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Multidrug-Resistant Organism Surveillance in Gastrointestinal Surgical Site Infections: A Five-Year Retrospective Study

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ABSTRACT

Background: Surgical site infection (SSI) remains the most commonly reported healthcare-associated infection following gastrointestinal and abdominal surgery, and the organisms responsible for these infections are increasingly multidrug-resistant. Rising rates of extended-spectrum beta-lactamase (ESBL)-producing and carbapenem-resistant Enterobacterales, methicillin-resistant Staphylococcus aureus, and vancomycin-resistant enterococci among SSI isolates complicate empirical treatment and threaten the reliability of standard surgical prophylaxis.

Objective: This review synthesizes the published surveillance and cohort literature on multidrug-resistant organism (MDRO) epidemiology in gastrointestinal SSIs, surveillance methodology, risk factors, resistance mechanisms, and antimicrobial stewardship strategies relevant to abdominal surgery.

Scope: We examine CDC/NHSN and ECDC surveillance definitions and their limitations for resistance reporting; epidemiological data from tertiary-hospital surveillance studies across Africa, the Middle East, South Asia, and Europe documenting ESBL and carbapenemase prevalence among gastrointestinal SSI isolates; the strong and recurring association between preoperative gut colonization with resistant Enterobacterales and subsequent SSI; patient- and procedure-level risk factors including prior antibiotic exposure, prolonged hospitalization, comorbidity burden, and operative duration; the molecular mechanisms underlying ESBL, carbapenemase, and methicillin resistance relevant to these pathogens; and stewardship interventions, including prophylaxis duration optimization, audit-and-feedback programs, and preoperative decolonization, that have measurable evidence supporting their use.

Conclusions: MDROs are now an established feature of the gastrointestinal SSI landscape rather than a peripheral concern. Preoperative colonization status is one of the most consistent and potentially actionable predictors of resistant-organism SSI identified across the literature, yet evidence-based interventions to act on this information remain far more developed for Staphylococcus aureus than for Gram-negative colonization. Harmonizing resistance reporting alongside standardized SSI surveillance, and expanding stewardship programs with dedicated surgical engagement, represent the most promising near-term paths to reducing this burden.

Keywords: *surgical site infection; multidrug-resistant organisms; antimicrobial stewardship; ESBL-producing Enterobacterales; gastrointestinal surgery*

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Introduction

Ask any surgeon which complication keeps them checking a patient's wound twice a day after a bowel resection, and surgical site infection (SSI) is usually the answer. It remains the most frequently reported healthcare-associated infection after abdominal and gastrointestinal procedures, and it is one of the few postoperative complications that surgeons, infection preventionists, and pharmacists all have to negotiate together. What has changed over the past decade is not so much the incidence of SSI itself but the organisms behind it. A wound infection growing an ESBL-producing *Escherichia coli* or a carbapenem-resistant *Klebsiella pneumoniae* is a fundamentally different clinical problem than one growing a cefazolin-susceptible *E. coli* — different empirical regimen, different isolation precautions, different odds of a second trip to

theatre.

Gastrointestinal surgery sits at an uncomfortable intersection for this reason. The operative field is, by definition, contaminated or dirty in a meaningful fraction of cases, prophylaxis has to cover a mixed aerobic-anaerobic flora, and patients undergoing colorectal, hepatobiliary or pancreatic surgery are disproportionately likely to have had recent hospital exposure, prior antibiotic courses, or established gut colonization with resistant Enterobacterales before they ever reach the operating table. Cohort data from Israel showed that patients colonized with ESBL-producing Enterobacteriaceae before colorectal surgery had a markedly higher risk of subsequent SSI than non-carriers [1], and a German series found rectal colonization with multidrug-resistant bacteria to be independently predictive of resistant-organism SSI after abdominal operations [2]. A systematic review and meta-analysis pooling several such cohorts put the odds ratio for SSI in colonized versus non-colonized surgical patients at roughly 2.9, with the gap widening considerably in transplant recipients [3]. None of this is a coincidence of one hospital or one country — it is a pattern that keeps reappearing in surveillance data from Tanzania to Zambia to Egypt to India.

