# Epidemiology of infectious disease outbreaks on cruise ships: a systematic review and meta-analysis

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## Summary

**Background** Commercial cruise ships are high-density environments which may facilitate transmission of microbes responsible for infectious disease outbreaks. While acute gastroenteritis outbreaks are well-documented, emerging respiratory and zoonotic threats highlight the need for a comprehensive epidemiological understanding. We therefore undertook a systematic review and meta-analysis to evaluate infectious disease outbreaks on commercial cruise ships.

**Methods** In this systematic review and meta-analysis, we searched PubMed, Embase, Web of Science, and Scopus from inception to May 17, 2026, without language restrictions, alongside grey literature. We included observational studies reporting infectious disease outbreaks among passengers and/or crew. Quantitative outcomes (pooled attack risks, mortality risks, and case fatality proportions) were analysed using random-effects meta-analysis with logit transformation. Risk of bias was assessed using appropriate tools, and certainty of evidence was appraised by Grading of Recommendations Assessment, Development and Evaluation (GRADE). Heterogeneity was assessed using the $I^2$ statistic and Cochran's Q test, and publication bias was evaluated using funnel plots and Egger's test when at least 10 studies were included. This study is registered with the International Prospective Register of Systematic Reviews (PROSPERO), number CRD420261389422.

**Findings** We identified 8619 records and included 107 studies, yielding 619 outbreak event records from 236 distinct ships between 1969 and 2026. Acute gastroenteritis was the predominant clinical syndrome, and norovirus was the most frequently identified pathogen. Norovirus pooled attack risk was 6.5% (95% confidence interval [CI] 6.0–7.0) among passengers and 1.5% (95% CI 1.4–1.6) among crew members. Conversely, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) attack risk was 5.1% (95% CI 1.2–19.4) among passengers and 26.4% (95% CI 12.9–46.4) among crew. Influenza attack risks were comparable (6.5% among passengers and 7.3% among crew members; estimates were not age-specific). Mortality risks and case fatality proportions were generally low across pathogens, although *Legionella* outbreaks showed a higher pooled case fatality proportion of 11.4%. Norovirus passenger attack risk declined over time and was lower during the coronavirus disease 2019 (COVID-19) era than before 2020 (5.50% vs. 6.76%; p = 0.040). Considerable heterogeneity was observed in most pooled estimates ($I^2$ 60.4–99.8%). Egger's tests indicated potential small-study effects in the norovirus and influenza attack-risk analyses, but not in the evaluated SARS-CoV-2 analyses. Qualitative evidence indicated substantial variation in transmission dynamics across pathogen groups.

**Interpretation** Our findings show that although norovirus remains the leading cause of cruise-ship outbreaks, the epidemiological landscape has diversified to include respiratory viruses and other emerging pathogens. Distinct infection patterns between passengers and crew members highlight the need for pathogen-specific prevention strategies, strengthened surveillance, and targeted occupational protections for onboard personnel.

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Keywords: Cruise ships; Infectious disease outbreaks; Norovirus; SARS-CoV-2; Meta-analysis; Systematic review

## Research in context

### Evidence before this study

We searched PubMed, Embase, Web of Science, and Scopus from database inception to May 17, 2026, without language restrictions. Search terms combined concepts related to cruise ships and passenger vessels with a broad range of infectious diseases, including gastrointestinal, respiratory, foodborne, waterborne, and vaccine-preventable infections, as well as terms related to systematic reviews and meta-analyses. We identified several reviews examining infectious diseases in maritime travel settings. Existing reviews primarily focused on individual pathogens (such as norovirus and severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2]), foodborne or waterborne disease outbreaks, particular passenger-ship outbreak settings, or the impacts of infectious disease outbreaks on ports, port communities, and other land-based operations. A small number of narrative reviews summarised infectious disease events on passenger ships, but these studies generally did not quantitatively synthesise epidemiological outcomes across pathogens. We did not identify a systematic review and meta-analysis that applied a common framework to integrate outbreak-level epidemiological outcomes and available transmission parameters across pathogen categories in commercial cruise-ship settings.

### Added value of this study

To our knowledge, this review provides a standardised cross-pathogen synthesis of outbreak-level epidemiological outcomes and available transmission parameters in commercial cruise-ship settings. We synthesised evidence from 619 outbreak event records involving 236 ships over more than five decades. Using a common eligibility, extraction, and outcome framework, we provide a comparative overview of passenger- and crew-specific attack risks, mortality risks, case fatality proportions, and reported transmission parameters across pathogen categories, complementing pathogen-specific reviews. We found that gastrointestinal pathogens continued to account for a substantial proportion of reported cruise-ship outbreaks, while respiratory pathogens, particularly SARS-CoV-2, emerged as important contributors to outbreak burden. Distinct epidemiological patterns were observed between passengers and crew members, with higher attack risks generally reported among passengers during norovirus outbreaks and substantially higher attack risks among crew members during SARS-CoV-2 outbreaks. We also identified temporal changes in outbreak epidemiology, including lower passenger attack risks for norovirus after 2020 and the emergence of outbreaks involving a broader range of pathogens. In addition, we synthesised qualitative evidence on transmission dynamics, including basic reproduction numbers, secondary attack rates, incubation periods, serial intervals, and generation times, highlighting the considerable transmission potential of infectious diseases in semi-closed maritime environments.

### Implications of all the available evidence

Taken together, the available evidence indicates that infectious disease outbreaks on cruise ships are no longer confined primarily to acute gastroenteritis and increasingly involve a broader range of respiratory and other emerging pathogens. The distinct attack risk patterns observed between passengers and crew members suggest that outbreak prevention and response strategies should account for differences in exposure settings, living conditions, and occupational roles onboard. These findings support strengthening maritime public-health preparedness through pathogen-specific prevention and control approaches. For enteric pathogens, priority measures include enhanced environmental sanitation, food and water safety management, and early outbreak detection. For respiratory pathogens, improved ventilation, rapid case identification, isolation procedures, and surveillance systems may be particularly important. The higher pooled SARS-CoV-2 attack risk among crew members than passengers further highlights the need for targeted occupational health protections and dedicated monitoring programmes for ship personnel. At a broader public-health level, our findings support the development of standardised outbreak reporting frameworks and integrated surveillance systems, including the incorporation of emerging approaches such as onboard wastewater monitoring, to facilitate earlier detection, more timely interventions, and improved outbreak preparedness in the cruise industry.

## Introduction

The commercial cruise industry has seen significant growth in recent decades, with millions of passengers and crew members travelling annually. Cruise ships represent unique, semi-closed, and high-density environments where individuals from diverse geographical

backgrounds live and dine in close proximity. These conditions, combined with shared facilities and repeated passenger turnover, facilitate the transmission of infectious agents through person-to-person contact, contaminated food and/or water, and environmental reservoirs, potentially leading to infectious disease outbreaks.[1]

Historically, acute gastroenteritis, particularly norovirus, has been the most frequently reported cause of cruise ship outbreaks, continuing to account for a substantial proportion of documented incidents. Previous research has identified environmental contamination and consecutive voyage operations as key factors in sustaining these gastrointestinal outbreaks.[2] The US Centers for Disease Control and Prevention (CDC) Vessel Sanitation Program (VSP) conducts surveillance of acute gastroenteritis on cruise ships within its jurisdiction, providing an important source of surveillance evidence for this review.[3] However, alongside these persistent gastrointestinal threats, recent decades have seen an increase in respiratory outbreaks, including influenza and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which demonstrated the potential for these vessels to serve as amplification settings for emerging pathogens.[4–7] Furthermore, recent events, including the 2026 Andes hantavirus outbreak linked to an expedition cruise ship, highlight the emerging risks associated with zoonotic and ecological exposures during expedition travel.[8,9]

Despite the recurring nature of these events, previous reviews have primarily focused on single pathogens, including norovirus and SARS-CoV-2, foodborne or waterborne disease outbreaks, or particular passenger-ship outbreak settings.[2,10–13] A recent systematic review also examined the impacts of infectious disease outbreaks on large passenger ships on ports, port communities, and other land-based operations.[14] Nevertheless, a synthesis that applies a consistent outbreak-record framework across pathogen categories and integrates epidemiological outcomes with available transmission parameters remains limited. A broader understanding of the epidemiological characteristics of infectious disease outbreaks in shipboard environments is needed to strengthen public health preparedness and to inform integrated surveillance and response strategies.

Based on these research gaps, we conducted a systematic review and meta-analysis integrating quantitative and qualitative evidence to evaluate the epidemiology of infectious disease outbreaks on commercial cruise ships. This review provides a standardised cross-pathogen synthesis of outbreak-level epidemiological outcomes and available transmission parameters in commercial cruise-ship settings. By applying a common eligibility, extraction, and outcome framework, it complements pathogen-specific reviews with a comparative overview of passenger- and crew-specific attack risks, mortality risks, case fatality proportions, and reported transmission parameters across pathogen categories. Specifically, we aimed to: (1) estimate the pathogen-specific pooled attack risks, mortality risks, and case fatality proportions among passengers and crew members; (2) explore sources of between-outbreak heterogeneity, including temporal trends and methodological factors, using subgroup and meta-regression analyses; and (3) characterise key transmission parameters, such as reproduction numbers, secondary attack rates (SAR), and incubation periods, through a narrative synthesis of all eligible reports.

