

Network analysis of the roles of fomites, patients, and healthcare personnel to identify potential superspreaders of healthcare-associated infections (HAIs) within a hospital setting

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Abstract: Healthcare-associated infections (HAIs) increase patient morbidity, mortality, and healthcare burdens, yet the complex interplay between patients, healthcare workers, and fomites in transmission dynamics remains poorly understood. Traditional static network models fail to capture temporal fluctuations in contact patterns or the combined dynamics of patients, healthcare personnel, and fomites.

Objective: To investigate the roles of patients, fomites, and healthcare personnel in HAI transmission dynamics within a hospital ward, identify superspreaders using temporal network analysis, and evaluate screening and isolation intervention effectiveness.

Methods: We constructed a directed, weighted, temporal network comprising 94 nodes (18 patients, 6 nurses, 4 doctors, 14 allied health staff, 8 porters, and 44 fomites) based on published contact probabilities and staffing ratios. Bootstrapped temporal exponential random graph models (bTERGM) with 100 replica-

tions identified mechanisms structuring contact patterns. A Susceptible-Exposed-Asymptomatic-Infected-Recovered (SEAIR) compartmental network model was developed to simulate *Clostridioides difficile* transmission across 100 network iterations and 100 SEAIR iterations per scenario (40,000 total simulations). We evaluated screening and isolation interventions at 25%, 50%, and 75% effectiveness, comparing full dynamic, static person-to-person, and homogeneous environmental models.

Results: The networks exhibited moderate density (0.092, 95% CI: 0.088–0.096) and modularity (0.249, 95% CI: 0.233–0.266) indicating moderate tendency of nodes to form triangular relationships and moderate connectivity typical of hospital contact patterns. bTERGM revealed sparse baseline connectivity (OR = 0.062), strong reciprocity (OR = 3.28), and homophily by node type (OR = 1.54) indicating that hospital networks are organized by professional roles and bidirectional relationships rather than random interactions. Nurses had the highest degree

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centrality (43.9 ± 2.1), followed by patients (37.3 ± 1.8) and doctors (26.0 ± 3.2) highlighting their different superspreader potential in healthcare settings. Nurses, patients and allied health worker groups were in the top 15 superspreaders in the SEAIR model simulations with six, six and three nodes respectively. The static model underestimated outbreak size by 29.0% ($p = 2.62 \times 10^{-15}$). Screening interventions produced “dose-dependent” reductions: 21.2% at 25%, 44.2% at 50%, and 70.1% at 75% effectiveness, progressively flattening the transmission hierarchy as the top 10 superspreaders infection contribution declined from 80.3% to 57.5% events.

Conclusion: This study demonstrates that patients and connected healthcare workers are the main drivers of HAI transmission, with screening and isolation achieving substantial HAI reductions while simultaneously attenuating superspreader contributions. These findings provide an evidence-based framework for optimizing infection control strategies through targeted patient screening and recognition of structured healthcare worker-patient-fomite interaction patterns.

Keywords: healthcare-associated infections, temporal network analysis, superspreaders, bootstrapped temporal exponential random graph models, *Clostridioides difficile*

I. INTRODUCTION

Healthcare-associated infections (HAIs) are a significant challenge in healthcare settings, contributing to increased morbidity, mortality of patients and higher costs for healthcare facilities, as recently aggravated during the COVID-19 pandemic [1]. The transmission dynamics of HAI pathogens (such as *Clostridioides difficile*) are complex, involving various entities, including patients, healthcare personnel, and fomites, including objects or surfaces that can harbor and transmit pathogens [2].

The concept of superspreaders, individual hosts or surfaces that contribute to the spread of infections disproportionately, has garnered attention in infectious disease research [3]. Also, contact networks are an important concept and technique in characterizing transmission dynamics, indicating the frequency, duration, and nature of interactions among individual hosts and between individual hosts and surfaces. A study highlighted that superspreaders were responsible for the largest onset of COVID-19 during 2 weeks in a study conducted in a university in Cambridge in the United Kingdom during the early periods of the COVID-19 outbreak in the United Kingdom [3]. Therefore, identifying superspreaders plays a crucial role in evaluating transmission risks of HAIs [4,5].

In a hospital setting, the interactions among patients, nurses, clinicians, allied healthcare staff, and

fomites can be conceptualized as a network where nodes represent individual hosts and surfaces, and edges represent the interactions between nodes [4]. Fomites such as contaminated surfaces, serve as reservoirs and play a critical role in the indirect transmission of HAI pathogens, but their roles are not fully incorporated in modeling efforts [2]. Also, healthcare personnel are often the first point of contact with infected patients. Healthcare personnel could be an important node in the transmission network if proper HAI control, such as hygiene protocols, are not adequately followed. The interactions between healthcare personnel and patients, as well as their interactions with fomites, create a complex network with various transmission pathways.

Accurately characterizing and analyzing such networks will lead to identifying key factors contributing to superspreading events [6,7].

Superspreading is especially important in HAI contexts, as certain individuals or surfaces disproportionately contribute to pathogen transmission, amplifying infection risks [3]. Current models often fail to capture the complexity of interactions, leaving gaps in understanding how superspreading events unfold [4]. Network models can address these gaps by identifying key nodes and interactions that drive superspreading, helping to inform more targeted infection control strategies [8–10].

However, previous network-based studies of hospital transmission have predominantly employed static network approaches or leveraged retrospective data that did not adequately capture the temporal dynamics of healthcare worker-patient interactions [4, 11]. For instance, Rocha et al. 2020 [4] demonstrated the utility of contact networks for *methicillin-resistant Staphylococcus aureus* (MRSA) transmission but acknowledged the limitation of their static framework in representing shift-based variations in staffing patterns.

