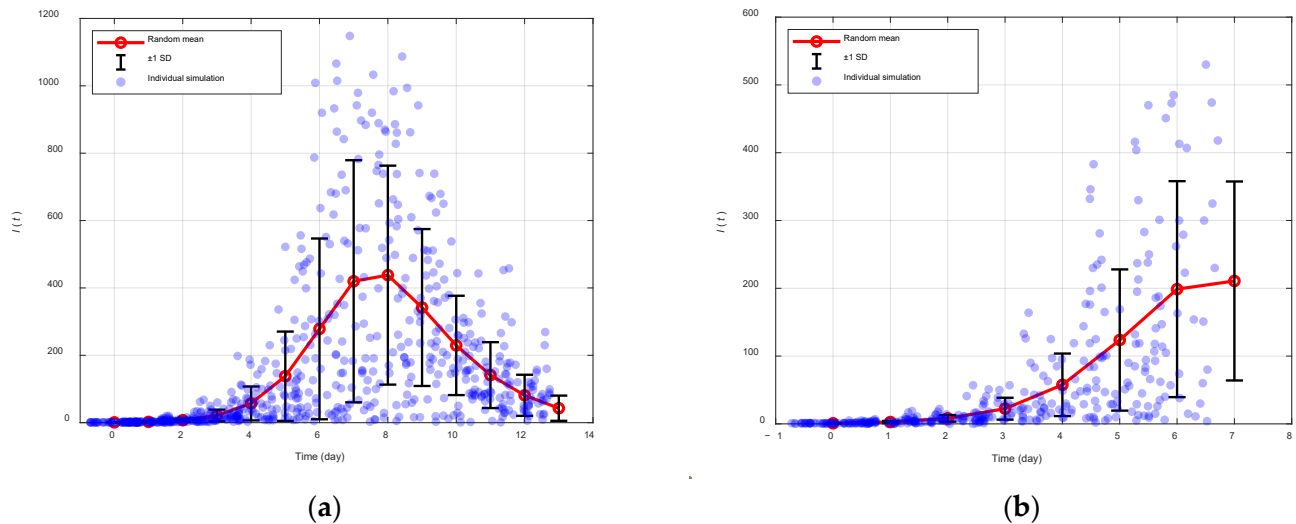
Based on the parameters in Table 2, the compartment model in Equation (1) is solved numerically using a discrete approximation. The resulting smooth SEIR curve of the deterministic model is shown as the red dashed line in Figure 7. By introducing a Poisson function to generate random integers, the outcomes of individual stochastic simulations (shown as scatter points) and the average over multiple stochastic simulation runs (shown as the black dashed line) are also presented in Figure 7. Differences between the deterministic model and the average of multiple stochastic simulations are evident, particularly in the peak region.



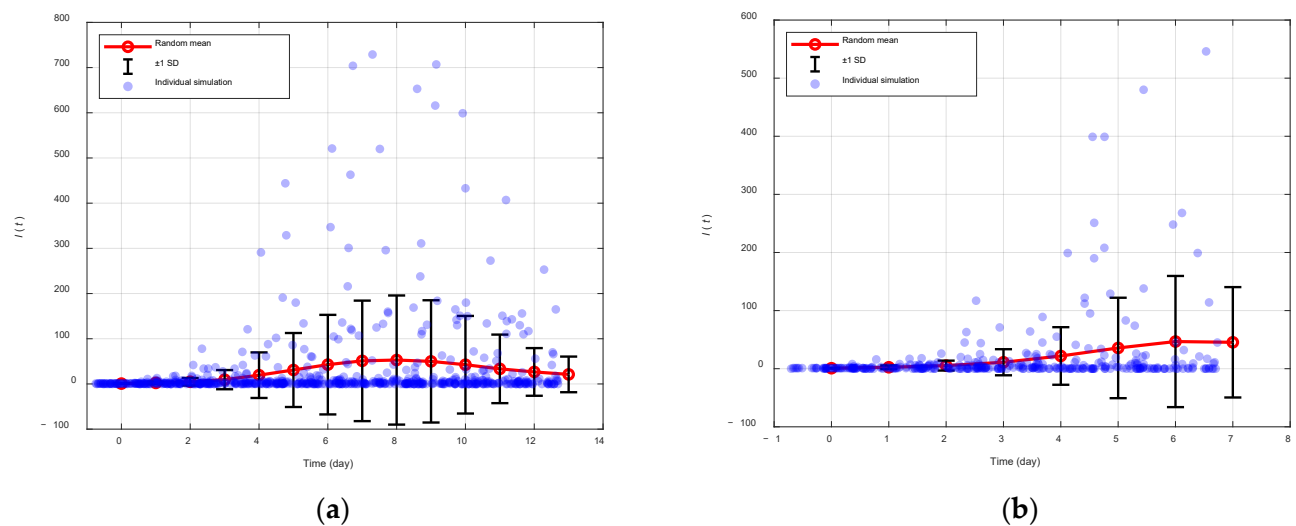
**Figure 7.** Statistical analysis of multiple numerical simulations of norovirus in compartmental models. (a) Description of cruise ship *Caribbean Princess*. (b) Description of cruise ship *Ambition*.