## Methods

We adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[15] The review protocol was registered in the International Prospective Register of Systematic Reviews (PROSPERO; CRD420261389422). The protocol is accessible via the PROSPERO record. No amendments were made to the information provided at registration or in the study protocol.

### Ethics

This systematic review and meta-analysis used aggregate data extracted from previously published studies and publicly available outbreak reports. As no identifiable individual-level data were collected and no participants were directly enrolled, ethical approval and written informed consent were not required.

### Search strategy and selection criteria

Two independent reviewers (SW and LZ) systematically searched Embase (from 1947), PubMed (from 1946), Web of Science Core Collection (from 1900), and Scopus (from 1966) to May 17, 2026, with no language restrictions. The search strategy combined Medical Subject Headings (MeSH) and free-text terms covering three domains: cruise ships and passenger vessels, general infectious diseases and outbreaks, and specific target pathogens (e.g., norovirus, *Salmonella* spp., *Legionella* spp., influenza, SARS-CoV-2, and measles). To ensure comprehensive coverage, we supplemented this with searches of grey literature and relevant public health sources, specifically the CDC VSP, World Health Organization (WHO) Disease Outbreak News and European Centre for Disease Prevention and Control (ECDC). Additionally, we performed both backward and forward citation searching ("snowballing") of the included studies and relevant systematic reviews to identify further eligible literature. Duplicates were removed using reference management software and by manual checking. The complete search strategies for all databases are provided in Supplementary Table S1. Study selection was conducted in two stages. Following

training in the prespecified eligibility criteria, including pilot screening with feedback from a senior reviewer, title and abstract records were divided among members of a multi-person screening team, with each record screened by one team member. Full-text eligibility was initially assessed by members of the screening team. During this revision, two reviewers (SW and LZ) independently rechecked the full-text eligibility decisions and exclusion reasons. Disagreements were resolved by discussion, and when consensus could not be reached, a third reviewer (JL) adjudicated. No automation tools or artificial intelligence-assisted methods were used for screening or study selection.

The main outcomes were outbreak-level epidemiological indicators, including attack risks and mortality risks for the overall, passenger, and crew populations, as well as case fatality proportions. For quantitative synthesis, we included observational and descriptive studies (e.g., outbreak reports, surveillance reports, cohort, case–control, cross-sectional, and modelling studies) that (1) documented infectious disease outbreaks among passengers and/or crew members on commercial cruise ships; (2) reported outcomes relevant to at least one prespecified main outcome; and (3) provided extractable or derivable data for meta-analysis. For narrative synthesis, we included eligible studies describing infectious disease outbreaks on commercial cruise ships to qualitatively summarise key transmission parameters, such as basic reproduction numbers ($R_0$), incubation periods, serial intervals, generation times, and SAR.

We excluded (1) non-human or animal-only studies; (2) outbreaks occurring on non-commercial vessels (e.g., military ships, cargo ships, fishing vessels, private yachts, or sailing boats) or solely in land-based settings and non-cruise transport (e.g., aircraft, trains, buses) unlinked to a commercial cruise voyage; (3) randomised controlled trials, reviews, editorials, commentaries, and conference abstracts without original data; and (4) reports without useable outcome data for synthesis.

### Data extraction

We conducted separate syntheses for quantitative and qualitative data. Two independent reviewers (SW and LZ) extracted data using a piloted standardised data extraction form, with discrepancies resolved by a third reviewer (JL). Extracted data were organised into four domains: (1) basic information, spatiotemporal details, and settings: literature and outbreak IDs, study design, ship name, route, voyage duration, and outbreak start and end times (2) pathogen and clinical characteristics: pathogen and disease names, case definitions, and laboratory confirmation methods; (3) human infections and population denominators: total ship capacity and actual onboard populations (stratified by overall, passengers, and crew), alongside corresponding case counts and deaths to derive attack risk, mortality risk, and case fatality proportion; and (4) transmission parameters and interventions: $R_0$, incubation period, serial interval, generation time, SAR, and implemented public health control measures.

For the quantitative synthesis, we extracted the exact number of infected cases or deaths and their specifically matched exposed populations on board. In instances of multiple reports describing the same outbreak, we retained the most complete source while extracting any unique, complementary information from the related reports to ensure data richness. Independent outbreaks reported within a single study were extracted as separate data entries when they involved different ships, distinguishable voyages, or clearly separate outbreak periods (Supplementary Table S2). For the narrative synthesis, we extracted the means of transmission parameters ($R_0$, incubation period, serial interval, and SAR), and if unavailable, medians and ranges were used. Additionally, we summarised pathogen profiles, spatiotemporal trends, and public health control measures.

A commercial cruise ship was defined as an ocean, river, or expedition passenger vessel. An onboard infectious disease outbreak was defined as an event occurring among passengers and/or crew members during, or directly linked to, a cruise voyage. We used the outbreak designation or surveillance definition reported by the source where available, including CDC VSP definitions for acute gastroenteritis events, and otherwise applied no minimum case threshold; single, isolated cases were not eligible (Supplementary Table S2).

Quantitative outcomes: The attack risk was defined as the number of infected or symptomatic individuals divided by the corresponding exposed population on board (overall, passengers, or crew members). The mortality risk was defined as the number of deaths divided by the corresponding exposed population on board. The case fatality proportion was defined as the number of deaths divided by the number of infected individuals.

### Critical appraisal

The methodological quality and risk of bias of included studies were assessed using standardised tools. Cohort and case–control studies were evaluated with the Newcastle-Ottawa Scale (NOS), which has a maximum of nine points across three domains: selection, comparability, and outcome/exposure.[16] For cross-sectional studies and case series, the Joanna Briggs Institute (JBI) Checklist was applied, which considers factors such as clear inclusion criteria, valid measurement of exposure and outcomes, and adequacy of follow-up.[17] Outbreak investigation or surveillance reports were assessed using a custom 7-item quality appraisal checklist, which evaluates case definition, ascertainment, denominators, laboratory confirmation,

data completeness, transmission routes, and control measures. Two reviewers (SW and LZ) independently evaluated study quality, and discrepancies were resolved through discussion or consultation with a third reviewer (JL). In addition, we assessed the certainty of evidence for pooled outcomes using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach across five domains: risk of bias, inconsistency, indirectness, imprecision, and publication bias.

### Statistical analysis

For the meta-analysis of proportions, pooled attack risks, mortality risks, and case fatality proportions were calculated for each outbreak based on our predefined operational definitions. When studies reported multiple nested case definitions, we extracted the combined count of both laboratory-confirmed and clinical/suspected cases to capture the overall outbreak burden. Alternative outcomes were considered in sensitivity analyses restricted to laboratory-confirmed cases. For denominators, we used the actual onboard population whenever available. For overall attack risk, reported totals were prioritised; when unavailable, totals were derived by summing passenger- and crew-specific values. Proportions were pooled using random-effects models with logit transformation, the inverse-variance method, restricted maximum-likelihood estimation of between-outbreak variance, and Hartung–Knapp adjustment with an ad hoc variance correction. Between-study heterogeneity was assessed using the $I^2$ statistic and Cochran's Q ($\chi^2$) test ($I^2 > 50\%$ or $p < 0.10$ indicating substantial heterogeneity). Primary meta-analyses were stratified *a priori* by distinct pathogen categories.

Sensitivity analyses were performed separately by (1) sequentially excluding individual studies (leave-one-out); (2) restricting data to records reporting laboratory confirmation methods; (3) excluding brief reports with limited descriptive information; (4) applying alternative proportion transformations (i.e., Freeman–Tukey double-arcsine and untransformed raw proportions); (5) utilising alternative between-study variance estimators (DerSimonian-Laird and Paule-Mandel); (6) excluding extreme sample sizes (<50 or >3000 persons on board); (7) excluding CDC VSP surveillance records; and (8) excluding explicitly identified grey literature. Two additional post-hoc sensitivity analyses were performed by (1) repeating all affected meta-analyses after event-level reconciliation of overlapping reports and (2) replacing multiple estimates from the same continuing-outbreak series with a single series-level contribution within each pathogen–outcome meta-analysis. Subgroup analyses were conducted according to coronavirus disease 2019 (COVID-19) era (pre-COVID-19 vs. COVID-19 era), route or region, and whether laboratory confirmation methods were reported; between-group differences were evaluated using a mixed-effects model (Cochran's Q-test). Exploratory post-hoc analyses examined norovirus overall attack risk by cruise route and calendar period (<2010 vs. ≥2010), while reported control measures were summarised descriptively. Meta-regression analyses were additionally performed to explore potential sources of between-outbreak heterogeneity using random-effects models fitted to logit-transformed proportions with restricted maximum-likelihood estimation and Hartung–Knapp adjustment. Individual moderators were first examined in univariable models; outbreak year, log-transformed denominator, and whether a laboratory confirmation method was reported were then included together in multivariable models to assess their associations after accounting for the other included moderators. Publication bias was evaluated using funnel plots and Egger's test when at least 10 studies were included in a meta-analysis.

Results were tabulated in evidence tables and displayed in forest plots. All statistical analyses were performed using R version 4.4.2 (packages meta, metafor, and ggplot 2), with two-sided p values < 0.05 considered statistically significant.

### Role of the funding source

This work was supported by the Prevention and Control of Emerging and Major Infectious Diseases-National Science and Technology Major Project (China). The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. All authors had full access to all the data in the study. JL had final responsibility for the decision to submit for publication.