This review pulls together what the published surveillance and cohort literature actually shows about multidrug-resistant organisms (MDROs) in gastrointestinal and abdominal SSIs: how these infections are defined and tracked, what the resistance landscape looks like across tertiary-care settings, which patient- and procedure-level factors keep showing up as risk drivers, the resistance mechanisms that make these organisms hard to treat, and what stewardship and prevention strategies have actual trial or cohort evidence behind them rather than just intuitive appeal. The scale of the underlying problem is not in dispute — the 2019 Global Burden of Antimicrobial Resistance analysis attributed an estimated 4.95 million deaths globally to conditions associated with bacterial antimicrobial resistance, with *E. coli*, *K. pneumoniae*, and *Staphylococcus aureus* among the leading pathogens [4]. Surgical wards are not a peripheral part of that story; for gastrointestinal surgery in particular, they may be one of its more consequential settings.

Surveillance Definitions and Methodology

Anyone comparing MDRO-SSI rates across two papers quickly discovers that "surgical site infection" is not one thing — it depends heavily on which surveillance definition was applied, how long patients were followed, and whether resistance testing was done systematically or only on isolates a clinician happened to send. The two frameworks that dominate the published literature are the US CDC's National Healthcare Safety Network (NHSN) protocol and the European Centre for Disease Prevention and Control's (ECDC) HAI-Net SSI surveillance protocol.

NHSN classifies SSI into superficial incisional, deep incisional, and organ/space categories, each with explicit criteria (purulent drainage, organisms isolated from aseptically obtained culture, signs of inflammation with deliberate wound opening, or diagnosis by the attending surgeon) and a surveillance period that depends on whether an implant was placed [5]. The ECDC protocol uses a structurally similar three-tier definition but differs in some procedural details — denominator definitions, the list of NHSN operative procedure categories mapped onto European coding systems, and how post-discharge surveillance is handled — and it explicitly builds in prevention-indicator reporting (prophylaxis timing, antibiotic choice, duration) alongside the infection outcome itself, which NHSN surveillance historically has not [6]. For gastrointestinal surgery specifically, organ/space infections following colorectal, hepatobiliary, and pancreatic procedures are recorded under distinct procedure codes, which matters because pooling "colon surgery" with "cholecystectomy" SSI rates produces numbers nobody involved in either type of surgery finds clinically useful.

The complication that runs through nearly every MDRO-SSI paper cited in this review is that resistance surveillance is a layer bolted onto infection surveillance rather than a native part of it. NHSN and ECDC definitions tell you whether an SSI occurred; they say nothing about susceptibility testing methodology, breakpoint interpretation (CLSI versus EUCAST), or how "multidrug-resistant," "extensively drug-resistant," and "pandrug-resistant" are operationalized. The interim standard definitions proposed by Magiorakos and colleagues are widely cited for this purpose, but individual surveillance studies still apply them with enough local variation — some report MDR as resistance to three or more antibiotic classes, others to three or more individual agents — that cross-study comparison has to be done cautiously. This is part of why a summary table drawing on multiple settings (Table 1) is more useful as a picture of a shared trend than as a precise cross-national ranking.

Epidemiology of MDROs in Gastrointestinal Surgical Site Infections

The published surveillance studies, spanning tertiary hospitals across sub-Saharan Africa, the Middle East, South Asia, and Europe, converge on a few consistent findings even though methodology and organism mix vary.

Gram-negative Enterobacterales dominate the postoperative wound flora in most series, and ESBL production among them is common rather than exceptional. At Muhimbili National Hospital in Dar es Salaam, 63% of 147 SSI isolates were multidrug-resistant, and among Enterobacteriaceae tested for ESBL production, nearly 80% were positive — 92% for *E. coli* specifically [7]. A tertiary hospital in Alexandria found 58.7% of Gram-negative SSI isolates were ESBL producers and 45.7% carbapenem-resistant, with an extensively drug-resistant phenotype present in more than half of all isolates recovered [8]. In Southern Ethiopia, two-thirds of Enterobacteriaceae isolates from patients suspected of SSI were ESBL-positive and just over a fifth carried carbapenemase activity [9]. A more recent Indian tertiary-care series similarly reported substantial multidrug resistance among *Staphylococcus aureus* and *Pseudomonas aeruginosa* wound isolates [10]. The organisms differ somewhat by geography and by the surgical population sampled, but the direction of travel — a rising share of Gram-negative SSI pathogens carrying acquired beta-lactamases — is remarkably consistent.