Figure 8 presents the simulation results for small-world networks in the *Caribbean Princess* and the *Ambition*. Both the dispersion and variance are large, exceeding twice the mean at certain points.

Figure 9 shows the simulation results for scale-free networks in the *Caribbean Princess* and the *Ambition*, where the dispersion is very large—some points exceed 10 times the mean.

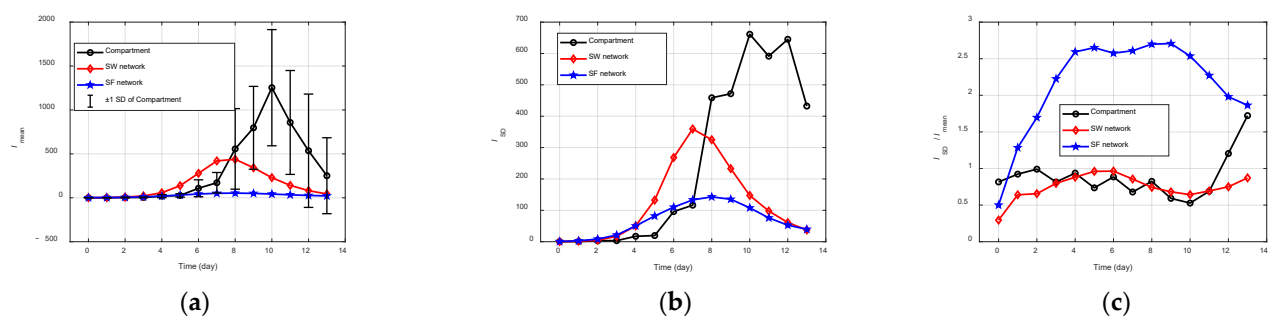


**Figure 8.** Statistical analysis of multiple numerical simulations of norovirus in small world network. (a) Description of the *Caribbean Princess*. (b) Description of the *Ambition*.

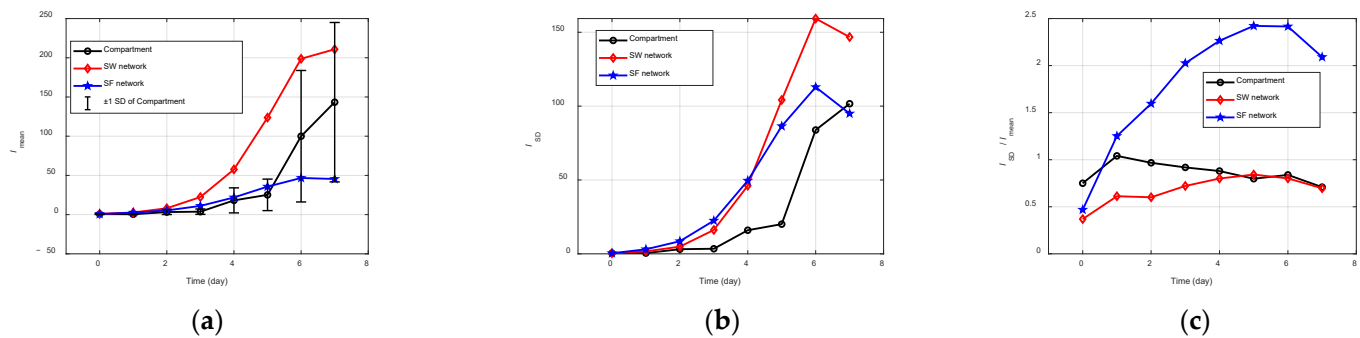


**Figure 9.** Statistical analysis of multiple numerical simulations of norovirus in scale free networks. (a) Description of the *Caribbean Princess*. (b) Description of the *Ambition*.

Figure 10 compares the statistical results of the numerical simulations for the three models applied to the *Caribbean Princess*, while Figure 11 presents the corresponding results for the *Ambition*. These results are consistent with the observations made for the MV *Hondius* (Figure 6).



**Figure 10.** Comparison of simulation results of norovirus from three models in the *Caribbean Princess*. (a) Mean of infected individuals  $I_{mean}$ , (b) standard deviation of the number of infected individuals  $I_{SD}$ , (c) relative standard deviation of the number of infected individuals  $I_{SD}/I_{mean}$ .



**Figure 11.** Comparison of simulation results of norovirus from three models in the *Ambition*. (a) Mean of infected individuals  $I_{mean}$ , (b) standard deviation of the number of infected individuals  $I_{SD}$ , (c) relative standard deviation of the number of infected individuals  $I_{SD}/I_{mean}$ .

