

# Final Report: Quantifying Norovirus Intervention Impact in Restaurant-Associated Outbreaks

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## Abstract

We evaluate two feasible restaurant norovirus control policies: symptomatic food-worker exclusion and enhanced hygiene. A stochastic, discrete-time, SEIR-like transmission model tailored to restaurant operations is calibrated to CDC NORS outbreak-size data using grid search and cross-validation. The calibrated model simulates sixteen scenarios spanning policy type and compliance. Outcomes include expected outbreak size, percentile reductions, and infections averted. Results provide data-anchored, decision-relevant estimates of the benefits and limits of exclusion and hygiene interventions in restaurant settings. Strict hygiene produced the largest outbreak reductions, cutting outbreak size by up to half when fully compliant. In contrast, moderate hygiene and exclusion alone provided only modest and comparable benefits. The strongest impact occurred when strict hygiene was combined with worker exclusion, particularly in reducing the risk of large outbreaks.

## Keywords

Norovirus, Restaurant Outbreaks, SEIR Model, Staff–Patron Transmission

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## 1 Introduction

Restaurants are compact, high-contact settings where a single norovirus case can rapidly spread. Norovirus causes acute gastroenteritis, requires only a small infectious dose, and persists in the environment, so one ill worker or a few exposed diners can trigger many secondary cases. Although policies on illness exclusion, hand hygiene, and glove use exist, compliance is uneven and turnover is high.

Control measures focus on who works and how contact occurs—keeping infectious workers home, strengthening hand-washing and glove protocols, and limiting cross-station contact to interrupt transmission links between staff and patrons. Most norovirus models assume homogeneous mixing, offering little insight into restaurant-scale dynamics shaped by shifts, table turnover, and staff–patron interactions.

## 1.1 Problem Definition

We ask two questions. First, what is the impact on transmission if sick workers remain home for 48 hours after symptom resolution, consistent with public health guidance. Second, what is the impact if restaurants improve hand hygiene and glove use. By centering these questions, the study aims to provide decision-relevant estimates of the benefits and limits of illness exclusion and hand hygiene controls in restaurant environments.

## 2 Survey on Relevant Prior Work

The previous work we review here is helpful in our attempt to quantify the impact of our focal interventions on norovirus prevalence in restaurants across the U.S. Many models estimate the reproductive number,  $R_0$ , but few of them show outbreak outcomes from their suggested restaurant policies. We will organize our review into transmission modeling foundations, restaurant policies and outcomes, and anchors and complements.

A fundamental value that governs the stability of an epidemic is the reproductive number,  $R_0$ . This value determines if a disease outbreak will explode into an epidemic or if it will die out, assuming a population that begins with all individuals as susceptible. Knowing the reproductive number for a disease improves our ability to model the spread of the disease, and interventions can be introduced earlier and more efficiently. [1] provides a linear algebra procedure to calculate  $R_0$  given a compartmental model of the disease spread. The procedure begins with converting the differential equations that define the model into a Jacobian matrix that will be decomposed. From there, up to three next-generation matrices (NGMs) can be calculated, each of them having the characteristic that its spectral radius produces the same  $R_0$ . The three NGMs are of different sizes, so the benefit of this procedure lies in its ability to produce a matrix whose size can be reduced for complex models. The computation is also relatively simple since the algorithm involves an intuitive calculation of a matrix inverse. However, this calculation is only useful for  $R_0$  and not  $R_e$ , the effective reproductive number. It is also limited to a community-level investigation, whereas [2] provides a dual-level evaluation of an epidemic. This paper considers the small outbreak case in which stochasticity is important and the community-level epidemic case in which the spread can be modeled deterministically. Because diseases behave differently at these two scales, the approach in [2] can be helpful in fully understanding the behavior of a disease. The paper also considers asymptomatic spread, which can be overlooked in simple models. Though this work did not focus on policy evaluations, we can use their approaches in congruence with other work that informs policy design.

[5] takes a direct approach to form epidemiological outcomes. By connecting restaurant policy data (NEARS) with epidemiological count and timing data (FDOSS), the paper studies restaurant-related norovirus outbreaks from 2014 to 2016. Their policy predictors included glove-use, cleaning policies, and ill-worker policies, which will match the predictors we investigate. Understandably, [5] is not able to find a causal relationship between the policies and the outcomes, and there is no guarantee that policy implementation implies policy adherence, but this work is a good baseline direction for our work. Their hygiene and ill-worker policies are detailed and contain a parameter space that can be usefully manipulated for our data.