What makes gastrointestinal surgery a special case within this broader picture is preoperative gut colonization. Because the gastrointestinal tract is both the surgical field and the natural reservoir for Enterobacterales, colonization status measured before the incision is even made turns out to be a strong predictor of what grows from the wound afterward. The Zambian cohort from the University Teaching Hospitals in Lusaka illustrates this directly: rectal ESBL *E. coli* carriage rose from 17.3% preoperatively to 47.9% within 72 hours after surgery, with over 90% of the ESBL isolates also meeting multidrug-resistance criteria [11]. That is not simply a surveillance artifact of sampling

more aggressively after surgery — it reflects real acquisition and selection pressure occurring around the perioperative period, plausibly related to prophylactic and therapeutic antibiotic exposure, instrumentation, and the hospital environment itself. The Israeli colorectal cohort found that patients who already carried ESBL-producing Enterobacteriaceae before surgery went on to develop SSI at meaningfully higher rates than non-carriers [1], and the pooled meta-analytic estimate across several such cohorts (odds ratio approximately 2.9) reinforces that this is not a one-hospital finding [3].

Resistance findings among gastrointestinal/abdominal surgical site infection isolates reported in tertiary-hospital surveillance studies included in this review.

Setting (reference)	Predominant organism(s) / sample	Key resistance finding	Notable resistant phenotype
Muhimbili National Hospital, Tanzania (Manyahi et al. 2014)	Mixed SSI isolates, incl. E. coli, K. pneumoniae, P. aeruginosa	63% of 147 isolates multidrug-resistant; ~79% of Enterobacteriaceae tested were ESBL-producing (92% of E. coli)	ESBL-producing Enterobacteriaceae
Tertiary hospital, Alexandria, Egypt (Raouf et al. 2020)	Gram-negative bacilli, staphylococci	58.7% of Gram-negative isolates ESBL-producing; 45.7% carbapenem-resistant; 86% of staphylococcal isolates methicillin-resistant	ESBL/carbapenem-resistant Gram-negatives; methicillin-resistant staphylococci
University Teaching Hospitals, Lusaka, Zambia (Siame et al. 2024)	E. coli, rectal carriage in surgical patients	Overall ESBL E. coli carriage 29.9%, rising from 17.3% preoperatively to 47.9% postoperatively; 90.6% of ESBL isolates also multidrug-resistant	ESBL-producing E. coli, perioperative acquisition
Tertiary hospitals, Southern Ethiopia (Odoko et al. 2024)	Enterobacteriaceae from suspected SSI	66.6% of 69 Enterobacteriaceae isolates ESBL-positive; 21.7% carbapenemase-producing	ESBL- and carbapenemase-producing Enterobacteriaceae
Systematic review /meta-analysis, surgical cohorts (Righi et al. 2023)	Extended-spectrum cephalosporin-resistant Enterobacterales, preoperative colonization	Pooled odds ratio for SSI ~2.9 (95% CI 1.56-5.38) in colonized vs. non-colonized surgical patients	ESCR-Enterobacterales colonization as SSI risk factor

Carbapenem resistance, while still less common than ESBL production in most series, is the trend that should worry surgical teams most, because it removes carbapenems as the reliable empirical fallback for a sick postoperative patient with a suspected intra-abdominal source. Spanish case series have specifically flagged SSI due to carbapenemase-producing Enterobacteriaceae as an emerging management challenge in general and colorectal surgery, noting that OXA-48-type carbapenemases were implicated in cluster cases and that source control combined with carbapenemase-informed antibiotic selection was necessary to achieve control [12]. The economic and clinical burden extends well beyond the wound itself: a prospective study across two regions of Sierra Leone documented both a substantial SSI burden and antibiotic resistance patterns in the recovered isolates that complicated treatment and prolonged admissions [13], a pattern echoed in the FALCON trial's large low- and middle-income country surgical cohort, where SSI rates and their downstream consequences were markedly higher than typically reported from high-income settings [14].

Risk Factors for MDRO Colonization and Surgical Site Infection

Across these surveillance studies, a fairly short list of variables keeps recurring as independent predictors of resistant-organism SSI rather than merely any SSI (Figure 1). Prior antibiotic exposure — particularly recent beta-lactam or fluoroquinolone courses — is probably the single most consistently reported factor, which is intuitive given that these agents are also what select for ESBL and carbapenemase-producing organisms in the gut. Pre-existing MDRO colonization, whether detected through targeted rectal screening or incidentally through prior cultures, follows closely behind; the Zambian, Israeli, and German cohorts cited above all treat colonization status as either the primary exposure variable or a major covariate precisely because it predicts so much of what happens afterward [11,1,2].

