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Genomic Surveillance and Epidemiological Modeling of Antimicrobial Resistance (AMR) in Urban Hospital Effluents A Methodological Framework Integrating Metagenomic Surveillance with Dynamic Transmission Modeling of the Hospital–Effluent–Community Continuum

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Abstract

Antimicrobial resistance (AMR) is among the most pressing threats to global public health, and urban hospitals are increasingly recognized as concentrated point sources of resistant organisms and resistance genes discharged into municipal sewer systems. This paper presents an integrated framework that combines genomic (metagenomic) surveillance of hospital effluent with dynamic epidemiological modeling to characterize the emergence, persistence, and downstream dissemination of AMR determinants from urban healthcare facilities. We synthesize current evidence on the resistome of hospital wastewater, describe a reproducible sampling and shotgun-metagenomic sequencing workflow spanning ward-level drains, wastewater treatment plant (WWTP) influent and effluent, and downstream receiving water, and couple this with a compartmental transmission model linking in-hospital colonization dynamics to an environmental reservoir compartment representing the effluent system. Using illustrative simulation informed by patterns reported in the literature, we show that the resistant-organism load discharged into effluent responds non-linearly to both clinical antimicrobial stewardship and effluent treatment intensification, and that combined interventions produce substantially larger reductions in downstream environmental resistance load than either intervention alone. We further present a simulated but literature-consistent resistome profile showing that beta-lactamase and aminoglycoside resistance genes

dominate hospital-associated effluent and are only partially attenuated by conventional wastewater treatment. We discuss the implications of these findings for One Health AMR surveillance architecture, the technical and governance barriers to operationalizing genomic wastewater-based epidemiology (WBE) at the hospital scale, and priority directions for coupling sequencing data streams with mechanistic and statistical models to generate actionable, near-real-time risk indicators. The framework is intended as a template that can be parameterized with site-specific sequencing and clinical data to support hospital infection control, environmental regulation, and regional AMR early-warning systems.

Introduction

Antimicrobial resistance (AMR) has moved from a projected future threat to a documented cause of substantial present-day mortality and morbidity, with resistant bacterial infections estimated to be directly associated with over a million deaths annually and a contributing factor in several million more. While clinical surveillance systems capture resistance among bacteria isolated from symptomatic, treatment-seeking patients, this represents only a fraction of the total burden of resistant organisms circulating within a population. Colonization with resistant organisms is frequently asymptomatic, and clinical microbiology laboratories in many settings, particularly in low- and middle-income countries, lack the infrastructure for systematic, population-representative resistance surveillance.

Hospitals occupy a distinctive position in the AMR landscape. They are simultaneously sites of the highest per-capita antimicrobial consumption, environments where the most resistant and clinically significant organisms (including carbapenem-resistant Enterobacterales, vancomycin-resistant enterococci, and multidrug-resistant Pseudomonas aeruginosa and Acinetobacter baumannii) are concentrated among colonized and infected patients, and sources of wastewater that is discharged, often with only partial in-house treatment, into municipal sewer networks. Hospital effluent therefore functions as a natural, continuously updated composite sample of the resistance burden carried by an inpatient population, analogous to the role wastewater played in community-level tracking of SARS-CoV-2 during the COVID-19 pandemic.

Two methodological developments have converged to make systematic genomic surveillance of hospital effluent newly feasible. First, shotgun metagenomic and metatranscriptomic sequencing now allow culture-independent, genome-resolved characterization of the resistome (the collective set of antimicrobial resistance genes, ARGs) and its bacterial hosts directly from wastewater, without the biases introduced by selective culturing. Second, advances in compartmental and network epidemiological modeling have begun to formally incorporate environmental reservoirs, including wastewater systems, as dynamic compartments that interact with human colonization states rather than as passive, terminal sinks.

