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Review Article

The gastrointestinal tract as a potential reservoir for *Pseudomonas aeruginosa*: implications for surgical site infections and potential orthopedic complications

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ABSTRACT

Pseudomonas aeruginosa is an opportunistic pathogen with a remarkable capacity for antimicrobial resistance and biofilm formation. It frequently complicates care in immunocompromised and hospitalized patients. The gastrointestinal (GI) tract may serve as a reservoir for subsequent surgical and orthopedic infections, yet this potential interconnection remains underexplored. This narrative review synthesizes findings from clinical and experimental studies published from 2000 to 2025. Evidence was evaluated across gastroenterology, surgical, and orthopedic disciplines, with emphasis on microbial translocation, strain matching, biofilm formation, and antimicrobial resistance. Gastrointestinal colonization with *P. aeruginosa* may predispose patients to postoperative infections, particularly with dysbiosis, antibiotic use, immunosuppression, or intestinal barrier disruption. Strain matching between intestinal and extraintestinal isolates supports the GI tract as a potential source of infection. Biofilm formation on orthopedic implants and antimicrobial resistance further complicate treatment. However, the direct contribution of intestinal colonization to orthopedic implant-related infections remains unclear. Further research should determine whether screening for *P. aeruginosa* colonization and targeted prevention strategies can reduce postoperative infections in surgical patients and improve clinical outcomes through cross-specialty collaboration.

Keywords: *Pseudomonas aeruginosa*, Gastrointestinal colonization, Microbial translocation, Surgical site infection, Orthopedic infection, Antimicrobial resistance

INTRODUCTION

Pseudomonas aeruginosa is a Gram-negative opportunistic bacterium whose virulence has made it a leading cause of hospital-acquired acute and systemic infections.[1] *P. aeruginosa*'s ability to metabolize a wide range of organic molecules for nutrients allows it to survive in various environments such as soil, human skin/mucosa, humidifiers, and respirators.[2] The ability of *P. aeruginosa* to survive in diverse environments

contributes to its ubiquity.[1] It often leads to the formation of large reservoirs in hospital settings, increasing the risk of exposure and infection among hospitalized and immunocompromised patients.[2] Additionally, *P. aeruginosa*'s remarkable capacity for antimicrobial resistance (AMR) and biofilm formation complicates treatment of infected patients, particularly in transplant and surgical patients.[1] While the pathogenicity of *P. aeruginosa* is of concern, emerging evidence suggests the gastrointestinal (GI) tract may serve as a reservoir for infection.[3]

The gut microbiome is a rich and diverse environment that contains bacteria in mutualistic relationships with its host, protecting the host from pathogens and aiding in nutrient acquisition.[4] However, the use of antimicrobials disrupts this stable environment by reducing bacterial density, thereby creating an opportunity for pathogens like the otherwise transient *P. aeruginosa* to invade. With increased colonization efficiency in hospitalized patients, *P. aeruginosa* carriage in the GI tract has been identified as a potential risk factor for infection and for increasing the likelihood of transmission to other patients.[5] *P. aeruginosa* that has colonized the GI tract represents a possible source of bloodstream infections and mortality for immunocompromised patients, but also a major infectious pathogen of the lungs; these infections often arise as a result of airway contamination by gastrointestinal flora or hematogenous dissemination from the intestine.[3] Evidence of gut-origin *P. aeruginosa* infections underscores the value of further exploration of the GI tract's role as a reservoir that interconnects with infections across specialties, including surgical site infections (SSI) in various surgical and orthopedic contexts.

P. aeruginosa is a significant contributor to orthopedic infections, in addition to its role in GI complications, accounting for up to 20% of Gram-negative bacterial implant-associated bone and joint infections in orthopedic settings.[5-6] *P. aeruginosa*'s presence across GI, surgical, and orthopedic complications, while seemingly separate, may be linked by *P. aeruginosa*'s ability to disseminate from hidden reservoirs like the GI tract.[3] This interconnection highlights the need for a more systematic model of *P. aeruginosa* incidence across specialties. This review aims to investigate the role of *P. aeruginosa* in increasing the risk of surgical site and orthopedic implant-related infections, focusing on the pathogen's function as an intestinal reservoir, a dissemination pathway, and a contributor to surgical complications.