## 4. Discussion

### 4.1. Stochastic Dominance in Low-Incidence Settings

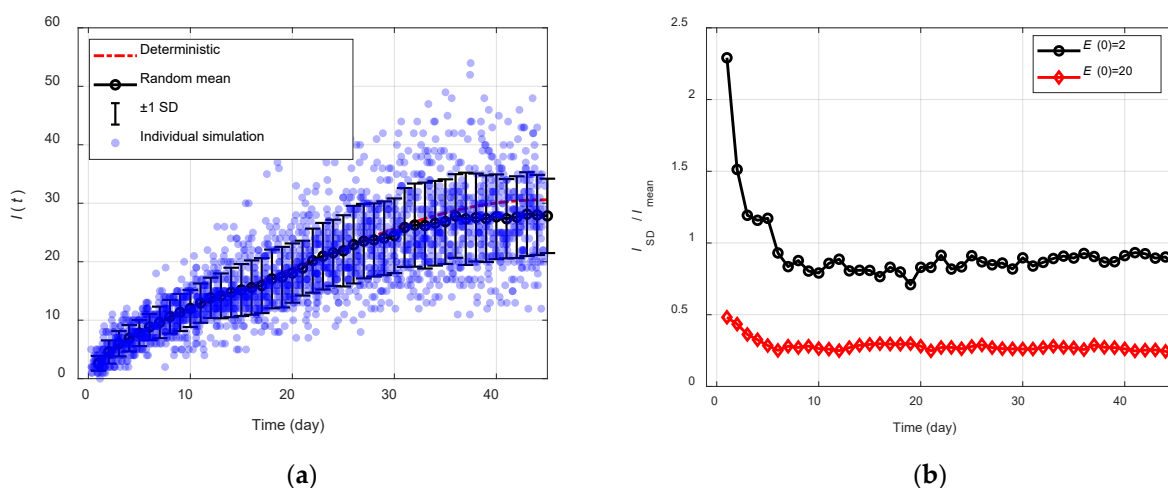
Unlike on land, passengers on a cruise ship typically travel only when they feel healthy. Simple health screenings may be conducted before boarding, and individuals with noticeable symptoms—such as fever or diarrhea—may be denied embarkation. To some extent, these measures reduce the likelihood of infected individuals boarding; therefore, the initial number of infected persons is usually small. According to case statistics, the three cruise ships discussed in this paper reported no individuals showing clear symptoms at the start of the voyage; consequently, the initial number of infected individuals is assumed to be zero. Based on epidemic tracking data, we infer that there were two initially exposed individuals on the *MV Hondius*, and one initially exposed individual on both the *Caribbean Princess* and the *Ambition*.

Due to the very small number of exposed individuals, viral transmission involves a significant element of “chance.” Figures 3 and 7 show that although the deterministic results of the compartmental model and the mean results of multiple stochastic simulations are consistent, the differences between individual simulation results and the mean are substantial. Therefore, when the initial number of infected or exposed individuals is very small, the compartmental model cannot predict the outcome of a single cruise ship disease transmission event. Although Table 3 shows that the simulation results for ANDV transmission on the *MV Hondius* are relatively close to the observed number of infected individuals at the end of the voyage  $I_{end}$  (11 cases of infection were identified, including 3 deaths; thus, the observed number of infected individuals was 8), the differences in transmission outcomes on the *Caribbean Princess* and the *Ambition* were quite large. In reality, the actual situation corresponds to only one stochastic simulation, so it is reasonable that it differs significantly from the mean simulation result.

To further quantify this stochastic dominance, we examined the effect of increasing the initial number of exposed individuals ( $E(0)$ ) in the ANDV model (Figure 12). When  $E(0)$  was increased from 2 to 20, the relative standard deviation decreased from approximately 80% to about 25%, indicating that stochastic fluctuations diminish as  $E(0)$  grows. This threshold behavior—where the system transitions from stochastic-dominated to more deterministic dynamics—suggests that early outbreak trajectories are inherently unpredictable until a critical number of infections has accumulated.

**Table 3.** Simulation results of different models.

| Cruise Ship               | Model               | $I_{\text{end}}$ | Number of $I_{\text{end}} = 0$ /Total Number |
|---------------------------|---------------------|------------------|----------------------------------------------|
| <i>MV Hondius</i>         | observed data       | 8                | -                                            |
|                           | compartmental model | 9.34             | 2%                                           |
|                           | small-world network | 3.88             | 2%                                           |
|                           | scale-free network  | 3.22             | 24%                                          |
| <i>Caribbean Princess</i> | observed data       | 160              | -                                            |
|                           | compartmental model | 251.02           | 4%                                           |
|                           | small-world network | 42.94            | 0%                                           |
|                           | scale-free network  | 21.16            | 38%                                          |
| <i>Ambition</i>           | observed data       | 80               | -                                            |
|                           | compartmental model | 143.36           | 0%                                           |
|                           | small-world network | 210.8            | 0%                                           |
|                           | scale-free network  | 45.42            | 32%                                          |

**Figure 12.** The effect of  $E(0)$  on numerical simulations of ANDV in compartmental models. (a) Statistical analysis of multiple numerical simulations with  $E(0) = 20$ . (b) Comparison of the relative standard deviation between  $E(0) = 2$  and  $E(0) = 20$ .