Similarly, studies quantifying fomite contributions to HAI transmission [2, 12] have typically treated environmental contamination as a homogeneous risk factor rather than as discrete nodes within a transmission network, potentially oversimplifying the spatial heterogeneity of pathogen distributions in hospital settings. While compartmental models can incorporate environmental reservoirs through indirect transmission terms [13], they may not be able to identify specific high-risk fomites or capture the network position of contaminated surfaces in relation to patient and healthcare worker movements. The present study addresses these gaps by: (1) integrating fomites as discrete, temporally varying nodes within a dynamic contact network, (2)

quantifying their centrality relative to human nodes, and (3) evaluating how this network structure modifies intervention effectiveness.

While various modeling techniques such as static Exponential Random Graph Models (ERGMs) and compartmental models have been used to study infectious disease transmission, these approaches often fall short in capturing the temporal and heterogeneous nature of contact patterns in hospital settings [4,11]. ERGMs, for example, assume fixed relationships and cannot account for changes in network structure over time.

Among various network modeling techniques, bootstrapped temporal exponential random graph models (bTERGM) allow for advanced analysis of the dynamic nature of interactions among nodes over time. By incorporating both directed and weighted edges, bTERGM can capture the strengths and directionality of contacts, thus providing evidence on how superspreading events would occur [14]. In an epidemiological context, directed edges represent the direction of pathogen transmission, such as from a healthcare worker to a patient, while weighted edges indicate the intensity or frequency of interactions, reflecting the potential for higher transmission risk with prolonged duration of contact [15]. Furthermore, incorporating the temporal dynamics of interactions is essential as interactions may vary between day and night shifts.

This study aims to investigate the roles of fomites, patients, and healthcare personnel in the transmission dynamics of HAI pathogens within a hospital ward, with a particular focus on identifying superspreaders through the application of temporal exponential random graph models (bTERGM). This study also creates a Susceptible-Exposed-Asymptomatic-Infected-Recovered (SEAIR) compartmental network model to investigate the impact of different intervention (“screening and isolation”) scenarios for the spread of *Clostridioides difficile* (*C. difficile*) in the network. By characterizing the modeling of temporal dynamics and identifying superspreaders, this study contributes valuable insights that can inform more targeted control strategies and ultimately reduce the risk of HAIs in healthcare settings.

II. METHODS

A. Simulated contact network with hosts and fomites

This study involved the simulation of a directed, weighted, stochastic, and temporal network in an in-patient hospital ward. Mathematically, a network (or graph G) consists of two main sets of components: a set of nodes (or vertices V) and a set of edges (E)

characterizing the connection of the nodes. Additionally, a network G whose structure varies over time t is known as a dynamic (or temporal) network such that $G_t = \{V_t, E_t, t\}$.

The simulated network was informed by literature on real-world in-patient general wards, which has a range of 12 to 30 patient beds, and we estimated a 75 percent occupancy rate for a 24-bed ward, hence, our network consists of 18 patients [16, 17] based on Organisation for Economic Co-operation and Development, OECD-reported occupancy rates for general hospital wards. Each patient will have a corresponding bedrail and bedside table, hence, there are 18 bedrails and 18 bedside tables. The number of healthcare workers was based on staffing ratios, for instance, there is usually 1 nurse for every 4 to 6 patients in an in-patient ward with two shift work patterns [18, 19].

The simulated network consisted of 94 nodes, representing different elements within the hospital setting including multiple patients, healthcare personnels, and fomites. There were 18 patients, 6 nurses, 4 doctors, 14 ancillary healthcare staff (AHS), and 8 porters host nodes. The fomites node included 18 bedrails, 18 bedside tables, and 8 laundry carts. These 94 nodes interacted with each other to form edges in the network, with each interaction contributing to the spread of the pathogen and potential superspreading.

B. Edge formation

Edges, or connections between these nodes, represented potential interactions (i.e. contact events) that lead to potential transmission. The edges were quantified by the contact probabilities based on contact frequency between node pairs [20] edge weights were based on contact durations, as described in [20] and [21]. These contact probabilities and edge weight varied depending on interactions of specific pairs of nodes. For example, interactions from nurses to patients were given a contact probability of 0.56, while interactions from nurses to bedrails had a lower probability of 0.27. These reflected both direct host-host and indirect host-fomite interactions in real hospital settings. The complete contact probability matrix and edge weight assignments for node pairs are provided in Table 6, with values derived from published studies of hospital contact patterns [20,21]. More details of the parameters can be found in Tables 1, 6, 7.

The model was simulated to capture contacts over seven days with two shifts (day and night shifts) per day. The two-shift schedule is commonly utilized in many hospitals [18, 22]. The 7-day simulation period

was to reflect the average length of stay of patients during the COVID-19 pandemic, and this is also the time where healthcare workers have about the same schedule or shift [23, 24]. Shift distributions were calibrated to match observed contact patterns from wearable sensor studies in hospital settings [20], with 60% of the interactions occurring during the day shift (from 7 am to 7 pm), with the remaining 40% occurring during the night shift (from 7 pm to 7 am) to reflect real-world hospital settings.

To further capture the realistic contact structures, a few node pairs were assigned with fixed interactions with other nodes in the network. For instance, patients were linked with their respective bedrails and bedside tables, as an admitted patient is usually assigned with a specific bed with a fixed bedrail and a bedside table. These fixed host-fomite interactions represented static elements of the hospital's realistic physical layout and operations. However, patients could interact with different doctors, nurses, and other nodes in the same network. Similarly, doctors, nurses, and other nodes could also interact with different fomites such as bedrails and bedside tables.