## Results

We identified 8619 records and removed 3007 duplicates, leaving 5612 for screening; 5195 were excluded at title/abstract stage and 417 full texts were assessed. A total of 107 studies were included. From these, we extracted 619 outbreak event records, of which 458 were eligible for at least one quantitative outcome. The detailed study selection process is shown in Fig. 1 and detailed in Supplementary Table S3.

### Main characteristics of included studies

The main characteristics of the outbreak event records are summarised in Table 1, and a comprehensive line-listing of all included studies and individual outbreak records is provided in Supplementary Table S4. The aggregated characteristics categorised by pathogen are further detailed in Supplementary Table S5, comprising geographic regions, exposed populations, laboratory confirmation status, and implemented interventions. Reported laboratory confirmation was available for 19 of 21 SARS-CoV-2 records and 13 of 19 influenza records,

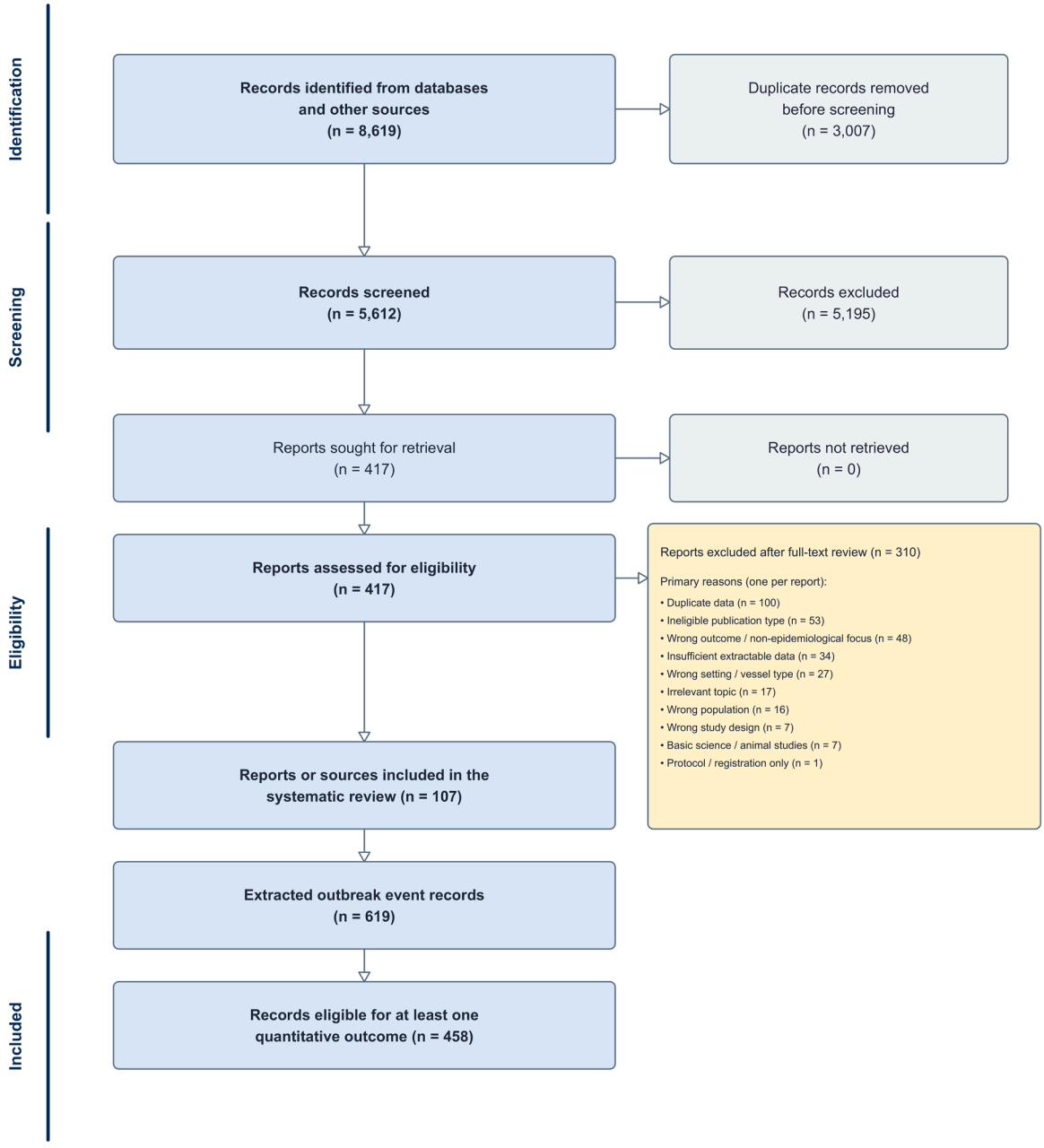


***Fig. 1:*** **PRISMA flow diagram of study selection.** Flow diagram showing record identification, duplicate removal, screening, full-text eligibility assessment, and inclusion in the systematic review and quantitative synthesis. The principal reasons for excluding 310 reports after full-text review are summarised; all full-text exclusions are listed in Supplementary Table S3. **Abbreviation:** PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

compared with 50 of 358 norovirus records (Supplementary Table S5). A descriptive summary of epidemiological outcome measures by pathogen category is provided in Supplementary Table S6. Overall, the 619 outbreak records occurred across 236 distinct ships between 1969 and 2026 (median year 2007, interquartile range [IQR] 2002–2015). The median total population on board was 1715 (IQR 924–2904). Of the 619 outbreak records, 432 (69.8%) involved gastrointestinal illness, the predominant clinical syndrome, and 358 (57.8%) involved norovirus, the most frequently identified pathogen. Enterotoxigenic *Escherichia coli* (ETEC) was identified in 31 records (5.0%), SARS-CoV-2 in 21 (3.4%), and influenza virus in 19 (3.1%),

| Characteristic | Value |
|---|---|
| Overall characteristics | |
| Included reports or data sources, No. | 107 |
| Extracted outbreak event records, No. | 619 |
| Distinct ships, No. | 236 |
| Outbreak year, median (IQR); range | 2007 (2002–2015); 1969–2026 |
| Records with unavailable outbreak year, No. (%) | 4 (0.6) |
| Data source of outbreak records, No. (%) | |
| Peer-reviewed publications | 194 (31.3) |
| CDC Vessel Sanitation Program (VSP) surveillance | 423 (68.3) |
| Other grey literature sources | 2 (0.3) |
| Population size | |
| Total persons on board, median (IQR); range | 1715 (924–2904); 9–7454 |
| Passengers on board, median (IQR); range | 1750 (954–2171); 0–8400 |
| Crew members on board, median (IQR); range | 788 (556–964); 2–3000 |
| Clinical disease group, No. (%) | |
| Gastrointestinal illness | 432 (69.8) |
| Other or unspecified clinical syndromes | 92 (14.9) |
| COVID-19 | 21 (3.4) |
| Respiratory viruses other than SARS-CoV-2 | 21 (3.4) |
| Vaccine-preventable diseases | 19 (3.1) |
| Specified foodborne or waterborne infections | 18 (2.9) |
| Legionellosis | 12 (1.9) |
| Other infectious diseases | 4 (0.6) |
| Major pathogens, No. (%) | |
| Norovirus | 358 (57.8) |
| Unknown or not laboratory confirmed | 108 (17.4) |
| Enterotoxigenic *Escherichia coli* (ETEC) | 31 (5.0) |
| SARS-CoV-2 | 21 (3.4) |
| Influenza virus | 19 (3.1) |
| Multiple pathogens | 15 (2.4) |
| Varicella-zoster virus | 13 (2.1) |
| *Legionella pneumophila* | 11 (1.8) |
| Other specified pathogens | 43 (6.9) |
| Availability of quantitative data, No. (%) | |
| Eligible for at least one quantitative outcome | 458 (74.0) |
| Narrative parameter-only records | 14 (2.3) |
| Descriptive-only records | 147 (23.7) |
| Reporting completeness, No. (%) | |
| Cruise region or route specified | 172 (27.8) |
| Laboratory confirmation method reported | 140 (22.6) |
| Reporting of outbreak response measures, No. (%) | |
| Records reporting at least one control measure | 145 (23.4) |

**Note:** Data are presented as No. (%) unless otherwise indicated. Percentages were calculated using the 619 extracted outbreak event records as the denominator. Continuous variables were calculated using records with available data. Multiple outbreak records reported in the same publication or data source were extracted separately. The most frequently represented pathogen labels within the "Respiratory viruses other than SARS-CoV-2" category were influenza A(H3N2), influenza B, influenza A(H1N1)pdm09, other or unspecified influenza A, and unspecified influenza virus. **Abbreviations:** CDC, Centers for Disease Control and Prevention; COVID-19, coronavirus disease 2019; ETEC, enterotoxigenic *Escherichia coli*; IQR, interquartile range; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; VSP, Vessel Sanitation Program.