Despite this convergence, genomic surveillance data and epidemiological transmission models have largely developed in parallel literatures: metagenomic studies characterize what resistance genes and organisms are present in hospital wastewater at a point in time, while transmission models simulate how resistance spreads through populations, often treating the environment as a simplified or absent compartment. This paper addresses that gap by proposing and demonstrating an integrated framework in which genomic surveillance outputs directly parameterize and validate a dynamic transmission model of the hospital–effluent–community continuum. The specific objectives of this paper are threefold: (i) to synthesize current evidence on the composition and dynamics of the hospital wastewater resistome; (ii) to describe a reproducible genomic surveillance workflow spanning intra-hospital sampling points through to downstream receiving water; and (iii) to formulate and demonstrate a compartmental epidemiological model that links in-hospital

colonization dynamics to environmental resistance load, illustrating how such a model can be used to evaluate the comparative impact of clinical stewardship and effluent treatment interventions.

Literature Review

The hospital wastewater resistome

Multiple metagenomic studies of hospital wastewater converge on a consistent qualitative picture: genes conferring resistance to beta-lactams, aminoglycosides, and macrolide–lincosamide–streptogramin (MLSB) antibiotics tend to dominate the hospital resistome, with OXA-type beta-lactamases frequently reported as the most abundant single gene family in national-scale surveys.

Genome-resolved metagenomic approaches, which reconstruct metagenome-assembled genomes (MAGs) rather than relying on read-level gene annotation alone, have enabled direct linkage of ARGs to their bacterial hosts and have shown that host associations shift measurably between wastewater treatment plant influent and effluent, indicating that treatment does not uniformly affect all ARG-carrying taxa.

Complementary metatranscriptomic work has demonstrated that a subset of ARGs detected at the DNA level are also actively transcribed, and that plasmid-borne resistance determinants often show disproportionately high mRNA-to-DNA ratios relative to chromosomal genes, suggesting that mobile genetic elements are a particularly active reservoir for functional resistance expression in wastewater bacterial communities.

Comparative studies contrasting hospital and community or municipal wastewater consistently report that hospital-derived wastewater carries a significantly higher abundance and greater heterogeneity of ARGs than community wastewater, and that the composition of the hospital resistome varies meaningfully by ward specialty and correlates, for some organism–drug combinations, with clinical antimicrobial usage and isolate-level resistance prevalence.

Persistence through wastewater treatment

A recurring and policy-relevant finding across the literature is that conventional wastewater treatment substantially reduces, but does not eliminate, resistant organisms and ARGs. Cross-sectional shotgun metagenomic sampling before and after hospital wastewater treatment has shown that while the relative abundance of some organisms, including Escherichia coli, declines through treatment, other clinically important species such as Staphylococcus aureus, Acinetobacter baumannii, and Pseudomonas aeruginosa can increase in relative abundance, and Klebsiella pneumoniae frequently persists in both raw and treated wastewater. This selective attenuation pattern implies that treated effluent discharged into receiving waters is not resistance-free, and that treatment processes may inadvertently favor the survival of a subset of resistant, environmentally hardy taxa.

Wastewater-based epidemiology and its integration with modeling

Wastewater-based epidemiology (WBE) has been proposed as a complementary, population-level surveillance tool for AMR, extending the paradigm validated for SARS-CoV-2 monitoring during the COVID-19 pandemic. Reviews of the WBE literature highlight its capacity for unbiased, non-invasive, catchment-level assessment of circulating resistance and its promise for early-warning detection of emerging multidrug-resistant pathogens, while also identifying persistent challenges: technical variability across sequencing and bioinformatic pipelines, ambiguity in attributing detected signal to human clinical, community, industrial, or infrastructural sources, and limited detection sensitivity for rare or emerging ARGs in low-biomass or highly diluted samples.

On the modeling side, a smaller body of work has begun to formally incorporate environmental compartments into transmission dynamic models of AMR. Compartmental models linking human, animal, and environmental resistance reservoirs have shown that explicitly modeling the environment changes both the predicted equilibrium prevalence of resistance and the effectiveness of interventions: environmental transmission can either amplify or dampen the impact of reducing antimicrobial consumption, depending on the relative magnitude of environment-to-host and host-to-host transmission parameters, and eradicating resistance within a hospital is markedly more difficult to achieve in models where the environment is treated as an active reservoir rather than ignored. These findings motivate the explicit environmental compartment adopted in the model developed in Section 3.3 of this paper.