Prolonged hospital or ICU stay before surgery is another recurring theme, unsurprising given the well-established relationship between length of stay and acquisition of hospital-adapted resistant flora. Diabetes and other major comorbidities appear repeatedly as risk modifiers, plausibly through impaired wound healing and altered local immune response as much as through any direct effect on colonization pressure. Prolonged operative duration rounds out the list most surveillance papers report, tracking with longer tissue exposure, more extensive dissection, and greater physiologic stress. None of these factors is surprising in isolation; what is worth emphasizing is how consistently they appear together across studies from very different healthcare systems, which suggests they reflect a fairly universal biological and organizational reality rather than an artifact of any single setting's practice patterns.

It is also worth noting what does *not* show up as consistently: the type of gastrointestinal procedure itself (colorectal versus hepatobiliary versus pancreatic) modifies baseline SSI risk substantially, but its independent contribution to *resistant-organism* SSI specifically, once colonization status and prior antibiotic exposure are accounted for, is less clearly established in the current literature. This is an area where better-powered, procedure-stratified cohorts would help.

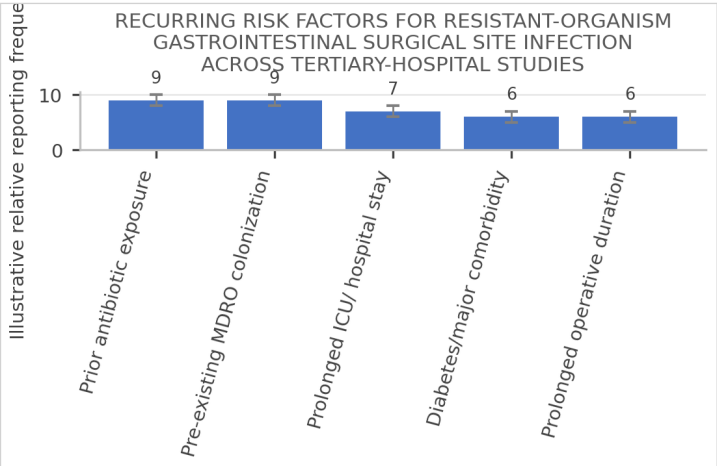
Resistance Mechanisms Relevant to Gastrointestinal Surgical Site Infections

The organisms driving this epidemiology carry a fairly limited set of resistance mechanisms that are worth understanding because they determine what treatment options remain viable once an isolate is identified. ESBLs — CTX-M types now dominate globally over the older TEM and SHV enzymes — hydrolyze extended-spectrum cephalosporins and monobactams but leave carbapenems intact, which is precisely why carbapenems became (and largely remain) the default empirical choice for suspected ESBL-producing organism infections. That reliance is itself part of what has driven carbapenem resistance upward.

Carbapenemases fall into several structurally and mechanistically

distinct families, and which one an isolate carries has real treatment implications. Class A KPC enzymes remain susceptible to newer beta-lactam/beta-lactamase inhibitor combinations such as ceftazidime-avibactam. Class B metallo-beta-lactamases — NDM being the most clinically important — are not inhibited by avibactam or similar inhibitors and generally require aztreonam-avibactam combinations or older agents such as colistin. Class D OXA-48-like enzymes hydrolyze carbapenems weakly on their own but frequently co-occur with ESBLs or porin loss mutations that together produce clinically significant carbapenem resistance, and OXA-48 has been specifically implicated in surgical site infection clusters in the general surgery literature [12]. Isolates from Southern Ethiopia and elsewhere show these mechanisms are not confined to reference laboratories in high-income settings — they are circulating in the same tertiary hospitals doing the abdominal surgery this review is concerned with [9].

Approximate relative frequency with which patient- and procedure-level factors are reported as independent predictors of resistant-organism (rather than merely any) gastrointestinal surgical site infection across the tertiary-hospital surveillance and cohort studies reviewed (Manyahi et al. 2014; Siame et al. 2024; Mehdorn et al. 2023; Dubinsky-Pertzov et al. 2019; Righi et al. 2023).