***Table 1:*** **Summary characteristics of infectious disease outbreak event records on cruise ships.**

whereas 108 records (17.4%) lacked laboratory confirmation (Table 1). Supplementary Fig. S1 provides a visual overview of the relative distribution of clinical disease groups and pathogen categories. Quantitative data were available in 458 records (74.0%), and outbreak response measures were reported in 145 records (23.4%), which predominantly included isolation or quarantine, enhanced cleaning or disinfection, and screening or active surveillance (Table 1; Supplementary Table S5). At the event-record level, median passenger attack risks were higher among passengers than crew members for norovirus (5.73% vs. 1.34%), whereas the opposite pattern was observed for SARS-CoV-2 (4.31% vs. 25.1%) and influenza

(5.12% vs. 8.53%; Supplementary Table S6). Voyage-related timing information was incomplete and insufficiently standardised for quantitative analysis: outbreak duration was reported for 12 records and paired outbreak start and end dates for 74 records, but these measures were not used as proxies for scheduled voyage duration (Supplementary Table S7). Overall, the methodological quality of the included studies was generally acceptable across designs: most outbreak and surveillance reports demonstrated high reporting quality (custom 7-item checklist scores of 6–7), and observational studies predominantly exhibited moderate to low risk of bias (NOS scores of 5–8; 9 of 10 and 6 of 10 "Yes" ratings on the JBI case-series checklist; Supplementary Tables S8–S10). The certainty of evidence was very low for the attack-risk estimates and low for the Legionella case fatality proportion (Supplementary Table S11).

The temporal distribution of the outbreak event records is shown in Fig. 2. The annual number of reported outbreaks remained relatively low and sporadic before the mid-1990s, followed by a marked increase that peaked during the 2000s (Fig. 2A). Following a sharp decline in 2021, outbreak frequency rebounded rapidly in subsequent years. Pathogen composition shifted over time, although norovirus accounted for the largest proportion of overall outbreaks from 1995 onwards (Fig. 2B1). Exclusion of CDC VSP surveillance records revealed a more heterogeneous, multi-pathogen landscape, with the emergence of SARS-CoV-2 during 2020–2024 (Fig. 2B2).

### Pooled attack risks, mortality risks, and case fatality proportions across pathogen categories

Fig. 3 shows the pathogen-specific pooled attack risks in cruise ship outbreaks (GRADE: very low). Under a random-effects model, the pooled attack risk for norovirus was 6.5% (95% confidence interval [CI] 6.0–7.0%; k = 291) among passengers and 1.5% (95% CI 1.4–1.6%; k = 283) among crew members ($I^2$ = 98.5% and 88.0%, respectively). For SARS-CoV-2, the overall pooled attack risk was 20.3% (95% CI 10.3–36.3%; k = 17), with 5.1% (95% CI 1.2–19.4%; k = 8) among passengers and 26.4% (95% CI 12.9–46.4%; k = 11) among crew members ($I^2$ = 99.0%, 98.4%, and 98.4%, respectively). For influenza, the pooled attack risk was 6.5% (95% CI 3.2–13.0%; k = 11) among passengers and 7.3% (95% CI 4.8–11.0%; k = 9) among crew members ($I^2$ = 99.5% and 92.1%, respectively; mixed or unspecified age populations; age-specific data were not consistently reported). Event-level forest plots for overall, passenger, and crew attack risks are provided in Supplementary Figs. S2–S24, respectively. Pathogen-specific pooled mortality risks (Fig. 4) and case fatality proportions (Fig. 5) were generally low across most evaluated pathogens, with the exception of a higher case fatality proportion observed for *Legionella* (11.4%; 95% CI 4.5–25.7%; k = 9; $I^2$ = 9.4%); estimates for both metrics were based on a limited number of eligible outbreak records (k ≤ 9 per pathogen). Event-level forest plots for overall, passenger, and crew mortality risks, alongside case fatality proportions, are shown in Supplementary Figs. S25–S38, respectively.

Subgroup analyses and meta-regression were conducted to explore heterogeneity in pooled attack risks, primarily focusing on norovirus and other gastrointestinal infections. Subgroup analysis results are summarised in Table 2, Supplementary Tables S12 and S13. The findings from univariable and multivariable meta-regression models are presented in Supplementary Table S14 and Table 3, respectively. For norovirus, passenger attack risk declined continuously over successive outbreak years (odds ratio [OR] 0.98, 95% CI 0.97–0.98; p < 0.001) and was modestly lower in the COVID-19 era (≥2020) than in the pre-COVID-19 era (≤2019) (5.50% vs. 6.76%; p = 0.040). No difference was observed for norovirus crew attack risk between these eras (1.45% vs. 1.65%; p = 0.36). When stratified by whether laboratory confirmation methods were reported, norovirus passenger attack risk was 9.49% for outbreaks with reported methods and 6.16% for those without (p = 0.014), showing a modest association in multivariable models (OR 1.27, 95% CI 1.01–1.61; p = 0.045). Larger exposed populations (log-denominator) were independently associated with lower norovirus attack risks across overall (OR 0.69, 95% CI 0.61–0.79), passenger (OR 0.72, 95% CI 0.62–0.83), and crew subsets (OR 0.64, 95% CI 0.52–0.79; all p < 0.001). For other gastrointestinal infections (primarily ETEC, *Shigella* spp., *Salmonella* spp., and *Vibrio* spp.), passenger attack risk was 9.02% pre-COVID-19 and 4.60% in the COVID-19 era (p = 0.099). By region, the overall attack risk for norovirus was 10.77% in the Caribbean/Bahamas and 6.29% in other specified routes (p = 0.16). Additional exploratory subgroup analyses showed differences in pooled norovirus overall attack risk across cruise routes (Q = 10.74; p = 0.013) and between outbreaks before 2010 and those from 2010 onwards (10.27% vs. 4.10%; Q = 8.27; p = 0.0040). Separately, among 145 records reporting control measures, 86 (59.3%) reported isolation or quarantine and 67 (46.2%) reported environmental cleaning or disinfection; detailed results are provided in Supplementary Table S7. Between-study heterogeneity persisted within most subgroups ($I^2$ = 73.8%–99.5%). Corresponding forest plots for subgroup analyses are provided in Supplementary Figs. S39–S47.

Sensitivity analyses demonstrated overall stability of pooled attack risks across alternative $\tau^2$ estimators, exclusion of extreme denominators (<50 or >3000), exclusion of minimally described events (Supplementary Figs. S48–S51), event-level reconciliation of overlapping reports, and targeted series-level aggregation of potentially correlated estimates from continuing outbreaks across consecutive voyages (Supplementary Tables S7

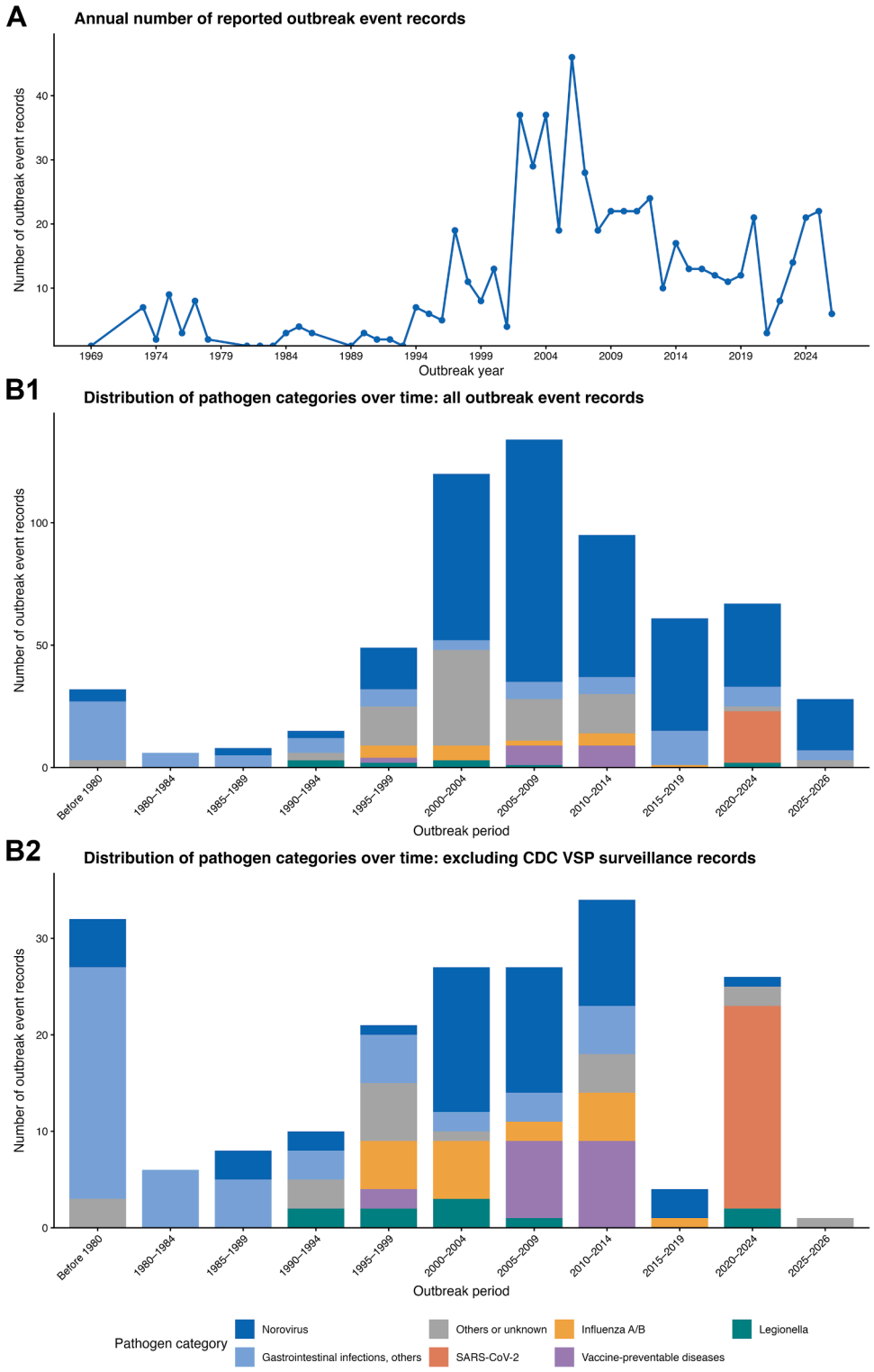


***Fig. 2:*** **Temporal distribution of cruise ship infectious disease outbreak event records.** Temporal distribution of the 619 outbreak event records identified between 1969 and 2026. (A) Annual number of reported outbreak event records. (B1) Distribution of pathogen categories across grouped time periods among all outbreak event records. (B2) Corresponding distribution after excluding surveillance records from the CDC VSP. Stacked bar segments represent the numbers of outbreak event records attributed to each pathogen category. **Abbreviations:** CDC, Centers for Disease Control and Prevention; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; VSP, Vessel Sanitation Program.