We focus on these two policies specifically because food handler contact with food in restaurants is the leading point of contamination (POC) for norovirus [4]. The secondary POC is at the food production and processing stage, but the prevalence of this POC among norovirus outbreaks is lower. However, we see in [8] that the same policies applied to farm and food production workers can reduce norovirus incidence. Along with the potency of food handler contact, [4] also finds that restaurants/delis are the most commonly reported settings of norovirus outbreaks. The paper recommends proper glove usage, handwashing, and ill-worker management as interventions to prevent norovirus outbreaks. There has been some work verifying the effectiveness of these policies on managing rises in norovirus cases. [11] found a tremendous decrease in the expected number of norovirus cases if sick workers stayed home and waited 48 hours after recovery to return to work. They examined a community-level population across the U.S. and found the policy implementations to be successful. Although our work will focus on the micro scale in restaurants, the methodology of [11] is similar to our proposed approach.

In general, this previous work provides important insight into the policy implementations that can curb norovirus outbreaks. These authors have made great progress in measuring the impact of these interventions on a large scale.

### 3 Proposed Research Direction

While previous work has looked at these policies at the national scale or in farm settings, there is little modeling at the restaurant-outbreak level, where most real transmission occurs. We aim to model on a small restaurant scale to understand how policies can influence the size restaurant-associated norovirus outbreaks.

We model transmission using a stochastic mechanistic SEIR-like process with two interacting populations: restaurant staff and daily patrons. Transmission occurs through (i) staff-to-staff contact, (ii) staff-to-patron exposure during food handling and service, and (iii) occasional food-contamination events. Restaurant characteristics (e.g., number of food handlers, patron volume, initial infections) are sampled stochastically each simulation to reflect realistic variability in venue operations.

To anchor the model to real-world behavior, transmission parameters are calibrated to the empirical distribution of outbreak sizes from restaurant-associated norovirus outbreaks reported to CDC NORS. After calibration, sixteen intervention scenarios, varying policy type and compliance, are simulated to assess changes in

expected outbreak size, reductions across percentiles of the distribution, and the degree of synergy when both policies are implemented together.

## 4 Methods

We plan to achieve our research goals by combining outbreak data from the CDC's National Outbreak Reporting System (NORS) with a stochastic SEIR-type model.

### 4.1 Data Collection and Processing

The primary dataset used in this study is the CDC National Outbreak Reporting System (NORS), restricted to restaurant-associated norovirus outbreaks. Filtering criteria applied to the raw NORS export were: Etiology = norovirus (confirmed or suspected), Setting = restaurant/food service (all public restaurant subtypes), Primary Mode = foodborne, and Years = 2011–2023. The initial filtered file contained 2,180 outbreaks. The FDA Food Code's 48-hour exclusion rule for symptomatic food workers provides the regulatory foundation for several intervention scenarios evaluated in this work.

Additional cleaning steps removed banquet facilities and outbreaks with unresolved or incompatible etiologies, resulting in a cleaned dataset of approximately 1,980 restaurant-associated norovirus outbreaks. Because the mechanistic model is calibrated solely to outbreak size, we used only the number of illnesses per outbreak for all parameter estimation and validation procedures.

All modeling and analysis code is implemented in Python and organized within the `src/` directory, which contains the outbreak simulator, calibration workflow, validation procedures, and the full policy analysis pipeline (Figures 1–5). The final cleaned NORS dataset used for calibration (`NORS_JS1.csv`) is stored directly in `src/`. All output generated by the pipeline, including summary tables and policy figures, is saved in the top-level `results/` directory. Documentation materials, such as the final report and poster, are maintained in the `doc/` directory.

### 4.2 Model Structure

We implemented a stochastic, discrete-time model where the system updates in time steps of one day per iteration. Inside each simulated day, the following processes occur in order: (i) staff progress through disease states, (ii) staff infect each other, (iii) lunch-shift infections occur, (iv) dinner-shift infections occur, and (v) contamination events are evaluated. This framework follows the class of discrete-time Monte Carlo epidemic simulations widely used for modeling foodborne illness transmission [2, 9].

**4.2.1 Two Populations.** The model contains two interconnected populations: staff and patrons. Staff are modeled explicitly as individual agents. Patrons are not modeled individually; instead, patrons are treated as a continuous inflow population because they arrive and leave quickly and do not remain long enough to progress through disease states [6]. We only simulate how many patrons become infected during their visit.

**4.2.2 Staff Disease Progression.** Staff disease progression is modeled as a simplified SEIR-like process. Each staff member is assigned: (1) a disease state; (2) an infection day; and (3) an identity indicating whether they are a food-handler or a non-handler. The model

uses Monte Carlo sampling to simulate stochastic outcomes and is calibrated to outbreak-size data. The disease states are as follows:

- **Susceptible ( $S_s$ ):** healthy and can become infected.
- **Exposed ( $E_s$ ):** infected but not yet infectious; latent period is fixed at 1 day, approximating a 1.5-day norovirus incubation period [6].
- **Asymptomatic infectious ( $I_{a,s}$ ):** infectious and able to transmit disease. Asymptomatic shedding is well-documented in food-service norovirus settings [9].
- **Symptomatic infectious ( $I_{s,s}$ ):** highly infectious, but to represent partial absence from work due to symptoms, we apply a 50% reduction in transmission probability, consistent with behavioral assumptions used in norovirus workplace models [2].
- **Recovered ( $R_s$ ):** no longer infectious.