Gap addressed by this paper

Taken together, the reviewed literature establishes that (i) hospital wastewater carries a distinctive, clinically relevant resistome; (ii) this resistome is only partially attenuated by conventional treatment; (iii) WBE is methodologically mature enough to generate quantitative, catchment-resolved surveillance signal; and (iv) environmentally explicit transmission models predict materially different intervention outcomes than models that omit the environment. What remains comparatively underdeveloped is a worked framework that treats genomic surveillance data as direct inputs to, and validation targets for, an environmentally explicit transmission model applied specifically at the scale of an individual urban hospital and its effluent catchment. Sections 3–5 develop such a framework.

Materials and Methods

Study design and sampling framework

The proposed surveillance architecture uses a nested, longitudinal sampling design spanning five point types along the hospital-to-environment continuum: (1) ward-level sewer drains stratified by clinical risk (e.g., intensive care unit, oncology/hematology, general medical/surgical wards); (2) the composite hospital effluent stream at the point it enters municipal sewer infrastructure or an on-site pretreatment unit; (3) WWTP influent, capturing the mixture of hospital and community wastewater; (4) WWTP effluent, after the full treatment train (typically primary settling, secondary biological treatment, and disinfection); and (5) the receiving water body downstream of the discharge point. Twenty-four-hour composite sampling, rather than grab sampling, is recommended at each point to average over diurnal variation in flow and clinical activity, consistent with approaches used in prior hospital wastewater metagenomic studies. A minimum longitudinal design of weekly composite sampling over at least twelve months is recommended to capture seasonal variation in antimicrobial prescribing and treatment plant performance.

Genomic surveillance workflow

Each composite sample undergoes a standardized processing pipeline: (i) concentration of bacterial biomass by centrifugation or membrane filtration; (ii) total nucleic acid extraction with parallel DNA and RNA workflows to support paired metagenomic and metatranscriptomic sequencing; (iii) shotgun sequencing on a high-throughput short-read platform, targeting a minimum depth sufficient for genome-resolved assembly (informed by prior studies, on the order of tens of gigabases per sample for deep hospital effluent characterization); and (iv) bioinformatic processing comprising quality filtering and host-read removal, de novo metagenomic assembly, binning into metagenome-assembled genomes (MAGs), taxonomic classification, and ARG annotation against curated resistance gene databases. Where feasible, selective culture enrichment (for example, on carbapenem- or meropenem-supplemented media)

is recommended as a complementary, lower-depth approach to increase sensitivity for rare, clinically critical ARGs that may be underrepresented in bulk metagenomic sequencing, consistent with findings that culture enrichment meaningfully improves detection of low-abundance resistance determinants.

To support source attribution — distinguishing human gut-associated resistance signal from sewer-infrastructure-associated or environmental background signal — MAGs and reads are additionally classified against reference human gut microbiome databases, allowing gut-derived and non-gut-derived components of the wastewater resistome to be analyzed separately. This distinction is important for interpreting whether a given surveillance signal more plausibly reflects colonization prevalence among patients versus persistence or amplification of resistant organisms within hospital plumbing and sewer biofilms.

Epidemiological modeling framework

To translate genomic surveillance signal into a dynamic, intervention-relevant picture of AMR transmission, we formulate a deterministic compartmental model coupling an in-hospital patient population to an environmental reservoir representing the effluent system. The patient population is divided into three states: susceptible/uncolonized (Su), colonized with antibiotic-susceptible organisms (Cs), and colonized with antibiotic-resistant organisms (Cr). A fourth, environmental compartment (E) represents the normalized resistant-organism load in the hospital effluent stream, which is replenished by shedding from resistant-colonized patients and is depleted by natural die-off and by wastewater treatment. The system is governed by the following coupled ordinary differential equations:

$$dSu/dt = -(\beta r \cdot Cr/N + k \cdot E) \cdot Su - \beta s \cdot (Cs/N) \cdot Su + \gamma s \cdot Cs + \gamma r \cdot Cr - \mu \cdot Su + \mu \cdot N$$

$$dCs/dt = \beta s \cdot (Cs/N) \cdot Su - \gamma s \cdot Cs - \alpha \cdot Cs - \mu \cdot Cs$$

$$dCr/dt = (\beta r \cdot Cr/N + k \cdot E) \cdot Su - \gamma r \cdot Cr + \alpha \cdot Cs - \mu \cdot Cr$$

$$dE/dt = s \cdot Cr - d \cdot E - \tau \cdot E$$

where βr and βs are patient-to-patient transmission rates for resistant and susceptible colonization respectively; k is the environment-to-patient transmission coefficient linking effluent resistant load to new resistant colonization (capturing, for example, contamination of shared clinical equipment, plumbing-associated exposure, or environmental biofilm contact); γs and γr are clearance (decolonization) rates; α is the within-host acquisition rate of resistance among patients already colonized with susceptible organisms, representing selection under antibiotic exposure; μ is the combined admission/discharge turnover rate; s is the shedding rate of resistant organisms into effluent per colonized patient; d is the natural decay rate of resistant organisms in the effluent system; and τ is the additional removal rate attributable to wastewater treatment intensity. This structure follows, and extends to the hospital effluent context, environmentally explicit AMR transmission models previously developed for human–animal–environment systems, in which the environment is treated as an active, dynamically coupled reservoir rather than a passive sink.

Genomic surveillance data parameterize this model in three ways: (i) longitudinal ARG abundance and MAG-based taxonomic data from ward-level and effluent sampling provide time-resolved estimates of Cr and E that can be used for parameter fitting; (ii) metatranscriptomic mRNA/DNA ratios inform which ARG-carrying taxa are functionally active, refining estimates of the effective shedding rate s; and (iii) comparison of resistome composition between WWTP influent and effluent samples provides an empirical estimate of the treatment removal parameter τ under current site-specific treatment conditions, which can then be varied in simulation to evaluate the effect of treatment intensification (for example, addition of ultraviolet disinfection or ozonation).

Illustrative simulation

To demonstrate the framework's behavior, we numerically integrated the model over a 180-day horizon under three scenarios, using parameter values selected to be broadly consistent with reported colonization and clearance rates in hospital AMR transmission studies (baseline values: $\beta r = 0.18$, $\beta s = 0.10$, $\gamma s = 0.12$, $\gamma r = 0.05$, $\alpha = 0.02$, $\mu = 0.10$, shedding rate $s = 0.30$, natural decay $d = 0.15$, per day): (1) a baseline scenario with conventional treatment and moderate environment-to-patient coupling; (2) an enhanced effluent treatment scenario (increased τ, representing addition of advanced oxidation or UV disinfection); and (3) a combined scenario adding a 30% reduction in the resistant-organism patient-to-patient transmission rate (βr), representing concurrent antimicrobial stewardship. These parameters are illustrative rather than site-calibrated and are intended to demonstrate model structure and behavior; real-world application requires fitting to site-specific longitudinal genomic and clinical surveillance data as described in Section 3.3.

Results

Simulated resistome profile across the hospital-to-environment continuum

Consistent with the literature synthesized in Section 2, an illustrative resistome profile constructed to reflect reported qualitative patterns shows beta-lactamase and aminoglycoside resistance gene classes dominating at ward-level and hospital effluent sampling points, with fluoroquinolone, MLSB, sulfonamide, and tetracycline resistance genes present at lower but non-trivial relative abundance. Figure 2 illustrates this pattern and shows partial, non-uniform attenuation of ARG classes moving from WWTP influent to WWTP effluent and further downstream, mirroring reports that conventional treatment reduces but does not eliminate the resistance signature of hospital-derived wastewater, and that attenuation is not uniform across gene classes or bacterial taxa.