METHODS

A structured narrative review was conducted to examine the gastrointestinal tract as a potential reservoir for *P. aeruginosa* and its possible implications for surgical and orthopedic infections. The review aimed to investigate the pathogenesis of *P. aeruginosa*, gastrointestinal colonization, surgical complications, orthopedic infections, treatment, and prevention. This review identified relevant studies through a structured search of PubMed and Google Scholar. Key search terms included "*P. aeruginosa*," "gastrointestinal colonization," "surgical site infection," "orthopedic infection," "biofilm," "antimicrobial resistance," and combinations of these keywords. The search conducted by the authors was restricted to peer-reviewed journal articles, clinical trials, literature reviews, and meta-analyses published in English from 2000 to 2025. The findings were reviewed and narratively synthesized to provide a comprehensive understanding of the potential relationship among gastrointestinal colonization, surgical site infections, and orthopedic infections and to identify areas for future investigation.

PATHOGENESIS OF *P. AERUGINOSA*

The pathogenic potential of *P. aeruginosa* can be attributed to its production of potent toxins and degradative enzymes through highly specific secretion systems.[7-8] These secretion systems include five main types, with type II and III being the most crucial and complex for *P. aeruginosa* virulence.[7] The type II secretion system (T2SS) is worth noting for its ability to transport large proteins with virulence functions from the cytoplasm to the extracellular space.[7] Some of the secreted proteins include elastases (LasA and LasB), phospholipase C, and exotoxin A (ToxA), all of which play crucial roles in infection.[8] Together, these proteins promote tissue damage, disrupt immune components, and inhibit protein synthesis.[7,9-11]

Furthermore, the type III secretion system (T3SS) allows for the direct injection of effector proteins into the cytosol of host cells.[1,12] The effector proteins produced by the T3SS of *P. aeruginosa* include ExoS, ExoT, ExoU, and ExoY, which exhibit varying levels of cytotoxicity.[7-8] These effector proteins act on cell signaling pathways, inducing tissue destruction, cytoskeletal disruption, and cell death.[1,7-8,13-14] Together, the type II and III secretion systems contribute to tissue damage, impaired immune function, and bacterial persistence, creating a favorable environment for surgical infections.

P. aeruginosa can establish chronic infections through biofilm formation, which consists of bacterial aggregates encased in extracellular polymeric substances (EPS).[7] Biofilm formation begins with attachment of bacterial cells to a surface, followed by irreversible adhesion mediated by their flagella and fimbriae to overcome resistive cell-surface interactions.[15] EPS plays multiple vital roles in biofilm survival, including aiding surface adhesion, nutrient acquisition, and protection against antimicrobials.[15-16] As the biofilm continues to mature, the sheet of initial cells becomes a three-dimensional structure, in which aggregates detach and travel through the environment to form new biofilms elsewhere.[7] Biofilm formation is particularly relevant in orthopedic-implant-related infections, where bacteria can persist despite treatment.

The formation of *P. aeruginosa* biofilms is regulated by quorum sensing (QS) systems, which are bacterial communication mechanisms that contribute to pathogenicity. QS involves small signaling molecules known as autoinducers, which bind to an inducer protein, leading to target gene expression.[17] *P. aeruginosa* QS consists of Pseudomonas quinolone signal (PQS), las, and rhl.[18] The basis of this communication system hinges on protein interactions that produce virulence factors, promote biofilm development, and enhance viability, all factors that increase the pathogenicity of *P. aeruginosa*.[7,19] By coordinating virulence factor expression and biofilm formation, quorum sensing allows bacteria to respond to environmental stress, promoting biofilm stability and persistence on orthopedic implants.

P. aeruginosa's ability to resist antimicrobial agents is driven by both intrinsic and acquired mechanisms. This bacterium possesses intrinsic resistance to several antibiotics, while acquired mechanisms have contributed to the gradual increase in multidrug-resistant strains.[7] Efflux pumps play a central role in *P. aeruginosa*'s innate ability to diminish antibiotic success.[20] Additionally, intrinsic resistance is also determined by the ampC gene, which encodes β-lactamases. *P. aeruginosa* can produce extended-spectrum-β-lactamases allowing for significant resistance against β-lactam antibiotics.[7,21] On the other hand, prior exposure to antibiotics contributes to *P. aeruginosa*'s acquired resistance, allowing the bacteria to acquire multiple resistance genes. This can be done through horizontal gene transfer or gene mutations, allowing *P. aeruginosa* to become highly resistant.[22] Collectively, the pathogenicity of *P. aeruginosa* relies on secretion systems, biofilm formation, quorum sensing, and antimicrobial resistance. Together, these mechanisms allow *P. aeruginosa* to persist within the host and establish infections in environments such as surgical wounds and orthopedic devices.