#### 4.2. The Effect of Model Structure

Figures 6, 10 and 11 show that the transmission outcomes of the compartmental model, SW network, and SF network are all different. The compartmental model assumes that any two individuals in the population have an equal probability of contact, which may lead to faster transmission. The figures indicate that the transmission results of the SF network model are lower than those of the compartmental model and SW network, particularly in the norovirus model. This finding is inconsistent with the theoretical analysis that, when the total number of nodes in the network is large, the disease spreads more easily in an SF network. This discrepancy arises because, in an SF network, very few nodes have a high degree, the initial number of infected individuals is extremely small, and most nodes have a relatively low degree. Consequently, the probability that an infected individual has a high degree is very low, which reduces the disease transmission rate. Due to stochasticity, if the initial transmission attempts fail, the epidemic may face extinction; success may trigger an outbreak.

Figures 4, 5, 8 and 9 show that in small-world (SW) and scale-free (SF) networks, the results of individual simulation runs exhibit significant variability. This contradicts findings from previous studies, where the results of a single simulation tend toward a stable value when the total number of nodes is large [29,30]. This discrepancy is also attributable to the

very low number of initial infected individuals. Table 3 lists the proportion of simulation runs that resulted in extinction ( $I_{\text{end}} = 0$ ) relative to the total number of runs. It shows that the results for the SW network and the stochastic compartmental model are relatively consistent, with low proportions ranging between 0% and 4%. In contrast, the proportions for the SF network are substantially higher, ranging between 24% and 38%. Although both are network models, the SW network, due to its homogeneity, exhibits transmission characteristics closer to those of the compartmental model. The SF network, however, owing to its heterogeneity, displays transmission characteristics that are markedly different from both the SW network and stochastic compartmental model.

Network heterogeneity essentially manifests as multiscale dynamics: degree disparities between hubs and peripheral nodes lead to heterogeneous transmission responses, challenging reduced-order modeling and conceptually linking to adaptive basis representations such as adaptive Fourier decomposition for complex systems [31]. This inherent structural variability poses substantial challenges for inverse parameter estimation and uncertainty propagation: observed outbreak outcomes may be dominated by rare, hub-driven events, so that conventional least-squares fitting—whose optimality corresponds to Gaussian likelihood assumptions and which inherently overlooks the heavy-tailed nature of the degree distribution—can yield severely biased estimates. While a formal treatment of inverse problems or adaptive modeling strategies lies beyond the scope of this study, our results underscore the need for uncertainty-aware, heteroscedastic inference frameworks when applying network models to low-incidence settings. Notably, the strong sensitivity to hub-dominated dynamics observed in our numerical experiments provides a clear empirical motivation for exploring such adaptive decomposition methods. We draw a conceptual analogy with schemes that construct problem-adapted basis functions for multiscale solutions: although such methods are originally designed for continuous spatial domains rather than graph-structured networks, their core philosophy—constructing data- or model-adapted bases to capture multiscale features—offers an intriguing perspective for representing the heterogeneous transmission patterns observed here. We acknowledge, however, that the concrete implementation of such adaptive basis approaches in network-based epidemiology would require substantial problem-specific adaptations, including the development of graph-adapted basis functions and corresponding inference algorithms.

#### 4.3. Relation to Modern Computational Paradigms

It is instructive to position our traditional stochastic modeling framework within the rapidly evolving landscape of modern computational mathematics. In recent years, the computational sciences have witnessed rapid advances in adaptive operator-learning methodologies and neural representation frameworks that aim to construct data-driven surrogates for complex dynamical systems (e.g., adaptive Mamba neural operators [32]). These approaches are particularly powerful when massive high-dimensional data are available and underlying physical or biological laws are partially unknown, typically exploiting low-dimensional structures in the solution manifold to achieve computational efficiency and generalization.

This study, by contrast, adheres to a classical mechanistic paradigm. We explicitly prescribe transmission rules, contact structures, and stochastic transition probabilities, which confers distinct advantages in interpretability, biological plausibility, and parameter identifiability—especially in the low-incidence scenarios we examine, where data are too sparse to train a data-hungry operator. In the absence of extensive observational data, classical modeling approaches remain a valuable and often irreplaceable tool. In this sense, our framework and emerging operator-learning paradigm should be viewed as complementary rather than competitive: the former provides a transparent causal backbone

suited for data-sparse regimes, while the latter offers scalability and adaptability when rich observational or simulation data become available.