C. Simulating temporal variability in network interactions

Temporal edges, representing interactions between different nodes over time, were generated stochastically based on predefined contact rates. To simulate the temporal variability in the network, we introduced a dynamic framework where edges were formed probabilistically at each time step, reflecting the nature of real-world interactions. Specifically, the probability of an edge forming between two nodes was determined by the contact probability matrix, which varied based on roles (e.g., nurse-to-patient, doctor-to-nurse) and interaction contexts.

Additionally, shift-based dynamics were incorporated to reflect the structured nature of hospital workflows, where healthcare workers (nurses, doctors, porters, and AHS staff) were assigned to alternating day and night shifts. During simulation, interactions were weighted differently based on the shift schedule, with higher probabilities assigned to contacts occurring within the same shift, while cross-shift interactions were much less frequent. This approach ensured that the network evolved in a realistic manner over time, capturing both the stochastic nature of host-host/fomite interactions and the structured dynamics of a hospital setting.

D. Network structure characterization

Global network properties were characterized across all 100 network simulations. Density was computed as the proportion of observed edges relative to the maximum possible edges in a directed network of 94 nodes. Transitivity (clustering coefficient) measured the tendency of nodes to form triangular relationships, calculated as the ratio of closed triangles to connected triples [8–11]. Modularity was calculated using the Louvain community detection algorithm applied to each aggregated network, with communities allowed to emerge without pre-specification of group membership. For all properties, we report means with 95% confidence intervals derived from the 2.5th and 97.5th percentiles of the distribution across simulations. Modularity was calculated using the Louvain community detection algorithm applied to the undirected version of each aggregated network [25]. This algorithm iteratively partitions nodes to maximize within-community edge density relative to a random null model, with communities allowed to emerge without pre-specification of group membership.

E. Superspreader identification

To identify key nodes in the network that play a disproportionate role in the spread of HAIs, commonly known as “superspreaders” for HAI transmission dynamics, the degree centrality measure was calculated across all nodes in the network. Among various network properties, the degree centrality, which measures the number of direct connections a node has, helps identify nodes with a large number of interactions with other nodes, and consequently with higher probability for HAI. This centrality measure has been commonly used in superspreader identification beyond the context of HAI [8, 26].

In this study, superspreaders were defined with an 80th percentile threshold for degree centrality measures of the corresponding nodes. The 80th percentile threshold was selected based on established literature defining the “20/80 rule” in infectious disease transmission, where approximately 20% of individuals contribute to 80% of transmission events [8, 27]. Nodes that exceeded this threshold in degree centrality were classified as network-based superspreaders. For transmission-based superspreader identification from SEAIR compartmental epidemic model simulations, we applied the same 80th percentile threshold to the distribution of secondary infections caused per node across all simulations.

F. Temporal network analysis

The Bootstrapped Temporal Exponential Random Graph Model (bTERGM) was employed to capture the evolving dynamics of contact structures within the hospital over time. Compared to static ERGM, bTERGM is well-suited for modeling networks where relationships are not static, but rather evolving [4, 28, 29]. By modeling the dynamic nature of interactions, the bTERGM framework provided insights into how individual nodes' positions and the temporal structure of interactions shape the emergence of new edges. bTERGM was applied to assess how the roles of healthcare workers (nurses, doctors, porters), their network attributes (degree and betweenness centralities), and dyadic effects influence the formation of edges in healthcare networks over time [28, 29].

The bTERGM model was specified with the following terms: edges (baseline propensity), mutual (reciprocity), and nodematch ("type") (homophily by node type). Estimation was performed using maximum pseudolikelihood with 100 bootstrap replications to obtain robust standard errors and 95% confidence intervals [28]. Goodness-of-fit was assessed by comparing observed network statistics (edge count, density, reciprocity) against distributions from 100 networks simulated from the fitted model.

G. SEAIR model for *C. difficile* transmission model and assessing the impact of "screening and isolation" intervention scenarios

This study employed a compartmental network model Susceptible-Exposed-Asymptomatic-Infected-Recovered (SEAIR) to simulate infection transmission dynamics in a healthcare setting for *Clostridioides difficile*. The SEAIR framework categorizes individuals into five compartments: Susceptible (S), Exposed (E), Asymptomatic (A), Infected (I), and Recovered (R). Using the contact network structure and parameters, with directed edges reflecting real world contact probabilities for the simulation. These bidirectional interactions define disease spread or who transferred-infection-to-whom such as nurse3-to-patient12, doctor5-to-nurse2, and patient5toallied health worker. Key SEAIR parameters were derived from published literature [30, 31] such as rate of progression from exposed to asymptomatic ($\sigma = 0.33 \text{ day}^{-1}$), proportion progressing to symptomatic infection ($\alpha = 0.134$), and recovery rate for asymptomatic individuals ($\gamma_A = 0.07 \text{ day}^{-1}$). The complete parameter set is provided in Table 1.

Parameter	Value	Source
β	0.042	[20, 21]
σ	0.15	[32, 33]
α	0.2	[34]
γ_A	0.04	[35, 36]
γ_I	0.18	[37, 38]
p_{contam}	0.7	[39]
δ	0.01	[40, 41]
k_{inc}	2.5	[42]
θ_{inc}	0.4	[42]
D_A	25	[43, 44]
D_I	5	[45, 46]

Table 1: SEAIR parameters *C. difficile*.

With previous studies showing that up to 10% to 20% of patients can have an infection from *Clostridioides difficile* when screened [30], this study models four patients randomly being infected. For each simulation, four patients were randomly seeded as initial infections, with all other host nodes as susceptible. We evaluated a "screening and isolation" intervention at 25%, 50%, and 75% effectiveness, corresponding to isolating 1, 2, or 3 of the four initially infected patients. Isolated patients were rendered non-infectious while remaining in the network. An infection log counter tracked transmission events, and superspreaders were defined as nodes exceeding the 80th percentile of secondary infections caused. For each intervention scenario, we conducted 100 network simulations by 100 SEAIR simulations (10,000 total simulations per scenario) to obtain stable estimates of mean secondary infections with 95% confidence intervals.