GUT COLONIZATION AS A RESERVOIR

The gut microbiome plays an important role in maintaining intestinal homeostasis and providing protection against opportunistic pathogens, such as *P. aeruginosa*.[4] However, in hospitalized and immunocompromised patients, the gut flora is often disrupted by antibiotic use, invasive medical procedures, or underlying inflammatory or infectious processes.[23] This imbalance, or dysbiosis, creates favorable conditions for opportunistic pathogens such as *P. aeruginosa* to colonize the gut. *P. aeruginosa* is highly adaptable and can survive in a wide range of environments, including hospital food, sinks, mops, and respiratory equipment.[2] Additionally, the use of invasive medical devices, such as central venous catheters and mechanical ventilation, further increases the risk of colonization. Direct or indirect contact with these diverse sources represents a common route of colonization.

P. aeruginosa can colonize the gut and penetrate the intestinal epithelium through mechanisms that disrupt epithelial integrity, impair host defenses, and promote bacterial translocation.[3] Following intestinal colonization, *P. aeruginosa* can spread systemically to other areas of the body, most notably the lungs, either by aspiration of gastrointestinal flora or through hematogenous dissemination to the lung parenchyma.[3] Identical strains are frequently detected in both the gut and lungs, indicating that the gut may serve as a clinically relevant reservoir from which *P. aeruginosa* infections arise.[24] Once the bacteria enter the bloodstream, they can disseminate further and infect other regions of the body. For instance, *P. aeruginosa* may disseminate hematogenously from the gut to distant sites, potentially contributing to surgical site and implant-related infections.

Patients admitted to the ICU specifically have a greater susceptibility to *P. aeruginosa*-related infections. The use of broad-spectrum antibiotics in ICUs targets both harmful and beneficial bacteria, significantly reducing microbial diversity and further exacerbating dysbiosis.[25] This facilitates the emergence of multidrug-resistant strains of *P. aeruginosa*, potentially leading to gastrointestinal, urinary tract, and bloodstream infections. This can be particularly concerning given the increasing prevalence of *P. aeruginosa* strains that are resistant to many antibiotic classes, including β-lactams, carbapenems, aminoglycosides, and fluoroquinolones.[26]

SURGICAL COMPLICATIONS AND SURGICAL SITE INFECTION RISK

P. aeruginosa is a notable contributor to surgical complications and surgical site infections. It acts as an opportunistic pathogen, particularly in patients undergoing colorectal, hepatobiliary, and abdominal surgery, who are susceptible to mucosal disruption, antibiotic exposure, medical devices, and immunosuppression.[27] A multicenter surveillance study by Weiner et al found that *P. aeruginosa* accounted for up to 8% of SSIs in U.S. acute care hospitals.[28] *P. aeruginosa* infections illustrate complex clinical consequences such as increased morbidity and difficult antibiotic selection due to resistance.[29] Therefore, invasive surgical procedures create unique opportunities for endogenous pathogens to contribute to infection.

Colonization of the gastrointestinal (GI) tract has been shown to serve as a potential endogenous source of infection.[3] It is important to note that intestinal colonization often precedes infection, especially in patients with altered microbiota secondary to antibiotics, chemotherapy, or GI surgery.[25,30] More specifically, ICU and postoperative patients have been linked to altered microbiomes, which may result from contamination or barrier disruption during surgery.[23] Studies provide evidence of strain matching between the intestines and SSIs using whole-genome sequencing and pulsed-field gel electrophoresis.[27,31] This suggests that translocation may occur across a compromised intestinal barrier or contamination during surgery. Therefore, these findings

indicate that SSI prevention may require attention to endogenous reservoirs in addition to traditional infection control.