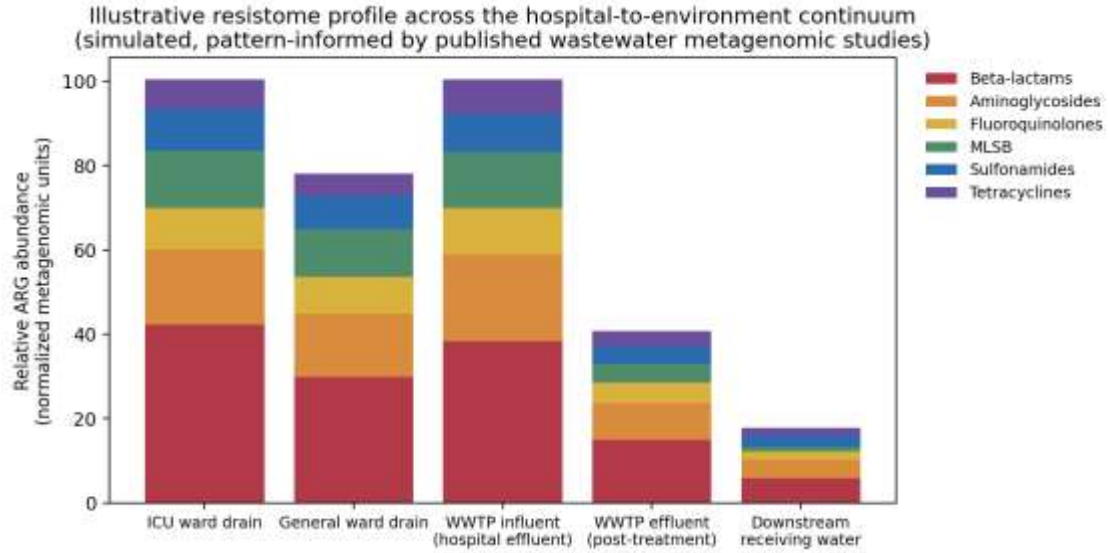

Figure 2. Illustrative relative abundance of major antimicrobial resistance gene (ARG) classes across five sampling points spanning intensive care and general ward drains, wastewater treatment plant (WWTP) influent and effluent, and downstream receiving water. Values are simulated for illustrative purposes but constructed to be qualitatively consistent with patterns reported in published hospital wastewater metagenomic studies.

Modeled impact of treatment and stewardship interventions

Simulation of the coupled compartmental model (Section 3.3–3.4) shows that hospital effluent resistant-organism load and in-hospital resistant colonization prevalence are both highly sensitive to the treatment removal parameter τ and, more modestly on its

own, to reductions in patient-to-patient transmission. Under the baseline scenario, resistant colonization prevalence among hospitalized patients rose to approximately 78.7% and normalized effluent resistant load reached approximately 1.18 (arbitrary relative units) by day 180. Intensifying effluent treatment alone (increasing τ) reduced day-180 resistant colonization prevalence to approximately 54.5% and effluent resistant load to approximately 0.23 — a reduction of roughly 80% in environmental load despite only a partial reduction in hospital-side colonization. Combining enhanced treatment with a 30% reduction in resistant-organism transmission (representing concurrent antimicrobial stewardship) produced the largest reductions across both compartments, lowering colonization prevalence to approximately 43.5% and effluent load to approximately 0.19.