H. Model comparison framework

To quantify the contribution of temporal dynamics to outbreak potential, we compared three nested modeling approaches. The Full Dynamic Model incorporated temporal contact sequences and host-to-host transmission, serving as the reference standard. The Static host-to-host Model aggregated all contacts across the 7-day simulation period into a single static network while retaining the same edge weights and transmission probabilities, thereby removing temporal ordering while preserving total contact intensity. The Homogeneous Environmental Model assumed perfectly mixed populations at each node type, using the same transmission probabilities but averaging contact rates across all possible dyads. For each intervention scenario, we compared mean secondary infections between each pair of models using two-tailed *t*-tests. Model performance was further assessed by calculating the

Hospital Contact Network (7 Days) - Standardized Terminology

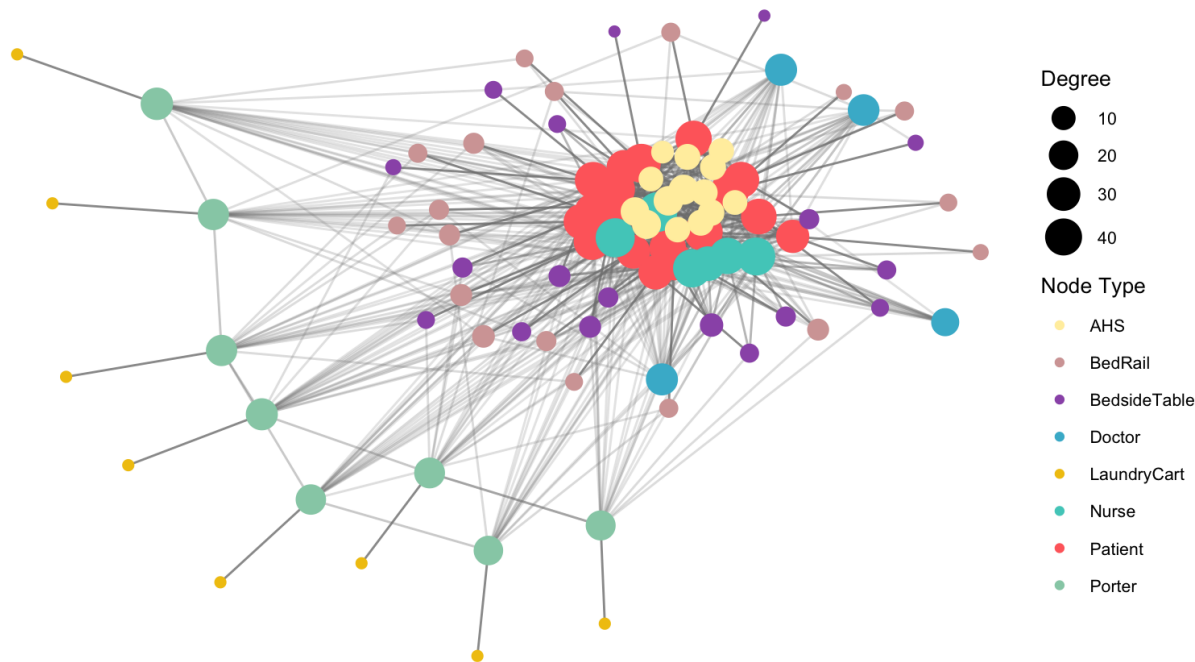


Fig. 1: Overall network structure visualization plot over 7 days.

Metric	Mean	Standard deviation	Confidence interval lower	Confidence interval upper
Density	0.092	0.002	0.088	0.096
Transitivity	0.387	0.015	0.355	0.414
Modularity	0.249	0.008	0.233	0.266
Total contacts/edges in aggregated network	803.460	17.799	767.275	839.525

Table 2: Network metrics characteristics.

percentage underestimation of outbreak size relative to the full dynamic model as $(full - static)/full \times 100$.

1. Key model assumptions

Several key model assumptions were made to simplify the representation of hospital contacts while retaining adequate details for analyses. Contact probabilities and weights for nodes of the same type (e.g., nurses, patients, or fomites) were the same. Additionally, contacts between a pair of nodes were independent of contacts between other pairs, treating each interaction as an independent event. Other key assumptions included a fixed set of pre-defined nodes with no additional nodes added or existing nodes removed during the simulation period. These assumptions can be easily relaxed in future studies.

J. Data preparation, statistical analysis, visualization, software, and tools

Data for this study were generated by a stochastic simulation process that modelled temporal interactions based on predefined contact probabilities and stored in a structured data frame. The data frame contains details of each contact, including the day, time period (day or night shifts), directions (source node and target node), and interaction weight. These data were used to visualizations of the dynamic bTERGM network. Node sizes in these visualizations were proportional to their corresponding degree centrality, and edge widths were scaled by interaction intensity (as indicated by edge weights).

The analysis was performed using *R* version 4.3.0. Key outcomes (average secondary infections, centrality

measures) were summarized as means with standard deviations and 95% confidence intervals derived from the 2.5th and 97.5th percentiles of the simulation distribution across 100 independent network simulations, each with 100 SEAIR simulations per intervention scenario, yielding a total of 10,000 simulations per scenario and 40,000 total simulations across all four intervention levels. Comparisons between model types used two-tailed *t*-tests, with statistical significance defined as $p < 0.05$. Several key libraries including “igraph” for network analysis, “btergm” for temporal network modeling, “dplyr” for data manipulation, and “igraph”, “ggraph”, and “ggplot2” for network visualizations.