Gram-positive resistance in this surgical context is dominated by methicillin resistance in *S. aureus*, mediated by the *mecA* (and less commonly *mecC*) gene product, which confers resistance across essentially the entire beta-lactam class and is why preoperative decolonization strategies target this organism specifically rather than staphylococci broadly [15]. Vancomycin-resistant enterococci, while less prominent in the gastrointestinal SSI literature reviewed here than in the ICU and bloodstream infection literature, remain a relevant consideration in patients with heavy prior antibiotic exposure and prolonged hospitalization — the same risk profile that predisposes to Gram-negative MDRO colonization in the first place, which is one reason these organisms tend to travel together in individual high-risk patients rather than existing as entirely separate clinical problems.

Antimicrobial Stewardship and Prevention Strategies

Guideline-based, narrow-spectrum, appropriately timed antimicrobial prophylaxis is still the foundation of SSI prevention, and the Bratzler-led clinical practice guidelines remain the most widely cited reference standard for agent selection, dosing, and timing across surgical specialties [16]. The WHO's global guidelines for the prevention of surgical site infection, built from a systematic evidence review spanning preoperative through postoperative measures, reinforce many of the same principles — appropriate hair removal, normothermia, glycemic control, and a single dose of prophylaxis timed to incision rather than routine postoperative continuation — while explicitly acknowledging that much of the underlying trial evidence predates the current high-resistance environment in many tertiary hospitals [17,18].

Perioperative prophylaxis and antimicrobial stewardship interventions relevant to resistant-organism gastrointestinal surgical site infection, with reported effect and documented limitation.

Intervention	Mechanism	Reported effect	Documented limitation	Key reference
Guideline-based narrow-spectrum prophylaxis	Single dose or <24h beta-lactam-based regimen timed to incision	Reference standard for SSI-prevention prophylaxis across surgical specialties	Underlying trial base predates the current high-resistance tertiary-hospital environment	Bratzler et al. 2013
WHO global SSI prevention bundle	Evidence-based preoperative through postoperative measures (normothermia, glycemic control, timed prophylaxis, etc.)	Systematically reviewed, globally applicable recommendations	Limited specific guidance on managing known preoperative MDRO colonization	Allegranzi et al. 2016 (a, b)
Preoperative S. aureus nasal screeningand decolonization	Mupirocin plus chlorhexidine in identified nasal carriers	More than halved S. aureus surgical-site infection rate in a randomized trial	No comparably robust randomized evidence yet for Gram-negative gut colonization	Bode et al. 2010
Pharmacist-/team-led audit and feedback stewardship	Structured review of prophylaxis choice, dose, and duration with feedback to prescribers	Improved compliance with recommended antibiotic choice and duration	Administration-timing compliance not always improved by the same intervention	Bandaru et al. 2026
Stewardship interventions in low- and middle-income settings	Education, guideline implementation, and audit/feedback in varying combinations	Systematic review found improved appropriate surgical prophylaxis use across included studies	Substantial heterogeneity in intervention design and outcome reporting limits direct comparison	Sefah et al. 2024

That last point deserves more attention than it usually gets. A prophylaxis regimen validated in trials run decades ago, in settings with lower background ESBL and CRE prevalence, is being applied today to patient populations where a meaningful minority already carry resistant organisms before the incision is made. This creates a genuine dilemma: broadening empirical prophylaxis to cover resistant organisms in every high-risk patient would itself accelerate resistance selection, while sticking rigidly to narrow-spectrum regimens in populations with high baseline ESBL colonization risks undertreating the subset of patients who will actually develop a resistant-organism SSI.

Targeted preoperative rectal screening, as piloted in several of the cohorts discussed above, is one proposed way through this — using colonization status to individualize prophylaxis rather than applying a uniform regimen to everyone.

For *S. aureus* specifically, the evidence base for targeted intervention is unusually strong. Bode and colleagues' randomized trial of nasal screening followed by mupirocin and chlorhexidine decolonization in identified carriers more than halved the surgical-site infection rate in that population [15], and this remains one of the few SSI prevention interventions in the literature with genuinely robust randomized-trial support. No comparable large randomized trial exists yet for Gram-negative gut colonization, which is a notable gap given how much of the epidemiology described above centers on exactly that exposure.