Furthermore, the finding that stochasticity dominates when the initial infection count is extremely small prompts a broader methodological consideration. The unpredictability of individual trajectories in low-incidence, early-stage outbreaks is not a deficiency of our model but an inherent property of the underlying branching process, where demographic stochasticity fundamentally governs extinction versus takeoff. This stands in marked contrast to the premises underlying many modern adaptive computational paradigms—including automated operator construction from large-scale scientific data or LLM-assisted neural operator design—which typically presume the existence of sufficient training data to learn implicit dynamical structures or low-dimensional solution manifolds. In our setting, however, the “signal” from a handful of initial cases is extremely weak, while stochastic fluctuations are disproportionately large. Under such severely data-sparse conditions, reliably reconstructing a single outbreak’s realized path via purely data-driven methods becomes impractical, because the randomness is path-dependent and manifests along one specific stochastic trajectory rather than being averaged over an ensemble. While data-driven methods could, in principle, approximate the underlying transition distributions given sufficiently rich training data covering the relevant state space, such data are rarely available in the extreme low-incidence, early-stage scenarios where public health decisions are most urgently needed. Their extrapolation reliability therefore remains an open and pressing question. Mechanistic stochastic models, despite their computational simplicity, thus provide an irreplaceable benchmark for quantifying uncertainties that are structural and path-dependent rather than merely parametric or numerical.

#### 4.4. Relation to Biological Reality

While this study is primarily methodological and computational, we recognize that the parameters governing our models— $\beta$ ,  $\gamma$ , and contact network topologies—are not arbitrary mathematical constructs but emergent manifestations of complex biological processes. The environmental persistence of norovirus on cruise ship surfaces, for instance, directly influences the duration over which fomite-mediated transmission remains viable; similarly, the aerosol infectivity of hantavirus depends on viral stability under different temperature and humidity conditions, as well as the dose–response relationship governing human infection probability. Moreover, host-related biological factors—including age-dependent susceptibility, pre-existing immunity, nutritional status, and behavioral patterns—introduce additional layers of heterogeneity that our stochastic simulations capture only in aggregated form through variability in infection outcomes.

We acknowledge that a mechanistically comprehensive understanding of disease transmission would require coupling epidemiological compartmental dynamics with submodels of microbial physiology, environmental virology, and host immunology. Recent efforts in systems biology have begun to explore such integration [33,34], for example, by embedding pharmacokinetic or immunodynamic equations within population-level frameworks or by linking pathogen metabolic activity to environmental survival curves. This style of interdisciplinary integration is not without precedent; as illustrated in the context of microbial production of high-value products [35], the incorporation of biological principles—such as the biosynthesis of straight-chain aliphatic carboxylic acids—can effectively bridge the gap between mechanistic biological understanding and mathematical abstraction without altering the core quantitative objectives of a study. Following this principle, our work leverages biological knowledge to inform and justify model assumptions, while retaining quantitative analysis as the central focus.

While such extensions lie beyond the scope of this work, our demonstration of stochastic dominance under extreme low-incidence conditions underscores the need for future interdisciplinary research that explicitly models the biological drivers of randomness—including stochastic viral shedding, heterogeneous host immune responses, and environmentally modulated pathogen decay—rather than treating them solely as statistical noise. A sustained biological perspective will be essential for mathematical epidemiology to move beyond phenomenological fitting toward mechanistically grounded predictions, particularly in the data-sparse, early-stage regimes where stochasticity dominates.

#### 4.5. Computational Performance and Evaluation Considerations

The deterministic ODE model scales as  $O(N^2)$ , where  $N$  is the total node number, and is computationally inexpensive. In contrast, the stochastic network simulations, when discretized with a fixed time step, scale as  $O(M \times T)$ , where  $M$  is the edge count and  $T$  is the number of time steps, rendering dense scale-free networks substantially more demanding. As both the initial number of infections and number of independent realizations,  $R$ , increase, the stochastic simulations converge to their deterministic limits, with the ensemble standard error decaying as  $O(1/\sqrt{R})$ . While qualitative patterns remain robust under minor parameter perturbations, the quantitative estimates—such as outbreak probabilities—exhibit significant sensitivity to  $\beta$  and  $\gamma$ . This underscores the importance of rigorous sensitivity analyses to properly contextualize our predictions. All simulations are reproducible using fixed random seeds, and our event-driven Gillespie algorithm ensures numerical stability by circumventing fixed-step discretization errors.

Beyond performance, it is instructive to position our mechanistic, edge-based simulations relative to emerging data-driven paradigms. For example, message-passing frameworks such as MPFVM incorporate information propagation across structured domains to improve computational representation and numerical performance in scientific computing [36]. While such approaches can learn complex propagation patterns from large datasets, their extrapolation reliability in extreme, low-incidence regimes—exactly the focus of this work—is less established. Our framework provides a causally transparent, stable, and training-free baseline. These two approaches should thus be viewed as complementary rather than competitive, highlighting the value of maintaining a diverse methodological toolkit in computational epidemiology.

## 5. Conclusions

The findings presented above, while focused on cruise ship disease transmission, resonate with several broader developments across computational mathematics, data science, and systems biology. We summarize our main conclusions as follows.