K. Data availability

The complete network parameters and their references (Tables 1, 6, 7) and the simulation code and analysis scripts are available on the GitHub repository [47].

L. Ethical approval

This study involved no human subjects, patient data, or clinical interventions. All data were generated through computational simulation, and therefore no ethical approval or informed consent was required.

III. RESULTS

A. Network characteristics and contact patterns

The constructed network consisted of 94 nodes which represented different entities in the hospital ward environment, including patients, healthcare workers (nurses, doctors, ancillary hospital staff, and porters), and fomites (bedrails, bedside tables, and laundry carts). The network exhibited a mean density of 0.092 (95% CI: 0.088-0.096) and transitivity of 0.387 (95% CI: 0.355–0.414), indicating moderate tendency of nodes to form triangular relationships and moderate connectivity typical of hospital contact patterns. Modularity analysis revealed a value of 0.249 (95% CI: 0.233–0.266), indicating moderate community structure consistent with the interdependent and interconnected nature of many hospital ward environments, where interactions span across different roles and fomites. From all simulations, approximately 400,000 individual transmission events were recorded and analyzed. A representative example of the overall network plot and metrics is provided in Figure 1 and Table 2.

Degree-based network analysis identified nurses as the most highly connected nodes, with mean degree centrality of 43.9 ± 2.1 , followed by patients (37.3 ± 1.8) and doctors (26.0 ± 3.2). Using the 80th percentile threshold for superspreader classification, nurses and

Rank	Node	Type	% of simulations as superspreaders
1	Nurse4	Nurse	21%
2	Patient15	Patient	20%
3	Nurse2	Nurse	19%
4	Nurse5	Nurse	19%
5	Nurse6	Nurse	19%
6	Nurse3	Nurse	18%
7	Nurse1	Nurse	17%
8	Patient11	Patient	16%
9	Patient3	Patient	13%
10	Patient4	Patient	13%
11	AHS4	AHS	11%
12	AHS5	AHS	11%
13	Patient14	Patient	12%
14	Patient17	Patient	9%
15	AHS7	AHS	9%

Table 3: Top 15 superspreaders ranked by frequency of superspreader identification.

patients showed the highest probability of being identified as network-based superspreaders with six nurses and sixteen patients meeting or being above the 80th percentile threshold based on their network degree.

B. SEAIR infection transmission analysis

The SEAIR model simulations revealed pronounced heterogeneity in individual transmission potential of *C. difficile*, with a small subset of healthcare workers and patients functioning as recurrent superspreaders. Nurse4 was identified as a superspreader in 21% of simulations, the highest frequency among all nodes. Patient15 followed closely, appearing as a superspreader in 20% of simulations, the highest among patients. Nurse2, Nurse5, and Nurse6 each appeared in 19% of simulations, with Nurse3 in 18% and Nurse1 in 17%.

In addition to these consistent potential asuperspreaders, Patient11 was identified in 16% of simulations, Patient3 and Patient4 in 13%, Patient14 in 12%, and Patient17 in 9% of all simulations. Allied health staff appeared in the ranking with AHS4 and AHS5 in 11% of simulations, AHS7 in 9%, AHS3, AHS1, and AHS12 in 8%, AHS8 in 7%, and AHS2 in 6%. Table 3 presents the top 15 superspreaders ranked by the percentage of simulations in which they were identified as superspreaders.

C. Transmission dynamics under baseline (“no intervention”) scenario

The full dynamic model, simulating outbreaks, yielded mean secondary infections of 18.77 ± 3.42 (95% CI: 12–26) from four initially infected patients



Fig. 2: Example of network graph at various intervention level scenario models.

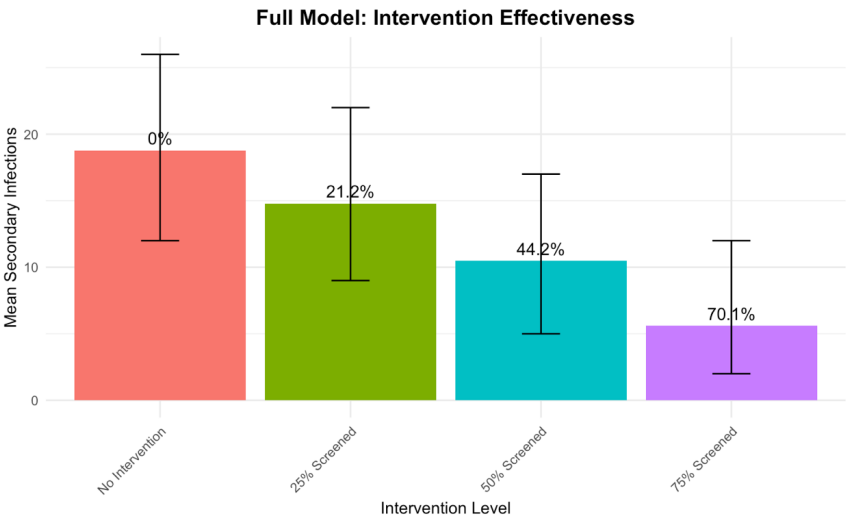


Fig. 3: Full dynamic model intervention effectiveness graph.

in the absence of interventions. The static person-to-person model, which aggregates all contacts without temporal dynamics, produced significantly smaller epidemic size (13.32 ± 5.82 secondary infections) compared to the full dynamic model ($p = 2.62 \times 10^{-15}$ using *t*-test), representing a 29.0% underestimation. The homogeneous environmental model, calibrated to reproduce the full model's dynamic potential and total outbreak size, produced nearly identical estimates (18.80 ± 0.80 , $p = 0.747$). The network graph of the various levels of intervention is shown in Figure 2.