Transitions are deterministic based on time since infection (not rate-based):

$$S_s \xrightarrow{\text{infection}} E_s \xrightarrow{1 \text{ day}} \begin{cases} I_{a,s} & 3 \text{ days} \\ I_{s,s} & \end{cases} \xrightarrow{} R_s. \quad (1)$$

Equation 1 shows the transitions followed by each infected staff member.

### 4.3 Transmission Processes

Transmission occurs through three primary pathways: (1) staff-to-staff transmission, (2) staff-to-patron transmission, and (3) food contamination events.

**4.3.1 Staff-to-Staff Transmission.** Staff-to-staff transmission is modeled using daily Bernoulli trials. Each infectious staff member interacts with every susceptible staff member once per simulated day, providing a single opportunity for transmission. A Bernoulli trial with probability  $\beta_{ss}$  determines whether transmission occurs [7]:

$$P(\text{staff infected by another staff}) = \beta_{ss}. \quad (2)$$

**4.3.2 Staff-to-Patron Transmission.** Staff-to-patron transmission is modeled using binomial processes representing exposure during service interactions [3, 9]. This pathway is divided into two components based on the role of the staff.

(a) *Food Handler to Patron.* Food handlers directly serve patrons. Each served patron is treated as an independent Bernoulli trial with infection probability  $\beta_{\text{handler\_patron}}$ . If a handler serves  $n$  patrons per shift, new patron infections follow:

$$\text{New patron infections} \sim \text{Binomial}(n, \beta_{\text{handler\_patron}}). \quad (3)$$

(b) *Non-Handler to Patron.* Non-handlers do not directly serve patrons, so their transmission probability is lower. Each patron has infection probability:

$$p = \beta_{\text{other\_patron}} \times (\text{number of infectious non-handlers}), \quad (4)$$

and total infections per shift are drawn from:

$$\text{New patron infections} \sim \text{Binomial}(\text{total patrons per shift}, p). \quad (5)$$

**4.3.3 Food Contamination (Superspreading).** Food contamination events are modeled using a two-stage superspreading process, following the frameworks in [2, 9].

Food contamination is modeled using a two-step stochastic process following the superspreading frameworks described in [2, 9].

**Step 1: Bernoulli Trigger.** A contamination event can occur only when at least one infectious food handler is present. A Bernoulli trial with probability  $\text{prob\_food\_contamination}$  determines whether contamination occurs:

$$\text{Contamination event} \sim \text{Bernoulli}(\text{prob\_food\_contamination}). \quad (6)$$

**Step 2: Lognormal Superspreading Event.** If a contamination event occurs, the number of infected patrons is sampled from a lognormal distribution:

$$\text{Infections} \sim \text{Lognormal}(\mu, \sigma). \quad (7)$$

The parameters  $(\mu, \sigma)$  are chosen to recreate the characteristically heavy-tailed distribution of foodborne norovirus outbreaks, in which most events are small but rare superspreading events infect dozens of individuals [2, 9].

### 4.4 Calibration Framework

Before evaluating policy interventions, we calibrated the stochastic norovirus transmission model to real outbreak data from the CDC NORS surveillance system. Calibration ensures that the underlying model reproduces empirical outbreak size distributions under baseline (no-intervention) conditions, so that subsequent policy analyses reflect realistic transmission dynamics.

**Table 1: Fixed model parameters used in the stochastic simulation.**

Parameter	Value / Description
Latent period ( $\ell$ )	1 day
Infectious period ( $d_I$ )	3 days
Probability symptomatic ( $p_{\text{sym}}$ )	0.7
Symptomatic reduction	50% reduction in contacts
Simulation duration	5 days
Shifts per day	2 (lunch, dinner)
Patrons per handler	30
Patrons per shift	100, 125, 150, 175, 200
Initial infected staff	1, 2, or 3
Food handlers	3–7
Non-handlers	3–6
$\beta_{\text{other\_patron}}$	0.001

Our calibration framework follows a three-step procedure:

(i) *K-fold Validation.* We first performed a reduced-parameter grid search using 200 stochastic simulations per parameter set within a five-fold cross-validation scheme. Stratification was implemented by assigning outbreaks to percentile-based bins and constructing folds by sampling proportionally from each bin to ensure balanced representation across small and large outbreaks,

typical of norovirus outbreak datasets. For each training set, we simulated outbreaks across a reduced parameter grid and estimated model-data mismatch using our scoring function. This step identifies parameter regions with consistently low error and checks for overfitting by comparing train vs. test scores across folds.