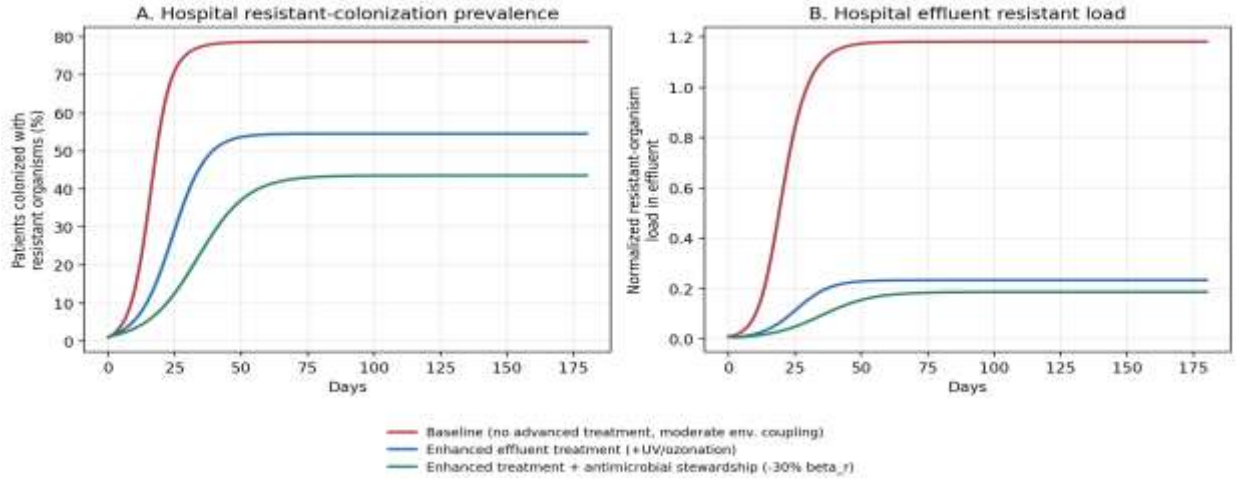

Figure 1. Simulated dynamics of (A) hospital resistant-colonization prevalence and (B) normalized resistant-organism load in hospital effluent under three intervention scenarios over a 180-day horizon, generated from the compartmental model described in Section 3.3.

These results, while illustrative rather than empirically fitted, reproduce a qualitative pattern reported in environmentally explicit AMR transmission models more broadly: because the environmental compartment acts as a self-reinforcing reservoir that re-seeds patient colonization even when patient-to-patient transmission is reduced, interventions targeting effluent treatment and clinical transmission in combination outperform either intervention alone, and the marginal benefit of stewardship-only interventions is attenuated when environmental coupling (k) is high. This has a direct practical implication: genomic surveillance data indicating a high or rising environmental resistant load, even in the presence of stable or declining clinical isolate resistance, may signal that effluent-side interventions (treatment upgrades, plumbing remediation) rather than antimicrobial stewardship alone are needed to achieve further reductions.

Illustrative surveillance summary table

Scenario	Day-180 resistant colonization prevalence (Cr, %)	Day-180 normalized effluent resistant load (E)
Baseline (conventional treatment)	78.7%	1.181
Enhanced effluent treatment	54.5%	0.234
Enhanced treatment + stewardship (−30% resistant transmission)	43.5%	0.187

Table 1. Simulated end-of-horizon (day 180) hospital resistant colonization prevalence and normalized effluent resistant-organism load under three intervention scenarios.

Discussion

This paper's central contribution is structural rather than empirical: it demonstrates how genomic surveillance outputs and epidemiological transmission models can be formally coupled, using hospital effluent as the linking compartment, to move beyond descriptive resistome characterization toward an intervention-evaluable system. Three implications follow from the illustrative results in Section 4.

First, the finding that environmental treatment intensification produced a proportionally larger reduction in effluent resistant load than in patient-side colonization prevalence underscores that hospital effluent should not be treated merely as a passive readout of clinical resistance, but as a compartment with its own dynamics — shaped by shedding, decay, and treatment — that can itself become a self-sustaining reservoir feeding back into colonization risk. This is consistent with reports that some resistant, environmentally hardy taxa persist or even increase in relative abundance through conventional wastewater treatment, and reinforces the case, made in prior environmentally explicit modeling work, for evaluating hospital-level and municipal WWTP-level infrastructure investments as AMR control interventions in their own right, alongside antimicrobial stewardship.