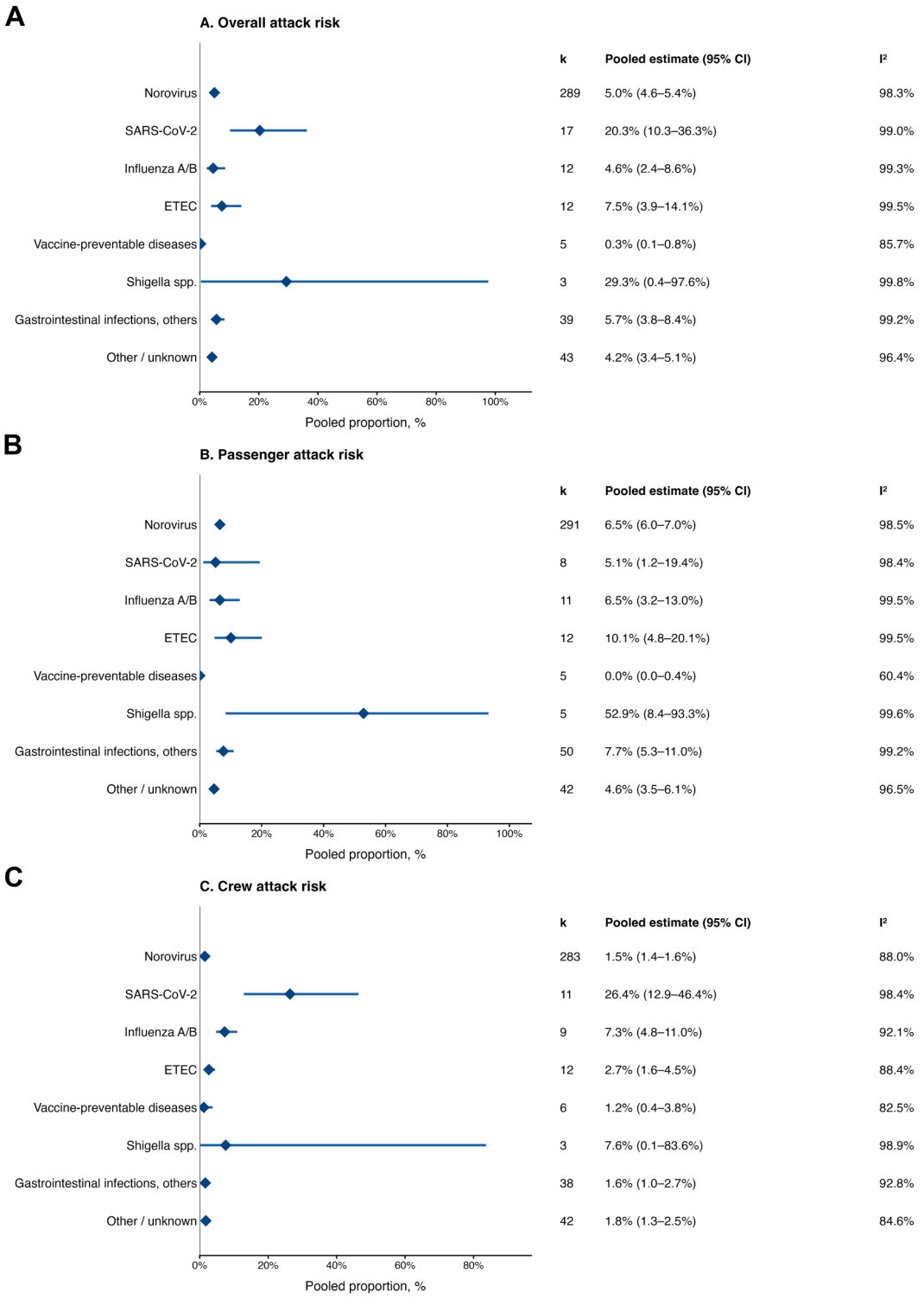


***Fig. 3:* Pathogen-specific pooled attack risks in cruise ship outbreaks.** Forest plots showing pathogen-specific pooled attack risks for (A) the overall onboard population, (B) passengers, and (C) crew members. Pooled proportions and 95% CIs were estimated using random-effects models with logit transformation, the inverse-variance method, restricted maximum-likelihood estimation of between-outbreak variance, and Hartung–Knapp adjustment with an ad hoc variance correction. Diamonds indicate pooled estimates, and horizontal lines indicate 95% CIs. The number of eligible outbreak event records contributing to each estimate is denoted by *k*, and between-outbreak heterogeneity is reported using $I^2$. **Abbreviations:** CI, confidence interval; ETEC, enterotoxigenic *Escherichia coli*; $I^2$, Higgins' I-squared statistic; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

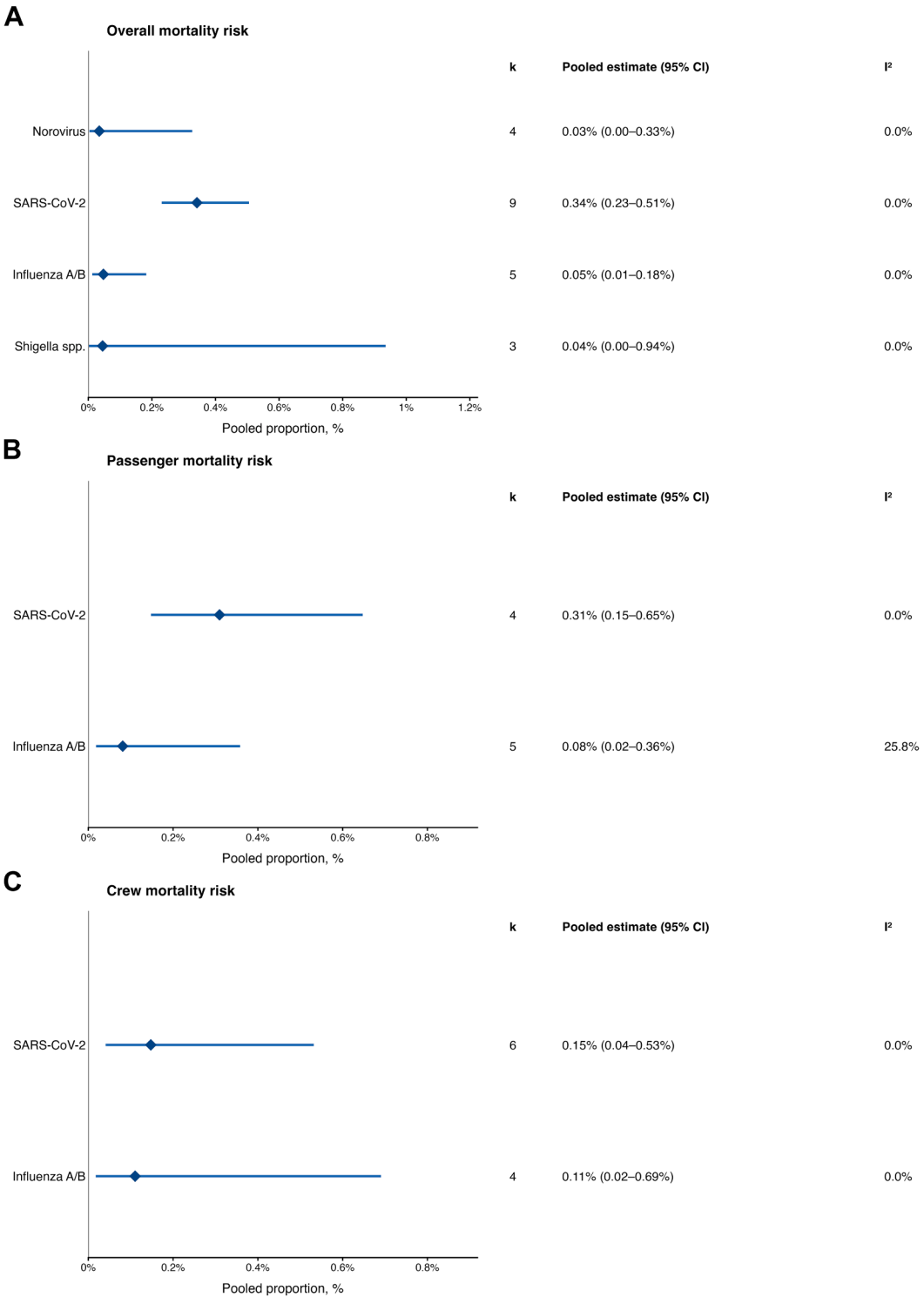


***Fig. 4:*** **Pathogen-specific pooled mortality risks in cruise ship outbreaks.** Forest plots showing pathogen-specific pooled mortality risks for (A) the overall onboard population, (B) passengers, and (C) crew members. Pooled proportions and 95% CIs were estimated using random-effects models with logit transformation, the inverse-variance method, restricted maximum-likelihood estimation of between-outbreak variance, and Hartung–Knapp adjustment with an ad hoc variance correction. Diamonds indicate pooled estimates, and horizontal lines indicate 95% CIs. The number of eligible outbreak event records contributing to each estimate is denoted by *k*, and between-outbreak heterogeneity is reported using $I^2$. **Abbreviations:** CI, confidence interval; $I^2$, Higgins' I-squared statistic; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