(ii) *Holdout Validation*. We then conducted a full grid search on a random 80/20 split of the full dataset. A wider grid of transmission and contamination parameters was evaluated using 300 stochastic simulations per grid point. The best-performing parameters on the training set were validated against the held-out test set to confirm generalization.

(iii) *Full Calibration*. Finally, we used the entire dataset to perform a final, high-resolution grid search using 500 simulations per grid point. The parameter set minimizing the scoring function was selected as the calibrated baseline model used for all subsequent analyses.

*Scoring Function.* Model fit was assessed using a weighted percentile distance metric comparing simulated and observed outbreak size distributions. Specifically, we computed the weighted average absolute difference across the 10th, 25th, 50th, 75th, 90th, 95th, and 99th percentiles, with larger weights placed on the upper tail to capture large-outbreak behavior.

**Table 2: Parameter ranges explored during calibration.**

Parameter	Grid Range
$\beta_{\text{handler\_patron}}$	0.015–0.035 (5 pts fast; 8 pts full)
$\beta_{\text{staff\_staff}}$	{0.01, 0.05, 0.1, 0.2} (fast) {0.01, 0.03, 0.05, 0.1, 0.2} (full)
$\beta_{\text{other\_patron}}$	Fixed at 0.001
prob_food_contamination	0.10–0.22 (4–6 pts)
contamination_size_mean	35–65 (3–5 pts)
contamination_size_std	30 (fixed)
Simulations / grid point	200 (K-fold) 300 (Holdout) 500 (Full)

*Output of Calibration.* The optimal parameter set from Step 3 produced simulated outbreak distributions closely matching the empirical NORS data, both in central tendency and in tail behavior. This calibrated model serves as the baseline transmission process upon which all policy interventions (exclusion, hygiene, and combined strategies) are applied.

**Table 3: Final calibrated baseline parameter set**

Parameter	Estimated Value
$\beta_{\text{handler\_patron}}$	0.0293
$\beta_{\text{staff\_staff}}$	0.05
prob_food_contamination	0.10
contamination_size_mean	35
contamination_size_std	30
Calibration score	9.655

## 4.5 Policy Implementation

We implemented two operational policy levers within the calibrated stochastic norovirus simulator: (i) exclusion of symptomatic workers and (ii) hygiene measures that reduce transmission. Both policies act directly on the underlying transmission parameters of the restaurant simulation, without modifying the natural history of infection. All policy logic is encoded in the function `simulate_outbreak_policy`, which governs day-level staff interactions, symptom progression, and patron exposures.

The simulator models a typical U.S. restaurant with 6–12 staff and patron volumes drawn from a discrete distribution of 100–200 patrons per shift. For each simulated day, the model updates worker infection states and draws new patron infections due to (i) handler-to-patron exposure, (ii) non-handler-to-patron exposure, (iii) staff-to-staff spread, and (iv) food contamination events. Policies modify one or more of these pathways based on compliance.

*Baseline Symptomatic Absenteeism.* Even in the absence of any policy, symptomatic workers already have a 50% probability of not working each day. This baseline limitation reflects realistic illness-related absenteeism and applies before any policy-driven exclusion. All policy effects therefore layer on top of this baseline 50% reduction.

*Symptomatic Worker Exclusion.* Exclusion is applied only to workers in the symptomatic infectious state. Each day, symptomatic workers undergo a Bernoulli trial with success probability

$$\xi_{\text{eff}} = c \xi_{\text{max}},$$

where  $\xi_{\text{max}} = 0.4 \text{ day}^{-1}$  is the maximum additional exclusion probability and  $c$  is the compliance level. Excluded workers are removed from all transmission routes for that day (staff-to-staff, handler-to-patron, and contamination events). This exclusion is *additive* to the baseline 50% symptomatic absenteeism. On subsequent days, excluded workers return to work with probability  $\omega = 0.2 \text{ day}^{-1}$ , at which point they transition directly into the recovered state. This discrete implementation reflects realistic day-to-day staffing decisions rather than continuous compartment flows.

*Hygiene Improvements.* Hygiene policies reduce transmission probabilities across three pathways: staff-to-staff, handler-to-patron, and contamination events. The model computes a compliance-adjusted hygiene factor,

$$h = 1 - (1 - \beta_{\text{mult}}) c,$$

where  $\beta_{\text{mult}}$  depends on hygiene intensity. Two levels are implemented:

$$\beta_{\text{mult}} = 0.7 \quad (\text{moderate hygiene}), \quad \beta_{\text{mult}} = 0.4 \quad (\text{strict hygiene}).$$

After computing  $h$ , the simulator applies the following transformations:

$$\beta_{ss}^{\text{eff}} = h \beta_{ss}, \quad \beta_{hp}^{\text{eff}} = h \beta_{hp}, \quad p_{\text{contam}}^{\text{eff}} = h p_{\text{contam}}.$$

This formulation ensures that stronger hygiene measures or higher compliance proportionally reduce all relevant modes of transmission, while preserving stochasticity in event-level draws.