D. Intervention effectiveness and model comparison across intervention levels

Progressive screening and isolation of index patients produced “dose-dependent” reductions in transmission as higher levels of interventions led to better reductions in infections. For the full dynamic model, at 25% intervention (isolating one of four index patients), secondary infections decreased to 14.79 ± 3.37 (21.2% reduction, 95% CI: 9–22). At 50% intervention, secondary infections fell to 10.48 ± 3.05 (44.2% reduction, 95% CI: 5–17). The 75% intervention level reduced further transmission to 5.61 ± 2.48 (70.1% reduction, 95% CI: 2–12).

Model comparison across intervention levels revealed important differences in estimated intervention efficacy. The static model significantly underestimated intervention benefits at 25% intervention (projecting 8.4% vs. 21.2% actual reduction, $p = 4.84 \times 10^{-5}$) and showed non-significant trends toward underestimation at higher levels (Table 4). The homogeneous model closely tracked full model estimates across all intervention levels ($p > 0.69$ for all comparisons).

E. Intervention impact on transmission heterogeneity

Increasing intervention levels progressively reduced both total transmission and the disproportionate contribution of highly connected individuals. At baseline, the top 10 superspreaders accounted for 80.3% of transmission events attributed to superspreaders, with Patient2 alone contributing 8.98% (898 events). At 75% intervention, the top 10 accounted for 57.5% of events, with Patient18 leading at 5.83% (583 events). The mean number of superspreaders per simulation decreased monotonically with intervention intensity: 1.39 ± 0.51 at baseline, 1.25 ± 0.48 at 25%, 1.15 ± 0.43 at 50%, and 1.00 ± 0.41 at 75% ($p < 0.001$). This progressive flattening of the transmission hierarchy demonstrates that screening and isolation not only reduce total epidemic size but also attenuate the disproportionate contribution

of highly infectious superspreading individuals to epidemic propagation.

F. Bootstrapped temporal exponential random graph model (bTERGM) analysis findings

Bootstrapped temporal exponential random graph modeling (bTERGM) with 100 replications revealed three fundamental mechanisms structuring hospital contact networks. The significant negative baseline edge term (OR = 0.062, 95% CI: 0.061–0.063; $p < 0.001$) confirmed the sparse nature of healthcare interactions, with only 6% of possible dyads forming contacts. Strong reciprocity (OR = 3.28, 95% CI: 3.11–3.42; $p < 0.001$) demonstrated that patient-provider and staff interactions are highly bidirectional, reflecting the mutual interactive nature of clinical care. Homophily by node type (OR = 1.54, 95% CI: 1.53–1.54; $p < 0.001$) showed that contacts are 54% more likely within professional groups (for instance, nurses with nurses and allied health staff with allied health staff) than between different groups, explaining the observed substantial modular network structure (modularity = 0.249).

The model diagnostics based on 100 bootstrap replications confirmed good model fit, with all observed network statistics including edge count, density, and reciprocity falling within the 95 percent confidence envelopes of networks simulated from the fitted model with *p* values of 1.00 for all statistics. The narrow bootstrap confidence intervals for all parameters indicate precise and stable estimation across replications. Furthermore, the close agreement between parameter estimates and their bootstrap means demonstrates excellent model stability with no evidence of misspecification or estimation error.

IV. DISCUSSION

This study provides a holistic investigation into the role of fomites, patients, and healthcare providers in the transmission dynamics of healthcare-associated infections (HAIs) in a hospital inpatient ward. Using a novel bootstrapped Temporal Exponential Random Graph Model (bTERGM) and network analysis, we identified key superspreaders and infection-causing individuals within the network of interactions, who were predominantly patients and healthcare professionals (nurses and allied health staff). This study further demonstrated how temporal dynamics influence HAI spread. While previous studies have employed static networks or focused solely on person-to-person interactions [4, 48], this study realistically integrates directed, weighted, and temporal network features while

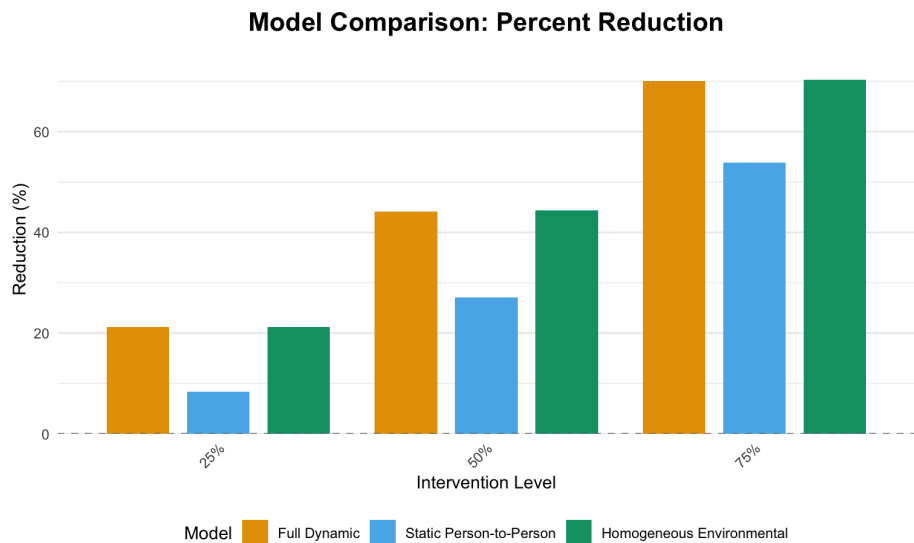


Fig. 4: Model comparison of the intervention effectiveness across models.