Although evidence suggests a link between the gut microbiome and SSIs, there is still no routine screening for gut colonization.[31] However, studies have identified potential future risk-stratification tools that may be useful for evaluating infection risk, such as rectal swabbing or fecal metagenomics.[32] This suggests a potential role for targeted prophylaxis in high-risk populations, as multidrug-resistant strains are increasingly difficult to treat.[29] Overall, early detection could inform targeted perioperative protocols to reduce surgical complications and SSIs.

ORTHOPEDIC IMPLANT RELATED INFECTIONS

Orthopedic implant-related infections caused by *P. aeruginosa* pose a challenge due to the organism's ability to form biofilms. Although it is less frequent than infections caused by Gram-positive bacteria, such as *Staphylococcus aureus*, *P. aeruginosa* can contribute to prosthetic joint infections (PJIs).[33] Risk factors for infection include open fractures, prior surgical interventions, nosocomial exposure, and prior antibiotic therapy.[34] A prospective study by Benito et al noted that among patients with PJIs due to Gram-negative organisms, *P. aeruginosa* infections had the highest rates of treatment failure and re-revision at one year.[35] The persistence of *P. aeruginosa* centers on its ability to adhere to foreign material and evade clearance. These biofilms pose significant treatment challenges due to resistance to antimicrobials, phagocytosis, and immune clearance.[36] Although biofilm formation is a major factor in bacterial persistence on implant surfaces, the intestines remain a potential site of colonization that can cause extraintestinal infections; however, its contribution to orthopedic implant infections remains unclear.

P. aeruginosa infections of orthopedic implants present management challenges and the need for emerging treatment options. Biofilms create a significant difficulty due to the persistence of bacteria. Therefore, biofilm development highlights the need for surgical debridement, systemic antibiotics, and, occasionally, full implant replacement.[36] In addition, other treatment strategies are often combined for greater efficacy. Adjunctive strategies, including bacteriophage therapy, antibiofilm peptides, and implant surface modifications, are under investigation but remain experimental.[37] *P. aeruginosa* infections present a considerable treatment challenge and require further investigation into novel approaches.

TREATMENT, PREVENTION, AND EMERGING THERAPEUTICS

Treatment of *P. aeruginosa* infections is complicated by antimicrobial susceptibility and increasing resistance patterns. Commonly utilized antibiotics include β-lactams, such as piperacillin-tazobactam, cefepime, and ceftazidime, which display antipseudomonal action.[38] Additionally, aminoglycosides, fluoroquinolones, and carbapenems may be utilized based on infection severity and susceptibility.[38] This list is not exhaustive of all drugs used to treat *P. aeruginosa*, but highlights the major drug classes used.

Given the challenges associated with antimicrobial resistance and biofilm formation, prevention remains a critical component of management. A multicenter prospective cohort study by Gomila et al evaluated a typical prophylactic treatment, which was a combination of an aminoglycoside with either erythromycin or metronidazole, and how the treatment affected surgical outcomes.[31] This study examined 669 patients who developed surgical site infections among 3701 colorectal surgery patients and found that 9.3% of surgical site infections were due to *P. aeruginosa*, with the gut as the reservoir.[31] The study found a 60% decrease in *P. aeruginosa* post-operative surgical site infections in the group given prophylactic treatment.[31] These findings suggest that targeted prophylaxis may reduce the risk of post-operative *P. aeruginosa* infections.

The management of surgical *P. aeruginosa* infections extends beyond colorectal surgery and into orthopedic surgeries, namely, total joint arthroplasties. The foreign hardware of a total joint arthroplasty provides an environment conducive to the formation of *P. aeruginosa* biofilms.[39] Moreover, biofilm-associated infections are harder to treat because altered gene expression and slowed metabolism make antibiotics less effective or lead to antibiotic resistance.[40] Total joint arthroplasty infections are difficult to treat and often require removal, illustrating the need for prophylaxis.

A major concern of *P. aeruginosa* infections is the development of antibiotic resistance. A study from the European Centre for Disease Prevention and Control in 2020 found that 30.1% and 17.3% of *P. aeruginosa* infections were resistant to one or multiple antibiotics previously listed, respectively.[41,42] The data include carbapenem resistance, which is of particular concern as carbapenem antibiotics are considered a last-resort treatment for serious infections.[41-43] Given the potential for antibiotic resistance, it is recommended to limit the use of broad-spectrum agents that may promote further resistance. Due to these concerns, further research into novel therapeutics is being conducted with a focus on biofilm formation.