*Compliance.* Compliance  $c$  modulates both exclusion and hygiene effects. Low compliance attenuates intervention strength (e.g., staff may not report symptoms or adhere to hygiene protocols), whereas full compliance enforces maximal exclusion and maximal hygiene improvements. Because both mechanisms are implemented as individual-level probabilities, the model naturally preserves heterogeneity across simulations even under the same compliance level.

**Table 4: Policy scenarios evaluated in the stochastic norovirus model.**

Scenario Group	Hygiene Level	Exclusion	Compliance
Baseline	None	None	None
Exclusion Only	None	Yes	0.30
	None	Yes	0.60
	None	Yes	1.00
Moderate Hygiene	Moderate	No	0.30
	Moderate	No	0.60
	Moderate	No	1.00
Strict Hygiene	Strict	No	0.30
	Strict	No	0.60
	Strict	No	1.00
Moderate + Exclusion	Moderate	Yes	0.30
	Moderate	Yes	0.60
	Moderate	Yes	1.00
Strict + Exclusion	Strict	Yes	0.30
	Strict	Yes	0.60
	Strict	Yes	1.00

*Scenario Structure.* We evaluate all policy combinations across three compliance levels ( $c = 0.30, 0.60, 1.00$ ), yielding a total of 16 scenarios: baseline (no intervention), exclusion-only (3 compliance levels), moderate hygiene (3 levels), strict hygiene (3 levels), moderate hygiene combined with exclusion (3 levels), and strict hygiene combined with exclusion (3 levels). The baseline applies no intervention. Exclusion-only scenarios modify only  $\xi_{\text{eff}}$ ; hygiene-only scenarios modify only the hygiene factor  $h$ ; and combined scenarios apply both interventions simultaneously. For each scenario, 1,500 independent stochastic simulations are generated, enabling estimation of mean and median outbreak sizes, percentile ranges, tail probabilities, and cases averted relative to baseline.

**Table 5: Policy parameters used in the stochastic norovirus simulation.**

Param.	Description	Value
$\xi_{\text{max}}$	Max. exclusion prob./day	0.4
$\omega$	Return-to-work prob./day	0.2
$\beta_{\text{mult}} (\text{mod.})$	Hygiene multiplier (mod.)	0.7
$\beta_{\text{mult}} (\text{str.})$	Hygiene multiplier (str.)	0.4
$c$	Compliance level	{0.3, 0.6, 1.0}
$h$	Hygiene factor	$1 - (1 - \beta_{\text{mult}})c$
$\xi_{\text{eff}}$	Effective exclusion	$c \xi_{\text{max}}$
$\beta_{\text{eff}}$	Hygiene-modified $\beta$	$h \beta$
$p_{\text{contam}}^{\text{eff}}$	Modified contam. prob.	$h p_{\text{contam}}$

## 5 Results

### 5.1 Experimental Questions and Testbed

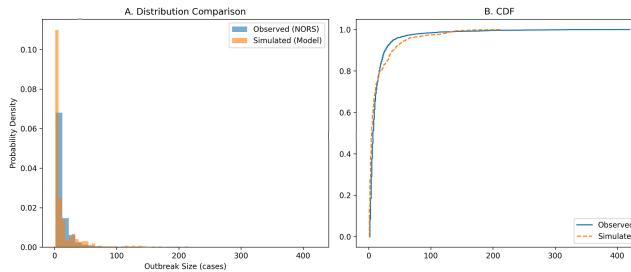
Our experiments were designed to answer the following core research questions:

- **RQ1: Calibration Validity** – How accurately does the discrete-time SEIR-type norovirus model reproduce the empirical NORS outbreak-size distribution after parameter calibration?
- **RQ2: Policy Effectiveness** – How do exclusion, hygiene, and combined interventions reduce outbreak size under compliance levels of 30%, 60%, and 100%?
- **RQ3: Compliance Dose–Response** – How strongly does increasing compliance reduce mean and upper-tail outbreak sizes across intervention types?
- **RQ4: Policy Interaction & Synergy** – Do exclusion and hygiene operate additively, or do combined interventions exhibit synergistic or sub-additive effects?
- **RQ5: Tail Risk Reduction** – How do interventions alter the probability of large outbreaks (e.g., >50, >100, >150 cases)?
- **RQ6: Cost-Effectiveness** – Which interventions yield the greatest outbreak reduction per unit implementation cost, and which strategies lie on the efficiency frontier?