Intervention level	Model type	Mean secondary infections
0%	Full Dynamic	18.772
	Homogenous Environmental	18.800
	Patient	13.320
25%	Full Dynamic	14.792
	Homogenous Environmental	14.820
	Patient	12.200
50%	Full Dynamic	10.478
	Homogenous Environmental	10.450
	Patient	9.720
75%	Full Dynamic	5.611
	Homogenous Environmental	5.590
	Patient	6.140

Table 4: Intervention efficacy comparison across models.

incorporating fomites as active and central nodes in the transmission pathway.

Our bTERGM analysis revealed three fundamental mechanisms characterizing hospital contacts: strong reciprocity ($OR = 3.28$), homophily by node type ($OR = 1.54$), and sparse baseline connectivity ($OR = 0.062$), providing statistical evidence that hospital networks are organized by professional roles and bidirectional relationships rather than random interactions. By simulating shift-based dynamics and fixed patient-fomite interactions, the model reflects the operational and temporal complexity of real hospital environments and represents a significant advancement over earlier modelling efforts that often neglect these granular and structural realities [11, 13].

Our network structure was relatively sparse with

moderate clustering which is typical of a healthcare setting, and certain healthcare providers, particularly nurses, alongside patients and allied health workers, acted as central nodes with high potential for super-spreading. Importantly, we found that contact frequency alone is an insufficient predictor of outbreak contribution in hospital settings, especially in situations such as *C. difficile* transmission where patients serve as the primary infectious reservoir rather than healthcare workers who function predominantly as vectors rather than sources of infection, despite healthcare workers' central network positions. The bTERGM analysis (Table 5) further illuminated this heterogeneity through its demonstration of strong reciprocity ($OR = 3.28$), confirming that patient-provider interactions are highly bidirectional, which creates the structural pathways for

	Estimate	Mean	CI2.5%	CI97.5%
edges	-2.7757	-2.7768	-2.7868	-2.7630
Nodematch	0.4286	0.4288	0.4227	0.4330
.type				
mutuals	1.1872	1.1916	1.1339	1.2299

Table 5: The bTERGM analysis result.

transmission from patients to healthcare workers. This bidirectional movement allows healthcare workers to acquire *C. difficile* from patients and then transmit it to new vulnerable individuals, perpetuating the cycle despite patients being the primary source.

Comparing these results with other studies that have utilized network analysis reveals both similarities and distinctions. Previous studies [3, 48, 49] corroborate the findings of this research regarding the importance of identifying superspreaders and how patients and healthcare workers were more likely to be potential superspreaders. However, this study enhances the analysis by further incorporating fomite interactions and temporality, offering a more comprehensive view of HAI transmission dynamics.

These results align with previous studies highlighting the critical role of healthcare workers in HAI transmission [1, 6, 7, 50], but extend them by demonstrating that healthcare workers' role is primarily as conduits rather than sources as they acquire infections from patients and subsequently transmit to other patients and staff, amplifying rather than initiating transmission chains. The bTERGM finding of homophily by node type (OR = 1.54) indicates that while cross-type interactions (patient-nurse) are crucial for transmission, within-group clustering (nurse-nurse) also contributes significantly to network structure and transmission propagation. These findings suggest that aimed at controlling HAI transmission should prioritize "screening and isolating" infected patients and ensure healthcare workers with high centrality, such as nurses, take adequate precautions as they serve as crucial nodes in the HAI transmission pathways [1, 6, 12, 50]. Also, infection control efforts should be tailored to the specific dynamics of hospital shifts, with heightened vigilance during periods of higher staff and network activity. For instance, a study by [13] explored reorganization of nurse schedules which reduced the incidence of HAIs [13].

The network model demonstrates that targeted interventions significantly reduce both the number of superspreaders and HAI events. At 25% intervention (isolating one of four index patients), secondary infec-

tions decreased by 21.2%. And at 50% intervention, infections fell by 44.2%, and at 75% intervention, transmission was reduced by 70.1%, with outbreak extinction probability increasing to 0.60. These findings highlight the importance of early identification and isolation of high-risk individuals in healthcare settings. This progressive flattening of the transmission hierarchy demonstrates that screening and isolation not only reduce total cases but also attenuate the disproportionate contribution of highly infectious individuals to transmission propagation, making outbreaks more predictable and controllable. The drastic decline in transmission events with increased screening suggests that a threshold exists where interventions become highly effective in breaking transmission chains.

The 29.0% underestimation by the static model reflects its limitation to capture multi-generational transmission that unfolds sequentially in real contact networks [31, 51]. Collapsing dynamic networks into static aggregates assumes all contacts are simultaneously available, creating an artificially dense network that obscures the temporal ordering of actual interactions [52, 53]. Temporal networks preserve this evolving sequence, enabling pathogens to propagate through successive generations of susceptible individuals, which is a process static approximations cannot reproduce [31, 54]. Dynamic networks consistently yield larger outbreak sizes than static counterparts, particularly for pathogens with low transmissibility and prolonged infectious periods, characteristics consistent with the HAI pathogen *C. difficile* modeled in this study [55, 56]. Static models therefore misrepresent the burst-like, sequential nature of human interactions that drives true transmission dynamics [31, 51].

V. STRENGTHS AND LIMITATIONS

A key strength of this study lies in its use of a dynamic network model to capture the temporal evolution of hospital interactions and infection transmission. By applying bTERGM with 100+ stochastic simulations, we were able to adequately quantify uncertainty in network structure and superspreader identification, providing statistical reliability absent from single-realization studies. The inclusion of both healthcare workers and fomites as discrete, temporally-varying nodes represents a significant advancement over models treating fomites as a single homogeneous environmental compartments or excluding fomites entirely.

However, the following limitations must be acknowledged when interpreting these findings. One limitation is the assumption of uniform contact probabilities

within node types, which simplifies the model but may not capture the full complexity of individual interactions. In reality, there may be considerable variability in contact frequency and intensity, even within the same category of healthcare worker or patient.