Second, the combined-intervention scenario outperforming either single intervention illustrates a general property of models with reservoir feedback: single-lever interventions are systematically less effective when an active environmental reservoir exists, because the reservoir continues to re-seed the compartment the intervention targets. Practically, this suggests that AMR action plans anchored solely in antimicrobial stewardship, without corresponding attention to hospital wastewater treatment and plumbing infrastructure, may substantially underachieve their targeted reductions in resistant colonization, particularly in older healthcare facilities with aging or under-maintained sewer infrastructure.

Third, the source-attribution component of the proposed genomic workflow (distinguishing gut-derived from sewer-infrastructure-associated resistance signal) is not a peripheral technical detail but a prerequisite for correctly parameterizing the model's shedding and environmental persistence terms. Without this distinction, a rise in effluent ARG abundance could be misattributed to worsening patient colonization when it in fact reflects biofilm-associated amplification within hospital plumbing, leading to a mismatched intervention (stewardship) being prioritized over the one actually needed (infrastructure remediation or targeted disinfection).

More broadly, these findings support the integration of genomic wastewater surveillance into One Health AMR monitoring architectures, as recommended in recent policy-oriented reviews. Hospital-effluent genomic surveillance, coupled to dynamic modeling as outlined here, offers three practical surveillance functions that complement clinical isolate-based systems: early detection of emerging resistance mechanisms before they are captured through clinical culture (given that colonization typically precedes and outnumbers clinical infection); population-level, unbiased estimation of resistance burden that does not depend on which patients happen to be tested; and a quantitative basis for comparing the relative value of clinical versus environmental interventions at a given facility.

A note on the data presented in this paper: *The resistome profile (Figure 2) and transmission-model outputs (Figure 1, Table 1) are illustrative simulations constructed by the author to demonstrate the proposed analytical framework and are not derived from a specific empirical hospital dataset. They are designed to be qualitatively consistent with patterns reported in the peer-reviewed and preprint*

literature cited in Section 2 and the reference list. Application of this framework to a real facility requires substitution of site-specific longitudinal genomic sequencing and clinical surveillance data as described in Section 3.

Limitations

Several limitations should be considered when applying this framework. The resistome profile and transmission model parameters presented in Section 4 are illustrative and pattern-informed rather than fitted to site-specific empirical data; real deployments require longitudinal genomic and clinical surveillance data of the kind described in Section 3.1–3.2 for proper parameter estimation and model validation. The compartmental model itself is deliberately parsimonious: it does not explicitly represent multiple resistance mechanisms or organism-specific dynamics, horizontal gene transfer between co-colonizing taxa, heterogeneity in antimicrobial exposure across ward types, or stochastic effects that may dominate in smaller patient populations. Metagenomic sequencing approaches, even at high depth, have documented limitations in detecting rare ARGs and in fully resolving strain-level or plasmid-level dynamics without complementary long-read sequencing or Hi-C-based genome linkage methods. Finally, attribution of wastewater signal to specific upstream sources (individual wards, specific patients, or non-human contributors such as pharmaceutical residues) remains only partially resolved by current bioinformatic methods and is an active area of methodological development.

Conclusion

Urban hospital effluent sits at a uniquely informative junction between clinical AMR burden and environmental AMR dissemination. This paper has argued, and illustrated through a worked simulation, that genomic surveillance of this effluent gains substantially greater public health and infection-control value when coupled to an epidemiological transmission model that treats the effluent system as an active, dynamically coupled reservoir rather than a passive sink. The proposed framework — spanning a five-point genomic sampling architecture, a genome-resolved metagenomic and metatranscriptomic sequencing workflow with source attribution, and a compartmental model linking patient colonization to environmental resistance load — provides a template that can be parameterized with site-specific data to support hospital infection control decision-making, regional wastewater treatment planning, and integration into broader One Health AMR early-warning systems. Future work should prioritize longitudinal, multi-site validation of the coupled framework, extension to explicitly represent horizontal gene transfer and multi-drug resistance co-occurrence, and development of standardized reporting formats that allow genomic wastewater surveillance data to be directly ingested into regional and national AMR risk models.

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