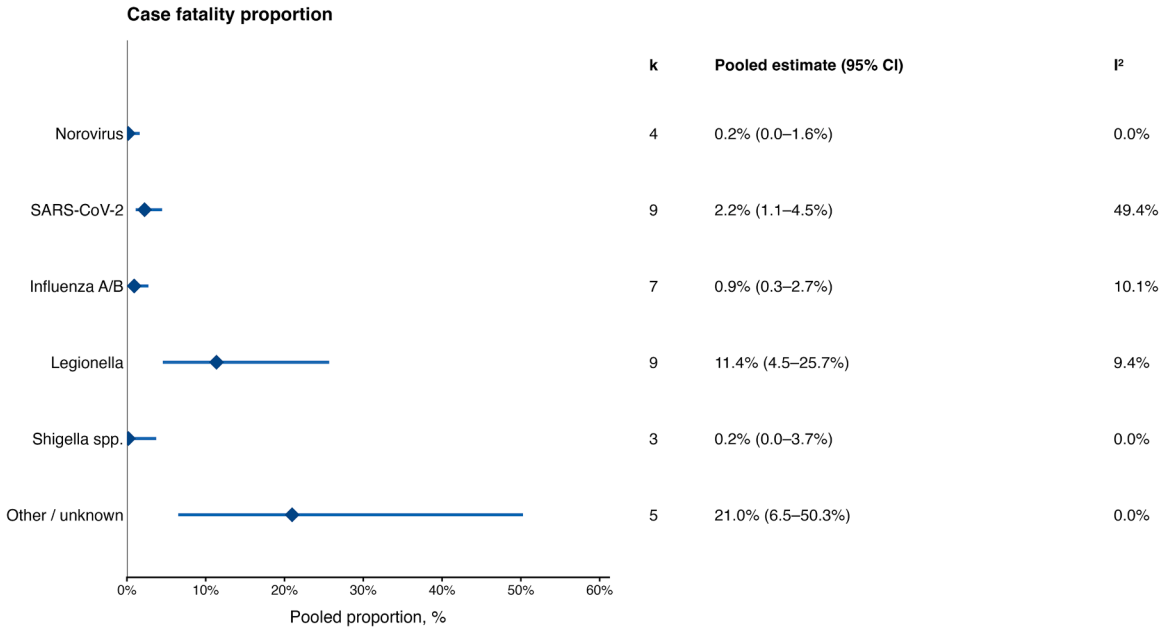


***Fig. 5:* Pathogen-specific pooled case fatality proportions in cruise ship outbreaks.** Forest plot showing pathogen-specific pooled case fatality proportions. Pooled proportions and 95% CIs were estimated using random-effects models with logit transformation, the inverse-variance method, restricted maximum-likelihood estimation of between-outbreak variance, and Hartung–Knapp adjustment with an ad hoc variance correction. Diamonds indicate pooled estimates, and horizontal lines indicate 95% CIs. The number of eligible outbreak event records contributing to each estimate is denoted by *k*, and between-outbreak heterogeneity is reported using $I^2$. **Abbreviations:** CI, confidence interval; $I^2$, Higgins' I-squared statistic; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

and S15). Leave-one-out analyses indicated robust pooled estimates, with variations restricted to pathogen categories characterised by small sample sizes (Supplementary Fig. S52). Restricting analyses to records reporting laboratory confirmation methods, excluding CDC VSP records, or excluding explicitly identified grey literature consistently yielded higher pooled attack risks for norovirus, ETEC, and other gastrointestinal infections (Supplementary Figs. S48–S51). Using the Freeman–Tukey double-arcsine transformation yielded comparable pooled attack-risk estimates (Supplementary Figs. S48–S51), and the principal era-based subgroup comparisons retained the same interpretation, although some exploratory comparisons varied (Supplementary Table S16). Funnel plots are shown in Supplementary Figs. S53–S70; Egger's tests were performed only when $k \geq 10$. Egger's tests indicated potential small-study effects in the norovirus and influenza attack-risk analyses, whereas no such evidence was detected in the evaluated SARS-CoV-2 analyses. Given the substantial heterogeneity, funnel-plot asymmetry was not considered definitive evidence of publication bias.

### Qualitative data synthesis of epidemiological and transmission parameters

Table 4 summarises the narrative synthesis of key epidemiological and transmission parameters. For SARS-CoV-2 outbreaks, transmission was documented within crowded, semi-closed environments with high population densities,[18,19] and a previous systematic review reported higher attack risks with increasing cabin occupancy.[20] $R_0$ spanned 2.00–5.55, with specific Diamond Princess estimates reaching 3.27 and 3.78 prior to quarantine implementation.[18,19,21,22] The incubation period was approximately 4–6 days (point estimates 5.0–5.2 days).[18,19,23–25] Serial intervals spanned 2.0–4.3 days, associated with multi-person cabins and shared quarters.[18,24] SAR reached 19.2%–28.4% across respiratory aerosol and droplet exposures in indoor settings.[18,26–28]

For norovirus, $R_0$ ranged from 1.50 to 15.60, reflecting differences in model structure, assumptions, and parameter derivation.[29–31] Generation times were 3.65–3.70 days, with a SAR of 18.2% and an incubation period of ~0.5–3 days (point estimates 1.15–1.46 days).[30,32] Other gastrointestinal infections exhibited widely variable incubation periods depending on the aetiologic agent and transmission vehicle, ranging from 1 to 8 h for foodborne bacterial toxins to 13 hours–3 days for waterborne or seafood-associated bacterial agents, and up to 25–40 days for predominantly asymptomatic hepatitis E outbreaks.[33–41]

Among influenza virus outbreaks, no cruise-ship-specific $R_0$ estimate was available in the extracted reports; however, one study reported an overall cabin-contact SAR of 55% (50 of 91), which was higher among children

| Pathogen category | Outcome | Subgroup variable | Subgroup category | Outbreak records, k | Pooled AR, % (95% CI) | $I^2$, % | p-between |
|---|---|---|---|---|---|---|---|
| Norovirus | Passenger attack risk | Era | Pre-COVID-19 (≤2019) | 233 | 6.76% (6.16%–7.41%) | 98.59 | 0.040 |
| | | | COVID-19 era (≥2020) | 54 | 5.50% (4.87%–6.22%) | 96.37 | |
| Norovirus | Crew attack risk | Era | Pre-COVID-19 (≤2019) | 225 | 1.45% (1.30%–1.62%) | 88.64 | 0.36 |
| | | | COVID-19 era (≥2020) | 54 | 1.65% (1.38%–1.98%) | 83.32 | |
| Norovirus | Overall attack risk | Route/region | Caribbean/Bahamas | 10 | 10.77% (6.54%–17.21%) | 99.17 | 0.16 |
| | | | Other specified routes | 25 | 6.29% (3.94%–9.91%) | 98.90 | |
| Norovirus | Passenger attack risk | Laboratory confirmation method reported | Laboratory confirmation method reported | 37 | 9.49% (6.59%–13.49%) | 99.23 | 0.014 |
| | | | No laboratory confirmation method reported/unclear | 254 | 6.16% (5.74%–6.61%) | 97.75 | |
| Norovirus | Crew attack risk | Laboratory confirmation method reported | Laboratory confirmation method reported | 32 | 1.66% (1.16%–2.37%) | 88.29 | 0.29 |
| | | | No laboratory confirmation method reported/unclear | 251 | 1.47% (1.34%–1.62%) | 87.82 | |
| Other gastrointestinal infections | Passenger attack risk | Era | Pre-COVID-19 (≤2019) | 38 | 9.02% (5.76%–13.86%) | 99.29 | 0.099 |
| | | | COVID-19 era (≥2020) | 12 | 4.60% (2.80%–7.46%) | 97.24 | |
| Other gastrointestinal infections | Crew attack risk | Era | Pre-COVID-19 (≤2019) | 26 | 1.89% (1.01%–3.50%) | 91.16 | 0.42 |
| | | | COVID-19 era (≥2020) | 12 | 1.21% (0.46%–3.18%) | 95.24 | |

**Note:** Pooled proportions and 95% CIs were estimated using random-effects models with logit transformation, the inverse-variance method, the restricted maximum-likelihood estimator for between-outbreak variance, and Hartung–Knapp adjustment with an ad hoc variance correction. Subgroup differences were assessed using the Q-between test under a mixed-effects framework with a common between-outbreak variance across subgroup categories. Only subgroup categories with at least three eligible outbreak event records were pooled. Route- or region-based results should be interpreted as exploratory because route information was missing for a substantial proportion of records. "Laboratory confirmation method reported" indicates whether a laboratory confirmation method was documented, rather than definitive confirmation of every reported case. p-between denotes the p value from the Q-between test for subgroup differences. **Abbreviations:** AR, attack risk; CI, confidence interval; COVID-19, coronavirus disease 2019; $I^2$, proportion of total variability attributable to between-outbreak heterogeneity.