Also, this model is most appropriate for contact-transmitted pathogens like *C. difficile* and MRSA, where transmission requires direct contact with contaminated individuals or surfaces. It may not well capture airborne pathogens (e.g., tuberculosis, measles) where aerosol transmission routes dominate. The specific fomites included (bedrails, bedside tables, laundry carts) were selected based on systematic review evidence identifying these as the most frequently contaminated high-touch surfaces in inpatient wards [12], hence the model's predictions regarding fomites transmission should be interpreted as a lower bound and limits its generalizability to other healthcare settings needs to be further evaluated.

Additionally, while the model includes a fixed hospital ward layout with static node composition, real hospital settings are more dynamic, with patient discharges and admissions. However, our bTERGM model was able to capture important elements such as staff rotations and the nuances in the interaction patterns during day and night shifts based on literature review and earlier publications [20].

VI. CONCLUSION

This study provides quantitative evidence from 40,000 total simulations highlighting the complex interplay between fomites, patients, and healthcare providers in the transmission of HAIs within a hospital ward. By employing bTERGM and epidemic modelling techniques and focusing on the identification of superspreaders, the findings provide valuable insights that can inform targeted HAI control strategies.

This study found that nurses and patients were more likely to be superspreaders and it also demonstrates that targeted screening and isolation of infected (high-risk) patients can achieve substantial reductions in *C. difficile* infections, with 75% "screening and isolation" intervention reducing transmission by 70.1% while simultaneously flattening the transmission hierarchy. This suggests that infection control efforts should prioritize early identification and isolation of infectious patients rather than focusing solely on highly connected healthcare workers.

These findings provide an evidence-based framework for optimizing resource allocation in infection prevention, suggesting that combination strategies targeting

both patient screening and the structured patterns of healthcare worker-patient-fomite interactions will be most effective in mitigating HAI outbreak risks.

AUTHOR CONTRIBUTIONS

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	Average daily contact/ contact episode ratio calculated	Estimated probability	Reference or explanations
Nurses to patients	8.4	0.56	[20]
Physicians to patients	11.4	0.76	[20]
Porter to patient	12.8	0.85	[20]
Porter to nurses	4.1	0.27	[20]
Nurses to nurses	5.3	0.35	[20]
Physician to nurses	12.0	0.80	[20]
Physician to physician	0.2	0.01	[20]
Patient to patient	5.1	0.34	[20]
Patient to bedrail	–	1.00	Estimated
Patient to physician	0.1	0.01	[20]
Patient to nurse	6.2	0.41	[20]
Nurse to bedrail	4.1	0.27	[57]
Doctor to bedrail	0.9	0.06	[57]
Porter to laundry cart	–	1.00	Estimated
Porter to physician	0.2	0.01	[20]
Porter to bedrail	2.5	0.17	[57]
Physician to porter	0.4	0.03	[20]
Patient to porter	0.2	0.01	[20]
Nurse to porter	0.1	0.01	[20]
Patients to patients	5.1	0.34	[20]
Nurse to physician	0.1	0.01	[20]
Porter to porter	1.5	0.10	[20]
AHS to patient	9.2	0.61	[20]
Patient to AHS	0.5	0.03	[20]
AHS to nurse	4.7	0.31	[20]
Nurse to AHS	0.5	0.03	[20]
AHS to physician	0.1	0.01	[20]
Physician to AHS	0.8	0.05	[20]
AHS to porter	0.1	0.01	[20]
Porter to AHS	0.6	0.04	[20]
AHS to bedrail	0.7	0.05	[57]
AHS to bedside table	0.5	0.03	[57]
Physician to bedside table	1.5	0.10	[57]
Nurse to bedside table	3.9	0.26	[57]
Porter to bedside table	1.8	0.12	[57]
Patient to bedside table	0.8	0.05	[57]

Table 6: Parameter estimation table and appropriate references.

	Duration (min)	Estimated weight	Reference or explanations
Nurses to patients	24.2	2.4	[20]
Physicians to patients	5.1	0.5	[20]
Porter to patient	2.1	0.2	[20]
Porter to nurses	2.1	0.2	[20]
Nurses to nurses	17.4	1.7	[20]
Physician to nurses	7.5	0.8	[20]
Physician to physician	4.8	0.5	[20]
Patient to patient	113.8	11.3	[20]
Patient to bedrail	–	4	Estimated
Patient to physician	5.3	0.5	[20]
Patient to nurse	15.1	1.5	[20]
Nurse to bedrail	–	1	[57]
Doctor to bedrail	–	1	[57]
Porter to laundry cart	–	4	Estimated
Porter to physician	1	0.1	[20]
Porter to bedrail	–	1	[57]
Physician to porter	1	0.1	[20]
Patient to porter	2.1	0.2	[20]
Nurse to porter	2.1	0.2	[20]
Nurse to physician	6.6	0.7	[20]
Patient to bedrail	–	4	Estimated
Porter to porter	24.8	2.4	[20]
AHS to patient	47.5	4.7	[20]
Patient to AHS	38.3	3.8	[20]
AHS to nurse	21.3	2.1	[20]
Nurse to AHS	23.4	2.3	[20]
AHS to physician	1.7	0.2	[20]
Physician to AHS	2.8	0.3	[20]
AHS to bedrail	–	1	Estimated
AHS to bedside table	–	1	Estimated
Physician to bedside table	–	1	Estimated
Patient to bedside table	–	1	Estimated
Nurse to bedside table	–	1	Estimated
Porter to bedside table	–	1	Estimated
AHS to porter	1.4	0.1	[20]
Porter to AHS	2.4	0.2	[20]

Table 7: Parameters for estimating edge weights (contact duration).