**Testbed.** All experiments were conducted using our calibrated stochastic, discrete-time SEIR-type restaurant transmission model. The simulator includes staff and patron populations, food-handling and contamination pathways, and optional intervention modules (exclusion, hygiene, and combined policies). For each baseline and intervention scenario, we ran **1,500 stochastic simulations** to generate outbreak-size distributions. Policy intensity and compliance were varied across 16 total scenarios, enabling comparison of distributions, percentile reductions, upper-tail behavior, interaction effects, and cost-effectiveness.

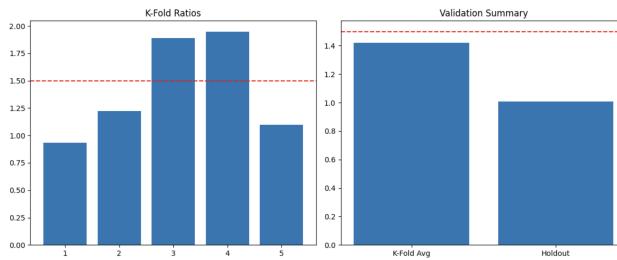
### 5.2 Model Calibration

The calibrated model closely matched the empirical NORS outbreak size distribution across central and upper percentiles. As shown in Figure 1, the histogram and CDF comparisons exhibited substantial overlap between observed and simulated outbreaks, indicating that the calibrated parameters accurately reproduced the baseline epidemic behavior. The QQ plot further demonstrated good agreement across most quantiles, with only minor deviations in the extreme upper tail. This was largely due to the right tailed nature of our dataset



**Figure 1: Comparison of observed NORS outbreak sizes and model-simulated outbreak sizes. (A) Histogram of outbreak size distributions showing close alignment across most of the range. (B) Empirical cumulative distribution functions (CDFs) demonstrating agreement between observed and simulated outbreak sizes, with minor deviations in the upper tail.**

Validation metrics supported the robustness of the calibration. The five K-fold validation ratios ranged from approximately 0.9 to 1.8, with a mean ratio near 1.4, indicating acceptable performance across training and validation subsets. The holdout validation ratio was 1.0, confirming generalizability to unseen data (Figure 2). Percentile comparisons showed minimal error in the 25th–75th percentiles and moderate error in the 95th and 99th percentiles, reflecting expected variability in rare large outbreaks.

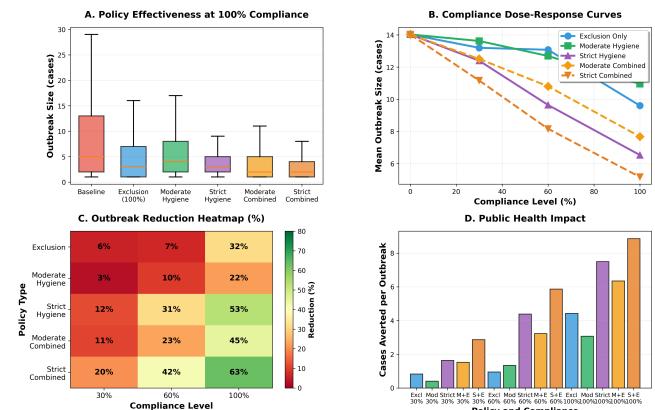


**Figure 2: K-fold validation ratios and holdout validation ratio for the calibrated model.**

Overall, the calibration framework produced a stable and well-fitting baseline model suitable for evaluating intervention policies in the subsequent analyses.

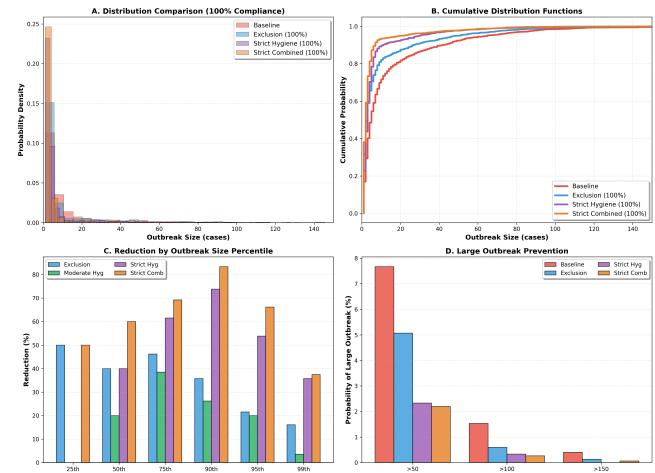
### 5.3 Policy Effectiveness Under Full Compliance

Under 100% compliance, all intervention policies reduced outbreak sizes relative to the baseline model. As shown in Figure 3, strict hygiene produced larger reductions in outbreak size than either moderate hygiene or exclusion-only strategies. The strict combined intervention (strict hygiene + exclusion) resulted in the smallest overall outbreak sizes among all policies, with a substantially compressed distribution compared to the baseline.