***Table 2:*** **Subgroup analyses of pooled attack risks for selected pathogen categories.**

aged < 12 years (76%; 16 of 21) than among those aged >12 years (49%; 34 of 70).[42] *Legionella* spp. infections emerged following a 2–10 day incubation period, linked to aerosolised contaminated water from whirlpool spas.[43,44] Varicella SAR reached 90% among susceptible adult crew members recruited from regions lacking universal childhood vaccination programmes, with incubation periods spanning 10–21 days (commonly 14–16 days).[45–48] Andes hantavirus exhibited an $R_0$ of 0.7 and an extended incubation period of 22 days.[49]

| Outcome | Moderator | Outbreak records, k | OR (95% CI) | p value | Residual $\tau^2$ | Pseudo-$R^2$, % | P for moderators |
|---|---|---|---|---|---|---|---|
| Overall attack risk | Outbreak year, per 1-year increase | 285 | 0.98 (0.98–0.99) | <0.001 | 0.332 | 21.2 | <0.001 |
| | Log denominator, per 1-unit increase | | 0.69 (0.61–0.79) | <0.001 | | | |
| | Laboratory confirmation method reported | | 1.32 (1.06–1.64) | 0.014 | | | |
| Passenger attack risk | Outbreak year, per 1-year increase | 287 | 0.98 (0.97–0.98) | <0.001 | 0.388 | 22.9 | <0.001 |
| | Log denominator, per 1-unit increase | | 0.72 (0.62–0.83) | <0.001 | | | |
| | Laboratory confirmation method reported | | 1.27 (1.01–1.61) | 0.045 | | | |
| Crew attack risk | Outbreak year, per 1-year increase | 279 | 0.99 (0.98–1.00) | 0.080 | 0.409 | 10.9 | <0.001 |
| | Log denominator, per 1-unit increase | | 0.64 (0.52–0.79) | <0.001 | | | |
| | Laboratory confirmation method reported | | 1.03 (0.75–1.41) | 0.86 | | | |

**Note:** Random-effects meta-regression models were fitted using logit-transformed proportions, restricted maximum-likelihood estimation, and Hartung–Knapp adjustment. The multivariable models included outbreak year, log-transformed denominator, and whether a laboratory confirmation method was reported. ORs for outbreak year represent the change per 1-year increase. ORs for the log denominator represent the change per 1-unit increase in the natural logarithm of the denominator. "Laboratory confirmation method reported" indicates whether a laboratory confirmation method was documented in the source record. Pseudo-$R^2$ represents the proportion of between-outbreak heterogeneity explained by the included moderators. The p values correspond to tests of individual moderator coefficients, and P for moderators denotes the omnibus test of all moderators in the model. **Abbreviations:** AR, attack risk; CI, confidence interval; OR, odds ratio; $\tau^2$, residual between-outbreak variance.

***Table 3:*** **Multivariable meta-regression analyses of attack risks in norovirus outbreaks on cruise ships.**

| Pathogen/ disease | Transmission parameter | No. of studies, N | Reported values: median/mean (range/95% CI) | Key contextual findings and sources of heterogeneity | Representative studies |
|---|---|---|---|---|---|
| SARS-CoV-2 (COVID-19) | Basic/effective reproduction number ($R_0$/ $R_t$) | 3 | $R_0$: 2.00–5.55; Diamond Princess estimates: 3.27 (95% CI, 3.02–3.54) and 3.78 (95% CI, 3.49–4.09) | Estimates varied across ship types, outbreak stages, and intervention strategies. Dense indoor spaces, shared cabins, and delayed isolation may have facilitated transmission. | Diamond Princess; Grand Princess; Greg Mortimer |
| | Incubation period | 4 | Approximately 4–6 days; point estimates: 5.0–5.2 days | Estimates were broadly consistent across cruise-ship outbreaks, although prolonged exposure periods made precise attribution of infection dates difficult. | Diamond Princess; MS Roald Amundsen; Greg Mortimer; Netherlands river cruise |
| | Serial interval | 2 | 2.0–4.3 days | Estimates may differ because of outbreak stage, contact patterns, and the timing of isolation or quarantine. | Diamond Princess; MS Roald Amundsen |
| | Secondary attack rate (SAR) | 3 | Overall SAR: 19.2%–28.4% | SAR varied according to testing strategies, case definitions, and contact intensity. Cabin isolation, quarantine, and universal testing influenced observed transmission. | Diamond Princess; Costa Atlantica; MS A'SARA |
| Norovirus/acute gastroenteritis | Basic reproduction number ($R_0$) | 3 | 1.50–15.60 | Wide variation reflected differences in modelling assumptions and the relative contribution of direct, foodborne, and environmentally mediated transmission. | Mitigating norovirus spread on cruise ships; Real-time spatiotemporal tracking; Quantifying the relative effects of environmental and direct transmission |
| | Incubation period | 5 | Approximately 0.5–3 days; model-based point estimates: 1.15–1.46 days | Incubation periods were generally short. Several repeated records were derived from shared model inputs rather than event-specific estimates. | Cruise A–G model study; Ship X; Cruise Ship-2002; European river cruise |
| | Generation time | 2 | 3.65–3.70 days | Values were derived primarily from modelling studies and should not be interpreted as multiple independent outbreak-specific estimates. | Forecasting norovirus cases on cruise ships; Mitigating norovirus spread on cruise ships |
| | Secondary attack rate (SAR) | 1 | 18.2% | Evidence was limited in the currently extracted dataset. Transmission was associated with contaminated cold dishes and asymptomatic food handlers. | Chongqing cruise ship outbreak, 2017 |
| Influenza A/B | Secondary attack rate (SAR) | 1 | 55% (50/91) among cabin contacts; <12 years: 76% (16/21); >12 years: 49% (34/70) | Estimated among passengers sharing a cabin with an infectious primary case-patient, after excluding co-primary cases. The estimate was symptom-defined and may overestimate virologically confirmed secondary transmission because not all symptomatic contacts were tested and other respiratory infections were not excluded. | Outbreaks of Pandemic (H1N1) 2009 and Seasonal Influenza A (H3N2) on Cruise Ship |
| *Legionella* spp. | Incubation period | 2 | 2–10 days | Outbreaks were linked to environmental exposures, particularly contaminated whirlpool spas or water systems. Values may represent established clinical ranges rather than ship-specific estimates. | Cruise-ship-associated Legionnaires disease; Outbreak among passengers exposed to a contaminated whirlpool spa |
| Varicella | Incubation period | 3 | 10–21 days; commonly reported values: 14–16 days | Long incubation periods complicated outbreak control because crew transfers and exposure across voyages could contribute to continued transmission. Several values reflected standard clinical ranges. | Management and control of varicella on cruise ships; Varicella at sea; Public health significance of chickenpox on ships |
| | Secondary attack rate (SAR) | 1 | 90% | High reported transmission risk highlights the importance of cabin isolation, post-exposure vaccination, and active symptom monitoring. | Maritime varicella illness and death reporting, U.S., 2010–2015 |
| Other gastrointestinal infections | Incubation period | 9 | Pathogen-specific ranges: 1–8 h for staphylococcal food poisoning; 13 h–3 days for bacterial gastroenteritis and waterborne outbreaks; 3–28 days for trichinosis; and 25–40 days for hepatitis E. | Incubation periods varied substantially according to pathogen type and transmission mechanism. Values were summarised narratively rather than pooled. | Hepatitis E; staphylococcal food poisoning; Brainerd diarrhoea; ETEC; *Vibrio parahaemolyticus*; trichinosis; multi-pathogen waterborne outbreak |
| Cyclospora infection | Incubation period | 2 | 7 days | Both studies reported a relatively consistent incubation period. Foodborne exposures were important considerations. | An outbreak of Cyclospora infection on a cruise ship; Update: outbreaks of cyclosporiasis—United States and Canada, 1997 |

(Table 4 continues on next page)

| Pathogen/ disease | Transmission parameter | No. of studies, N | Reported values: median/mean (range/95% CI) | Key contextual findings and sources of heterogeneity | Representative studies |
|---|---|---|---|---|---|
| (Continued from previous page) | | | | | |
| Andes hantavirus | Basic reproduction number ($R_0$) | 1 | 0.7 | Evidence was limited to a single outbreak. The result should be presented separately and not combined with respiratory-virus estimates. | MV Hondius outbreak, 2026 |
| | Incubation period | 1 | 22 days | The long incubation period has implications for contact tracing and post-disembarkation monitoring. | MV Hondius outbreak, 2026 |

**Note:** Values are reported as extracted from the included studies. **Abbreviations:** CI, confidence interval; COVID-19, coronavirus disease 2019; ETEC, enterotoxigenic *Escherichia coli*; R0, basic reproduction number; Rt, effective reproduction number; SAR, secondary attack rate; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

***Table 4:*** **Narrative synthesis of key epidemiological and transmission parameters for infectious disease outbreaks on cruise ships.**

## Discussion

Our results show that infectious disease outbreaks on cruise ships involve multiple pathogens and demonstrate distinct infection patterns between passenger and crew populations. Among 619 extracted outbreak records, acute gastroenteritis was the most common clinical syndrome, with norovirus representing the most frequently identified pathogen. The pooled attack risk for norovirus was 6.5% among passengers and 1.5% among crew members. Respiratory viruses exhibited different transmission profiles: SARS-CoV-2 attack risk was higher among crew members than passengers (26.4% vs. 5.1%), whereas influenza attack risks were comparable between the two groups (7.3% vs. 6.5%). Case fatality proportions were low across most evaluated pathogens, except for *Legionella* (11.4%). Temporal analyses indicated a shift from predominantly norovirus outbreaks to the concurrent emergence of respiratory and other pathogens, and subgroup analyses showed a reduction in norovirus passenger attack risk during the post-2020 period. These findings suggest that the epidemiological profile of maritime outbreaks is expanding beyond routine gastrointestinal infections. Public health interventions and onboard infection control protocols should account for these evolving pathogen dynamics and the differential vulnerabilities of passengers and crew members.