First, our results demonstrate that when the initial number of infected or exposed individuals is extremely small (on the order of 1–2 persons), stochastic variability overwhelms deterministic predictions. This holds across all three modeling frameworks—compartmental, SW, and SF networks—and for both pathogens considered (ANDV and norovirus). The unpredictability of individual outbreak trajectories is not a shortcoming of our models but a fundamental property of the underlying stochastic processes. This finding carries methodological implications beyond epidemiology: in the era of data-driven scientific computing, where neural operators and adaptive surrogate models are increasingly employed to accelerate simulations and learn implicit dynamical structures from large-scale datasets, our results serve as a cautionary reminder that in data-sparse, low-incidence regimes, intrinsic randomness cannot be easily eliminated by even the most sophisticated algorithms. Rather, mechanistic stochastic models—despite their computa-

tional simplicity—provide an irreplaceable benchmark for understanding uncertainties that are structural rather than numerical in nature.

Second, the systematic comparison across network topologies reveals that scale-free networks exhibit lower mean transmission and higher extinction probabilities than homogeneous (compartmental or SW) models under low initial infection conditions. This apparent departure from classical mean-field expectations is attributable to the skewed degree distribution of SF networks: with very few initial infectives, the probability of occupying a hub node is extremely low, and the outbreak either dies out rapidly or, in rare cases, ignites into a large epidemic if a hub is infected. This “all-or-nothing” behavior underscores the critical importance of network structure in shaping early outbreak dynamics and highlights the limitations of homogeneous mixing assumptions in closed, structured populations such as cruise ships. Our network-centric analysis also aligns with recent advances in graph-based scientific computing, where information propagation across structured domains is modeled using message-passing or adaptive decomposition strategies. While our use of small-world and scale-free networks remains within the classical epidemiological tradition, the conceptual parallels with modern graph neural networks and adaptive Fourier decomposition methods suggest promising avenues for future research.

Finally, we acknowledge that a complete understanding of disease transmission ultimately requires bridging mathematical abstraction with biological reality. Although our study does not explicitly model microbial metabolism, environmental persistence, or host immune responses, the transition probabilities and transmission rates we employ are emergent properties of underlying biological processes—including pathogen survival on fomites, dose–response relationships, and human behavioral patterns. Future interdisciplinary efforts could enrich our framework by incorporating physiologically based models of host–pathogen dynamics, climate-dependent pathogen viability, or immunodynamic submodels, thereby creating a more holistic modeling ecosystem that spans molecular, individual, and population scales.

In conclusion, for closed environments such as cruise ships with very few initial cases, stochastic variability is an essential and irreducible feature of disease transmission. Single outbreak outcomes should be understood as highly contingent rather than deterministically predictable. The choice of modeling framework—whether compartmental or network-based—qualitatively shapes transmission estimates, with heterogeneous networks amplifying stochastic uncertainty. Maintaining a diverse methodological toolkit, combining mechanistic stochastic models with emerging data-driven approaches, will be essential for robust epidemiological decision-making in the face of inherent uncertainty.

**Author Contributions:** Conceptualization, G.W. and W.Y.; methodology, W.Y.; software, W.Y.; validation, G.W. and W.Y.; formal analysis, G.W.; investigation, G.W.; resources, G.W.; data curation, W.Y.; writing—original draft preparation, G.W. and W.Y.; writing—review and editing, G.W. and W.Y.; supervision, W.Y.; project administration, W.Y.; funding acquisition, W.Y. All authors have read and agreed to the published version of the manuscript.

**Funding:** This research was funded by the National Natural Science Foundation of China (grant number: 12172092) and Shanghai Key Laboratory of Acupuncture Mechanism and Acupoint Function (grant number: 21DZ2271800).

**Data Availability Statement:** Available on request from the corresponding author.

**Conflicts of Interest:** The authors declare no conflicts of interest.

## Abbreviations

The following abbreviations are used in this manuscript:

|                     |                                                                  |
|---------------------|------------------------------------------------------------------|
| CLIA                | Cruise Lines International Association                           |
| VSP                 | Vessel Sanitation Program                                        |
| CDC                 | U.S. Centers for Disease Control and Prevention                  |
| ANDV                | Andes virus                                                      |
| EAKF                | Ensemble Adjustment Kalman Filter                                |
| SW                  | small-world                                                      |
| SF                  | scale-free                                                       |
| SEIRD               | Susceptible–Exposed–Infectious–Recovered–Deceased                |
| SEIR                | Susceptible–Exposed–Infectious–Recovered                         |
| WHO                 | World Health Organization                                        |
| ECDC                | European Centre for Disease Prevention and Control               |
| $R_0$               | basic reproduction number                                        |
| $\beta$             | effective contact rate                                           |
| $\omega$            | transfer rate from the exposed to the infected                   |
| $\gamma$            | the recovery rate                                                |
| $\langle k \rangle$ | average degree                                                   |
| SD                  | standard deviation                                               |
| $I_{\text{mean}}$   | mean of infected individuals                                     |
| $I_{\text{SD}}$     | standard deviation of the number of infected individuals         |
| $I_{\text{end}}$    | observed number of infected individuals at the end of the voyage |