**Figure 3: Policy effectiveness under 100% compliance. Panels include (A) distribution comparison across policies, (B) compliance dose-response curves, (C) outbreak reduction heatmap, and (D) cases averted per outbreak.**

Differences between policies were apparent in both median and upper-tail outbreak sizes. Distributional shifts at 100% compliance (Figure 4) showed a marked leftward shift for strict hygiene and strict combined strategies, indicating higher probabilities of small outbreaks and reduced probabilities of larger ones. Cumulative distribution functions (Figure 4) similarly showed steeper rises for strict hygiene and strict combined policies, reflecting an increased proportion of outbreaks contained at small sizes.



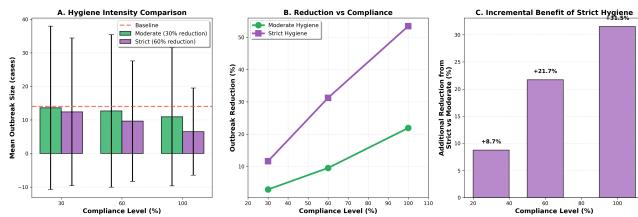
**Figure 4: Distributional comparisons under 100% compliance for baseline, exclusion-only, strict hygiene, and strict combined policies. Panels show distribution comparison (A), cumulative distribution functions (B), percentile reductions (C), and large-outbreak prevention (D).**

Upper-tail reductions were largest for strict combined hygiene. At the 75th, 90th, 95th, and 99th percentiles, strict combined hygiene produced the greatest decline relative to baseline, followed by strict hygiene alone. Exclusion-only and moderate hygiene showed

smaller reductions at these percentiles but still performed better than the baseline model.

#### 5.4 Compliance Dose–Response and Synergy

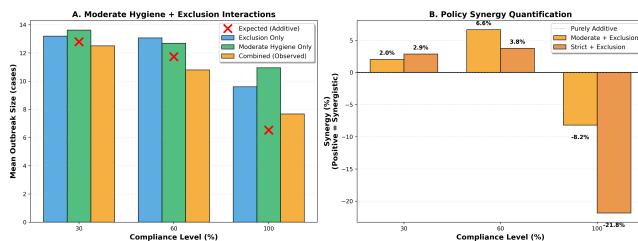
Across all intervention strategies, higher compliance levels produced progressively smaller outbreak sizes. As shown in Figure 5, strict hygiene exhibited the steepest dose–response relationship, with mean outbreak size declining substantially from 30% to 100% compliance. Moderate hygiene showed a similar but less pronounced pattern.



**Figure 5: Comparison of moderate and strict hygiene policies across compliance levels, including mean outbreak sizes (A), reduction vs. compliance (B), and incremental benefit of strict hygiene (C).**

Combined interventions consistently outperformed their individual components. In the policy interaction analysis (Figure 6), combined hygiene + exclusion strategies resulted in mean outbreak sizes below those expected from purely additive effects. This pattern was present at 30%, 60%, and 100% compliance and was most prominent under strict hygiene conditions.

Synergy quantification (Figure 6) further demonstrated that combined policies produced reductions greater than the sum of their individual contributions. Strict combined hygiene yielded the largest synergy values, indicating enhanced outbreak suppression when hygiene improvements and worker exclusion were implemented simultaneously.



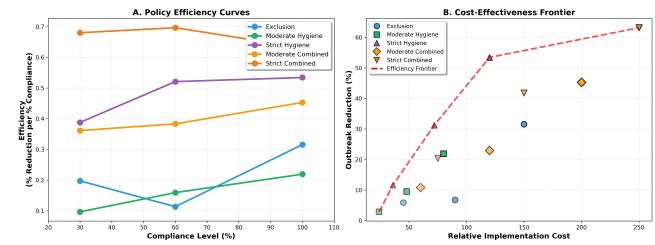
**Figure 6: Interaction effects between hygiene and exclusion. Panel (A) shows observed versus additive-expected outcomes for combined policies. Panel (B) quantifies synergy across compliance levels.**

#### 5.5 Cost-Effectiveness

Cost-effectiveness analysis demonstrated clear differences in how efficiently each intervention reduced outbreak sizes. As shown in Figure 7, strict hygiene and strict combined hygiene achieved the

largest outbreak reductions per unit of implementation cost. Moderate hygiene displayed intermediate efficiency across compliance levels, producing substantial reductions at lower cost than the strict interventions.

Exclusion-only policies produced the smallest reductions but also had the lowest associated implementation costs. Moderate combined hygiene occupied a middle position on the efficiency frontier, yielding notable outbreak reductions with a relatively modest cost increase. Across the evaluated strategies, strict combined hygiene achieved the highest absolute reduction in outbreak size, while moderate combined hygiene provided strong performance at lower cost.