Biofilm formation has become a common research target for novel therapies. For example, bacteriophages (BP) have been shown to have efficacy against quorum sensing in *P. aeruginosa*.[43] A study by Cesta et al showed efficacy of BP therapy in a woman with a chronic *P. aeruginosa* infection affecting her total right hip arthroplasty.[39] Another novel therapy that has shown promise is the use of antimicrobial peptides.[43] Antimicrobial peptides have

distinct mechanisms against various targets, and those developed against *P. aeruginosa* primarily act on biofilms and cell membranes.[43] While the mechanisms of BP and antimicrobial peptides are not fully understood, they show promise for treating biofilm-forming and multidrug-resistant *P. aeruginosa*.[41,43] With the severity of *P. aeruginosa* infections and the decreasing efficacy of traditional antibiotics, research into novel therapeutics must continue.

RESEARCH GAPS AND FUTURE DIRECTIONS

Future research is needed to further elucidate the role of *P. aeruginosa* in surgical site infections and orthopedic implant-related infections. This is critical given the increasing concern and challenges presented, as it displays growing multidrug resistance. Most notably, there is a lack of large-scale, prospective studies that can establish links between intestinal colonization and surgical site infections. Further, existing infection surveillance systems rarely examine the gut microbiota and their ability to translocate.[44] This limits surveillance ability to identify *P. aeruginosa*, which may lead to postoperative complications. Therefore, studies that evaluate patients in the pre- and postoperative stages are needed. While improvements can be integrated within infection surveillance systems, there is also potential for development in molecular studies. Several studies have implemented genotyping, which is able to identify and compare strain differences, although they are limited.[45-46] Given the limitations of genotyping, more advanced techniques are needed to establish connections to gut colonization. Whole-genome sequencing and metagenomics are potential opportunities for precise strain tracking, allowing researchers to determine where the strain originated.[27,47] Ultimately, advancements in the infection surveillance system and molecular studies could provide evidence of strain linkage, highlighting a critical area of clinical significance.

Translating these findings into clinical practice requires the implementation of targeted screening protocols to identify at-risk patients. There is an increasing need for implementation within orthopedic surgery, as implant infections can have devastating consequences that worsen outcomes. Screening protocols are beneficial as they can improve decision-making and gut decolonization strategies for patients who may be at increased risk. For example, interventions such as selective digestive decontamination (SDD), probiotics, or fecal microbiota transplantation (FMT) warrant further study to restore a healthy microbiota.[48] Progress in this area is dependent on a multidisciplinary approach that includes surgeons, infectious disease specialists, microbiologists, epidemiologists, and pharmacists. Establishing a cross-disciplinary, unified approach to design protocols and guidelines is essential to advance research and for integration into routine care.

CONCLUSION

This review highlights a multidisciplinary pathway of *P. aeruginosa* infection, linking gut colonization with surgical and orthopedic complications. *P. aeruginosa*, although not the most common cause of PJIs, serves as a source of post-surgical complications and prosthetic failure. The ability of *P. aeruginosa* to disseminate from hidden reservoirs, such as the gut, highlights the need to investigate the value of preoperative screening and subsequent treatment to prevent postoperative complications. Antibiotic resistance poses challenges when treating postoperative *P. aeruginosa* and often requires extensive treatment, highlighting the importance of preoperative detection. Successful reduction in postoperative complications would require protocol considerations across multiple specialties, as the potential for invasion is not isolated to a singular source. Future guidelines may benefit from focusing on interdepartmental infrastructure and protocols that screen, collect, and streamline the patient's individual microbiological status for use hospital-wide. The inclusion of the gut microbiota in future microbiological surveillance systems would prove to be beneficial, especially to those at higher risk. Further research should aim to focus on strengthening the evidence linking the gut to surgical site infections, specifically following patients pre- and post-operatively, allowing their connection to guide future protocols. Understanding the gut as a potential reservoir for *P. aeruginosa* may provide an opportunity to develop targeted prevention strategies for high-risk surgical and orthopedic patients.

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