The shift from norovirus-dominated outbreaks to outbreaks involving multiple pathogens may be related to several interrelated factors. Dense, semi-closed shipboard environments facilitate the rapid transmission of respiratory viruses, such as SARS-CoV-2 and influenza, following index case introduction.[24,50] Furthermore, expanding expedition itineraries may create zoonotic exposure pathways through onshore ecological excursions,[51] as demonstrated by the Andes hantavirus outbreak linked to the MV Hondius.[8,9] The expansion of the commercial cruise industry and the routine detection of multiple pathogens in ship wastewater and clinical data may also have contributed to their greater representation in the published literature.[1,52,53] These factors indicate that maritime public health strategies require expansion beyond routine gastrointestinal monitoring to encompass comprehensive respiratory and emerging zoonotic pathogen surveillance. In addition, temporal patterns in reported outbreak records may also reflect changes in diagnostic capacity and molecular testing, reporting requirements and practices, COVID-19 surveillance intensity, and publication activity, as COVID-19-related publications increased markedly and may have made SARS-CoV-2 outbreaks more likely to be identified in a literature-based review than influenza or norovirus outbreaks.[54–57] These findings should therefore be interpreted cautiously and should not be taken as direct evidence of changes in underlying maritime disease epidemiology.

Attack risks differed between passengers and crew members, which may reflect variations in exposure and containment measures. For norovirus, passengers exhibited a higher attack risk, consistent with previous findings.[58] As norovirus is primarily driven by contact and faecal-oral transmission, separate sleeping and dining areas and distinct boarding and exiting zones represent possible explanations for the lower attack risk among crew members, but these characteristics were not consistently measured across the included records.[56,58] However, while such spatial segregation might reduce opportunities for contact relevant to enteric pathogens, it may provide limited protection against droplet and short-range aerosol transmission. For SARS-CoV-2, the higher crew attack risk may reflect quarantine conditions in which passengers were isolated in private cabins, whereas crew members maintained essential operations in shared quarters and remained exposed to undetected asymptomatic or pre-symptomatic infections.[21,59,60] However, the wide confidence intervals suggest that these estimates may be influenced by individual outbreaks and should be interpreted cautiously. Influenza demonstrated comparable attack risks between groups, and the factors underlying this pattern warrant further investigation. Collectively, these findings suggest that infection prevention strategies on cruise ships should be tailored to pathogen-specific transmission dynamics, combining

environmental and hygiene interventions for enteric pathogens with pre-boarding vaccination and operational measures that reduce exposure among essential personnel during respiratory virus outbreaks.

Mortality risks and case fatality proportions were low across most pathogens. *Legionella* was an exception, with a pooled case fatality proportion of 11.4%, consistent with the established severity of Legionnaires' disease.[61–64] Older passengers and individuals with underlying health conditions are particularly susceptible to severe pneumonia following exposure to aerosolised bacteria from contaminated water systems, most commonly hot tubs affected by inadequate disinfection, water stagnation, or poor temperature control.[61,65,66] Given the potential for severe outcomes, comprehensive water system maintenance and routine *Legionella* surveillance should be prioritised as part of shipboard infection prevention programmes.

Subgroup analyses and meta-regression identified temporal and methodological factors associated with variation in outbreak-level attack risks. For norovirus, passenger attack risk was modestly lower in the post-2020 period than in the pre-COVID-19 era, whereas crew attack risk remained stable. The reduction in passenger attack risk may be associated with the implementation of enhanced public health measures targeting passenger environments, including isolation of symptomatic individuals and strengthened sanitation protocols.[67] However, direct evidence evaluating these specific post-2020 interventions remains limited, and the reasons why crew attack risk did not exhibit a corresponding decline require further investigation. Although a similar post-2020 reduction was observed for other gastrointestinal infections, the difference was not statistically significant. Additionally, norovirus passenger attack risk was higher in outbreaks with reported laboratory confirmation methods, possibly reflecting ascertainment bias because intensively investigated outbreaks are more likely to undergo testing and be documented.[3,12,68] Larger exposed populations were associated with lower attack risks, although previous surveillance studies have reported different relationships between ship size and gastrointestinal illness incidence,[56] suggesting substantial heterogeneity in transmission dynamics across cruise ship outbreaks. In separate exploratory subgroup analyses of norovirus overall attack risk, pooled estimates differed across cruise routes and between events before 2010 and those from 2010 onwards (Supplementary Table S7; Supplementary Figs. S46 and S47). The lower estimate from 2010 onwards coincided with the progressive standardisation of shipboard sanitation and outbreak-response practices under the CDC VSP.[69] High between-study heterogeneity across primary and subgroup meta-analyses resulted in very low certainty and warrants cautious interpretation of the pooled proportions, given the diverse spatiotemporal contexts and outbreak settings.[12]

To our knowledge, this is the first comprehensive systematic review and meta-analysis evaluating epidemiological parameters across infectious disease outbreaks on commercial cruise ships. However, our findings must be interpreted in light of several limitations regarding the available evidence. First, because each title and abstract record was assessed by one trained screener, some potentially eligible records may have been inadvertently excluded at this stage. Second, variations in methodological reporting and outbreak contexts contributed to substantial between-study heterogeneity, driven particularly by differences in surveillance data sources. Third, when overall onboard populations were not explicitly reported, overall attack risks were derived by summing passenger- and crew-specific values where all required components were available, potentially affecting estimate precision and comparability. Fourth, voyage duration and several ship and operational characteristics were incompletely and inconsistently reported, precluding quantitative assessment of their contribution to heterogeneity in outbreak-level attack risks. Fifth, pooled estimates for sparse outcomes, such as mortality risks and case fatality proportions, were based on a limited number of eligible outbreak records and therefore had limited precision. Sixth, inconsistent terminology across settings, publication bias, and investigation thresholds may contribute to heterogeneity in data availability, since studies with larger or more extensive outbreaks are more often described in the literature.[3,12,68] Seventh, individual participant data were unavailable, precluding uniform adjustment for individual-level confounders and the exploration of specific effect modifiers. Finally, most eligible reports, particularly outbreak reports, did not provide age- or sex-specific case counts or denominators, preventing assessment of age- and sex-related differences in study outcomes.

Future investigations should adopt standardised reporting frameworks with consistent case definitions and precise population denominators, alongside individual-level data to adjust for confounding and examine micro-environmental factors such as cabin characteristics and occupational networks. Furthermore, reported response measures were summarised descriptively because comparable effectiveness estimates were unavailable and measures were often implemented in combination, precluding assessment of their individual effects. Accordingly, rigorous evaluation of specific shipboard interventions, including respiratory virus transmission-mitigation measures and targeted occupational health protocols for crew members, is warranted. Given the increasing diversity of maritime pathogens, integrating genomic epidemiology and routine environmental surveillance, such as wastewater monitoring, could enhance early detection capabilities and clarify cross-voyage transmission dynamics.

In conclusion, this systematic review and meta-analysis shows that although norovirus remains the leading cause of cruise ship outbreaks, a wider range of pathogens, including respiratory viruses and *Legionella*, has been increasingly reported in recent years. Attack risks differed between passengers and crew members, with higher norovirus attack risks among passengers and higher SARS-CoV-2 attack risks among crew members. Case fatality proportions were generally low, except for *Legionella* infections. These findings support a broader approach to maritime infectious disease prevention that extends beyond routine gastrointestinal surveillance. Future outbreak preparedness should incorporate pathogen-specific control measures, including occupational protections for essential personnel, effective water system management, and containment strategies tailored to different transmission routes.

#### Contributors

JL conceptualised and supervised the study. SW and LZ performed literature screening and data extraction. LZ performed formal data analysis. SW drafted the original manuscript. SW, LZ, and JL accessed and verified the underlying data. YY, ML, DRL, and JL provided critical feedback, interpreted the findings, and revised the manuscript for important intellectual content. All authors read and approved the final version of the manuscript. All authors had full access to all the data in the study, and the corresponding author had final responsibility for the decision to submit for publication.

#### Data sharing statement

The comprehensive dataset extracted from the included studies and used for all quantitative and qualitative analyses is publicly available in supplementary materials (Supplementary Table S4). The template data collection forms, analytic code, and any other relevant materials used in this systematic review are available upon reasonable request from the corresponding author.

#### Declaration of interests

We declare no competing interests.

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#### Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.eclinm.2026.104325.

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