**Figure 7: Cost-effectiveness analysis across intervention strategies. Panels illustrate reductions in mean outbreak size as a function of implementation cost and identify strategies lying on the efficiency frontier.**

## 6 Discussion

Our findings revealed several striking patterns in how exclusion and hygiene policies impact norovirus transmission in restaurant settings.

Strict hygiene was the only standalone policy that led to a significant reduction in outbreak size, specifically by 30–52% across compliance levels. However, moderate hygiene alone closely mirrored the performance of the exclusion-only policy. This shows that hygiene matters is only impactful when implemented in a strict manner. This pattern is well supported in the literature: norovirus transmission is highly sensitive to hand hygiene and environmental decontamination effectiveness, and partial hygiene improvements rarely achieve meaningful reductions [4? ? ? ]. Our results therefore align with established evidence that substantial reductions require strong, consistent hygiene practices.

The benefit of combined policy was largely driven by the hygiene component rather than exclusion. This was clearly seen when moderate hygiene + exclusion produced reductions nearly identical to strict hygiene alone, and strict hygiene + exclusion only modestly improved outcomes compared to strict hygiene alone. This indicates that exclusion adds only incremental benefit. Prior studies support this finding: while symptomatic exclusion helps, norovirus transmission is heavily driven by asymptomatic and pre-symptomatic handlers, meaning exclusion cannot achieve large reductions in outbreak sizes on its own [1? ? ]. Therefore, the combined effect shows functional synergy.

Strict hygiene also nearly eliminates large outbreaks. This was seen in the tail-risk plot which showed that strict hygiene and strict combined interventions reduced the probability of large outbreaks

(>50–150 cases) by 60–90%. This decrease in superspreading events is consistent with evidence that very large norovirus outbreaks are driven by food contamination and handler caused superspreading [2? ]. Strong hygiene directly disrupts these high-intensity transmission pathways.

Strict hygiene shifted the entire distribution of the outbreak by shifting the heavy right tail to the left, meanwhile moderate hygiene and exclusion produced minimal shifting of the tail. This aligns with the literature finding that minority of high-contamination events drive the majority of transmission [3? ]. Interventions that reduce contamination therefore produce the largest distributional shifts.

Transmission dynamics are also driven by handler-to-patron transmission rather than non-handler to patron, also consistent with literature. This finding is imputed from the modest reductions seen when symptomatic workers are removed from direct contact routes. This is largely because symptomatic workers are not the primary drivers of spread. Literature repeatedly shows that norovirus transmission is dominated by contaminated food, bare-hand contact, and environmental persistence, not by symptomatic attendance alone [4, 10]. In our model, strict hygiene proportionally reduced staff-to-staff, handler-to-patron, and contamination routes via the hygiene factor ( $h$ ), leading to the strongest reductions in outbreak size.

These results collectively address the study's research objectives. Close agreement between the simulated and CDC reported NORS outbreak-size percentiles served as a strong validation for our model's calibrated parameters (RQ1) Model Calibration (RQ1). Policy effectiveness (RQ2) showed strict hygiene as the dominant driver of outbreak reductions. Compliance dose-response (RQ3) was monotonic but modest for exclusion and moderate hygiene. Policy interaction (RQ4) revealed small, hygiene-driven synergy. Tail-risk reduction (RQ5) demonstrated dramatic suppression of superspreading under strict hygiene. Cost-effectiveness (RQ6) showed that strict hygiene lies on the efficiency frontier, while exclusion and moderate hygiene remain low-impact, low-efficiency strategies.

## 7 Conclusion & Future Work

This study demonstrates that hygiene-focused interventions are the most impactful and reliable strategies for reducing norovirus transmission in restaurant settings. Using a calibrated discrete-time SEIR-type model anchored to 1,980 NORS outbreak reports, we show that strict hygiene consistently produces the largest reductions in outbreak size, substantially compresses the upper tail, and dramatically lowers the probability of superspreading events. In contrast, symptomatic worker exclusion alone provides modest benefits, reflecting the limited role of symptomatic staff relative to asymptomatic shedding and food-contamination pathways. When combined, exclusion and hygiene exhibit modest functional synergy, but the majority of the benefit is driven by the hygiene component itself. Cost-effectiveness analysis further highlights strict hygiene as the dominant strategy, with moderate combined hygiene offering a strong mid-cost alternative. Together, these findings suggest that investment in rigorous handwashing, glove use, and food-handling practices yields the greatest return for preventing restaurant-associated norovirus outbreaks. Future extensions

incorporating environmental persistence, temporal outbreak data, and uncertainty quantification will help refine these estimates and strengthen the evidence base for restaurant-focused norovirus prevention policies.

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