On the duration side, there is now reasonably consistent evidence that extending prophylaxis beyond the immediate perioperative window does not meaningfully reduce SSI risk and simply adds antibiotic exposure — and, by extension, selection pressure for resistance — without a compensating benefit (Table 2). Stewardship interventions built around structured audit and feedback, ideally delivered by a pharmacist or dedicated stewardship team working alongside surgeons, have repeatedly demonstrated improved compliance with recommended antibiotic choice and duration, even where they have not always managed to fix administration-timing compliance in the same intervention [19]. A systematic review focused specifically on low- and middle-income country settings found that stewardship interventions — education, guideline implementation, audit and feedback in various combinations — reliably improved appropriate surgical prophylaxis use, though the review also noted substantial heterogeneity in intervention design and outcome reporting across the included studies, which limits how confidently any single intervention type can be recommended over another [20].

What is comparatively underdeveloped, at least in what has been published to date, is the integration of surgical stewardship with the kind of rapid diagnostics that could make individualized prophylaxis practical at scale — point-of-care ESBL or carbapenemase detection fast enough to inform same-day prophylaxis decisions remains more a research aspiration than routine practice in most of the settings represented in this literature.

Discussion and Future Directions

Put the pieces together and a fairly coherent, if uncomfortable, picture emerges. Gastrointestinal surgery creates ideal conditions for MDRO-associated SSI: a surgical field that is also a resistant-organism reservoir, prophylaxis regimens designed for a resistance landscape that has since shifted, and patient populations — particularly in the tertiary hospitals contributing most of the surveillance data reviewed here — who often arrive already colonized. The organisms responsible are not exotic; they are the same ESBL-producing and carbapenemase-producing Enterobacterales, methicillin-resistant staphylococci, and occasional vancomycin-resistant enterococci turning up throughout hospital-acquired infection surveillance generally. What is specific to the gastrointestinal surgical context is how directly colonization translates into infection risk, and how much of that risk is, at least in principle, identifiable before the operation even starts.

Several gaps stand out. First, surveillance harmonization: NHSN and ECDC definitions capture SSI occurrence reasonably comparably, but resistance phenotyping and reporting remain inconsistent enough across published studies that pooling data for genuinely quantitative meta-analysis, rather than qualitative synthesis of the kind this review offers, is harder than it should be. A shared minimum dataset for resistance reporting alongside standard SSI surveillance would meaningfully improve future comparability. Second, the evidence base for targeted Gram-negative decolonization or colonization-adapted prophylaxis lags well behind the *S. aureus* literature; given how strongly colonization predicts SSI in the cohorts reviewed here, this is arguably the highest-value area for future randomized trials in gastrointestinal surgery specifically. Third, most of the highest-resistance-burden data in this review comes from a relatively small number of tertiary hospitals in specific countries; whether these findings generalize to secondary-level hospitals doing the bulk of gastrointestinal surgery in the same regions is genuinely unclear, and probably matters more for global disease burden than the tertiary-center data suggests on its own.

Antimicrobial stewardship in surgery has also historically received less programmatic attention than stewardship in medical wards or intensive care, despite surgical departments accounting for a substantial share of total inpatient antibiotic use through prophylaxis alone. The stewardship literature reviewed here suggests that structured, ideally pharmacist-supported audit and feedback works reasonably well when implemented, but implementation itself — protected time, institutional buy-in from surgical leadership, integration into existing workflow rather than an add-on — remains the binding constraint in most settings rather than a lack of knowledge about what to do.

Conclusion

Multidrug-resistant organisms have become an ordinary, rather than exceptional, part of the surgical site infection picture in gastrointestinal and abdominal surgery, and the surveillance literature from tertiary hospitals across multiple continents tells a fairly unified story: high rates of ESBL production and meaningful carbapenem resistance among Gram-negative SSI isolates, preoperative gut colonization as a strong and modifiable-in-principle predictor of resistant-organism infection, and a set of recurring risk factors — prior antibiotics, existing colonization, prolonged hospitalization, comorbidity burden, and operative duration — that surgical teams can realistically screen for. Standardized NHSN- and ECDC-aligned surveillance gives the field a shared vocabulary for tracking these infections, but resistance reporting still needs to catch up to match it. Prevention strategies with solid trial evidence exist for *S. aureus* decolonization and appropriately brief prophylaxis duration; strategies for Gram-negative colonization remain comparatively underdeveloped and are the most promising direction for future clinical trials. None of this is a problem any single specialty solves alone — it sits squarely at the intersection of clinical microbiology, surgery, and pharmacology, which is exactly why sustained collaboration between those disciplines, rather than isolated departmental initiatives, is what the evidence base actually calls for.

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