## References

1. CLIA. *State of the Cruise Industry Report*; 8 May 2024; Cruise Lines International Association, Inc.: Washington, DC, USA, 2024; pp. 1–43.
2. CLIA. *State of the Cruise Industry Report*; 19 May 2025; Cruise Lines International Association, Inc.: Washington, DC, USA, 2025; pp. 1–67.
3. Kak, V. Infections in Confined Spaces: Cruise Ships, Military Barracks, and College Dormitories. *Infect. Dis. Clin. N. Am.* **2007**, *21*, 773–784. [CrossRef] [PubMed]
4. Pavli, A.; Maltezou, H.C.; Papadakis, A.; Katerelos, P.; Saroglou, G.; Tsakris, A.; Tsiodras, S. Respiratory infections and gastrointestinal illness on a cruise ship: A three-year prospective study. *Travel Med. Infect. Dis.* **2016**, *14*, 389–397. [CrossRef] [PubMed]
5. Zhang, N.; Miao, R.; Huang, H.; Chan, E.Y.Y. Contact infection of infectious disease onboard a cruise ship. *Sci. Rep.* **2016**, *6*, 38790. [CrossRef] [PubMed]
6. Mouchtouri, V.; Lewis, H.; Hadjichristodoulou, C. A Systematic Review for Vaccine-Preventable Diseases on Ships: Evidence for Cross-Border Transmission and for Pre-Employment Immunization Need. *Int. J. Environ. Res. Public Health* **2019**, *16*, 2713. [CrossRef] [PubMed]
7. Rocklöv, J.; Sjödin, H.; Wilder-Smith, A. COVID-19 outbreak on the Diamond Princess cruise ship: Estimating the epidemic potential and effectiveness of public health countermeasures. *J. Travel Med.* **2020**, *27*, taaa030. [CrossRef] [PubMed]
8. Jenkins, K.A.; Vaughan, G.H., Jr.; Rodriguez, L.O.; Freeland, A. Acute gastroenteritis on cruise ships—Maritime Illness Database and Reporting System, United States, 2006–2019. *MMWR Surveill. Summ.* **2021**, *70*, 1–19. [CrossRef] [PubMed]
9. Centers for Disease Control and Prevention. Available online: <https://www.cdc.gov/vessel-sanitation/cruise-ship-outbreaks/star-princess-march-2026.html> (accessed on 27 May 2026).
10. CDC. Available online: <https://www.cdc.gov/vessel-sanitation/cruise-ship-outbreaks/caribbean-princess-may-2026.html> (accessed on 27 May 2026).
11. Casassus, B. Norovirus: 50 cruise passengers fall ill with viral gastroenteritis in France. *BMJ* **2026**, *393*, s949. [CrossRef] [PubMed]
12. Taylor, L. Hantavirus: Three dead in cruise ship outbreak as authorities investigate human-to-human transmission. *BMJ* **2026**, *393*, s875. [CrossRef] [PubMed]
13. Batista, B.; Dickenson, D.; Gurski, K.; Kebe, M.; Rankin, N. Minimizing disease spread on a quarantined cruise ship: A model of COVID-19 with asymptomatic infections. *Math. Biosci.* **2020**, *329*, 108442. [CrossRef] [PubMed]
14. Emery, J.C.; Russell, T.W.; Liu, Y.; Hellewell, J.; Pearson, C.A.; Atkins, K.E.; Klepac, P.; Endo, A.; Jarvis, C.I.; Davies, N.G.; et al. The contribution of asymptomatic SARS-CoV-2 infections to transmission on the Diamond Princess cruise ship. *elife* **2020**, *9*, e58699. [CrossRef] [PubMed]

15. De Bellis, A.; Bizzotto, A.; Anagnostopoulos, L.; Kourentis, L.; Marziano, V.; Mouchtouri, V.; Merler, S.; Guzzetta, G. Mitigating norovirus spread on cruise ships: A model-based assessment of diagnostic timing and isolation. *J. Travel Med.* **2025**, *32*, taaf059. [[CrossRef](#)] [[PubMed](#)]
16. Cui, J. Modeling the Impact of Exposed Cases in a Hantavirus Outbreak on a Cruise Ship. *arXiv* **2026**, arXiv:2605.07498. [[CrossRef](#)]
17. Chen, T. *Examining Climate-Driven Influences on Norovirus Transmission and Disease Patterns*; National Collaborating Centre for Environmental Health: Vancouver, BC, Canada, 2025; pp. 1–24.
18. Chan, M.C.W.; Kwan, H.S.; Chan, P.K.S. Structure and genotypes of noroviruses. In *The Norovirus*; Chan, P.K., Ed.; Academic Press: New York, NY, USA, 2017; pp. 51–63.
19. Martínez, V.P.; Di Paola, N.; Alonso, D.O.; Pérez-Sautu, U.; Bellomo, C.M.; Iglesias, A.A.; Coelho, R.M.; López, B.; Periolo, N.; Larson, P.A.; et al. “Super-Spreaders” and Person-to-Person Transmission of Andes Virus in Argentina. *N. Engl. J. Med.* **2020**, *383*, 2230–2241. [[CrossRef](#)] [[PubMed](#)]
20. Halwe, N.J.; Tscherné, A.; Krammer, F. Andes hantavirus—A primer. *npj Viruses* **2026**, *4*, 27. [[CrossRef](#)] [[PubMed](#)]
21. Gleeson, J.P.; Melnik, S.; Ward, J.A.; Porter, M.A.; Mucha, P.J. Accuracy of mean-field theory for dynamics on real-world networks. *Phys. Rev. E* **2012**, *85*, 026106. [[CrossRef](#)] [[PubMed](#)]
22. Barabási, A.L.; Albert, R. Emergence of scaling in random networks. *Science* **1999**, *286*, 509–512. [[CrossRef](#)] [[PubMed](#)]
23. van den Driessche, P.; Watmough, J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.* **2002**, *180*, 29–48. [[CrossRef](#)] [[PubMed](#)]
24. Galiceanu, M. Relaxation dynamics of scale-free polymer networks. *Phys. Rev. E* **2012**, *86*, 041803. [[CrossRef](#)] [[PubMed](#)]
25. Pereira, M.R. World Health Organization—Hantavirus cluster linked to cruise ship travel, Multi-country. *Am. J. Transplant.* **2026**, S1600–S6135. [[CrossRef](#)] [[PubMed](#)]
26. Pung, R.; Firth, J.A.; Spurgin, L.G.; Chang, A.; Kong, J.; Wong, J.; Jin, O.J.; Selvaraj, D.; Yong, D.; Lang, J.; et al. Using high-resolution contact networks to evaluate SARS-CoV-2 transmission and control in large-scale multi-day events. *Nat. Commun.* **2022**, *13*, 1956. [[CrossRef](#)] [[PubMed](#)]
27. Vial, P.A.; Valdivieso, F.; Mertz, G.; Castillo, C.; Belmar, E.; Delgado, I.; Tapia, M.; Ferrés, M. Incubation period of hantavirus cardiopulmonary syndrome. *Emerg. Infect. Dis.* **2006**, *12*, 1271–1273. [[CrossRef](#)] [[PubMed](#)]
28. Towers, S.; Chen, J.; Cruz, C.; Melendez, J.; Rodriguez, J.; Salinas, A.; Yu, F.; Kang, Y. Quantifying the relative effects of environmental and direct transmission of norovirus. *R. Soc. Open Sci.* **2018**, *5*, 170602. [[CrossRef](#)] [[PubMed](#)]
29. Wang, G.; Yao, W. An application of complex networks on predicting the behavior of infectious disease on campus. *Math. Methods Appl. Sci.* **2025**, *48*, 189–206. [[CrossRef](#)]
30. Wang, G.; Yao, W. Analysis of Multi-Interacting Networks and Their Application to HIV Transmission Among Men Who Have Sex with Men. *Symmetry* **2025**, *17*, 165. [[CrossRef](#)]
31. Song, Z.; Jiang, Z. Adaptive Fourier decomposition-guided neural operator design for inverse PDE Problems. In Proceedings of the International Conference on Learning Representations (ICLR), Rio de Janeiro, Brazil, 23 April 2026.
32. Song, Z.; Jiang, Z. Adaptive mamba neural operators. In Proceedings of the International Conference on Learning Representations (ICLR), Rio de Janeiro, Brazil, 23 April 2026.
33. Howard-Varona, C.; Lindback, M.M.; Fudyma, J.D.; Krongauz, A.; Solonenko, N.E.; Zayed, A.A.; Andreopoulos, W.B.; Olson, H.M.; Kim, Y.; Kyle, J.E.; et al. Environment-specific virocell metabolic reprogramming. *ISME J.* **2024**, *18*, wrac055. [[CrossRef](#)] [[PubMed](#)]
34. Hong, S.; Park, K. Multi-physiology modeling of the immune system in the era of precision immunotherapy. *Front. Immunol.* **2025**, *16*, 1548768. [[CrossRef](#)] [[PubMed](#)]
35. Zhuang, L.; Liu, Y.; Zhang, H. Microbial Biosynthesis of Straight-Chain Aliphatic Carboxylic Acids. In *Microbial Production of High-Value Products*; Rehm, B.H.A., Wibowo, D., Eds.; Microbiology Monographs; Springer: Cham, Switzerland, 2022; Volume 37. [[CrossRef](#)]
36. Song, Z.; Jiang, Z. MP-FVM: Enhancing finite volume method for water infiltration modeling in unsaturated soils via message-passing encoder-decoder network. *Comput. Geotech.* **2026**, *190*, 107745. [[CrossRef](#)]

**Disclaimer/Publisher’s Note:** The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.