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Bacterial Biofilm Virulence on Hospital Plastic Surfaces: Mechanistic Insights, Persistence Drivers, and Infection Control Challenges

Abbas J. Abed
Department of Biology, Microbiology, Thi-Qar Education Directorate, Thi-Qar, Iraq.

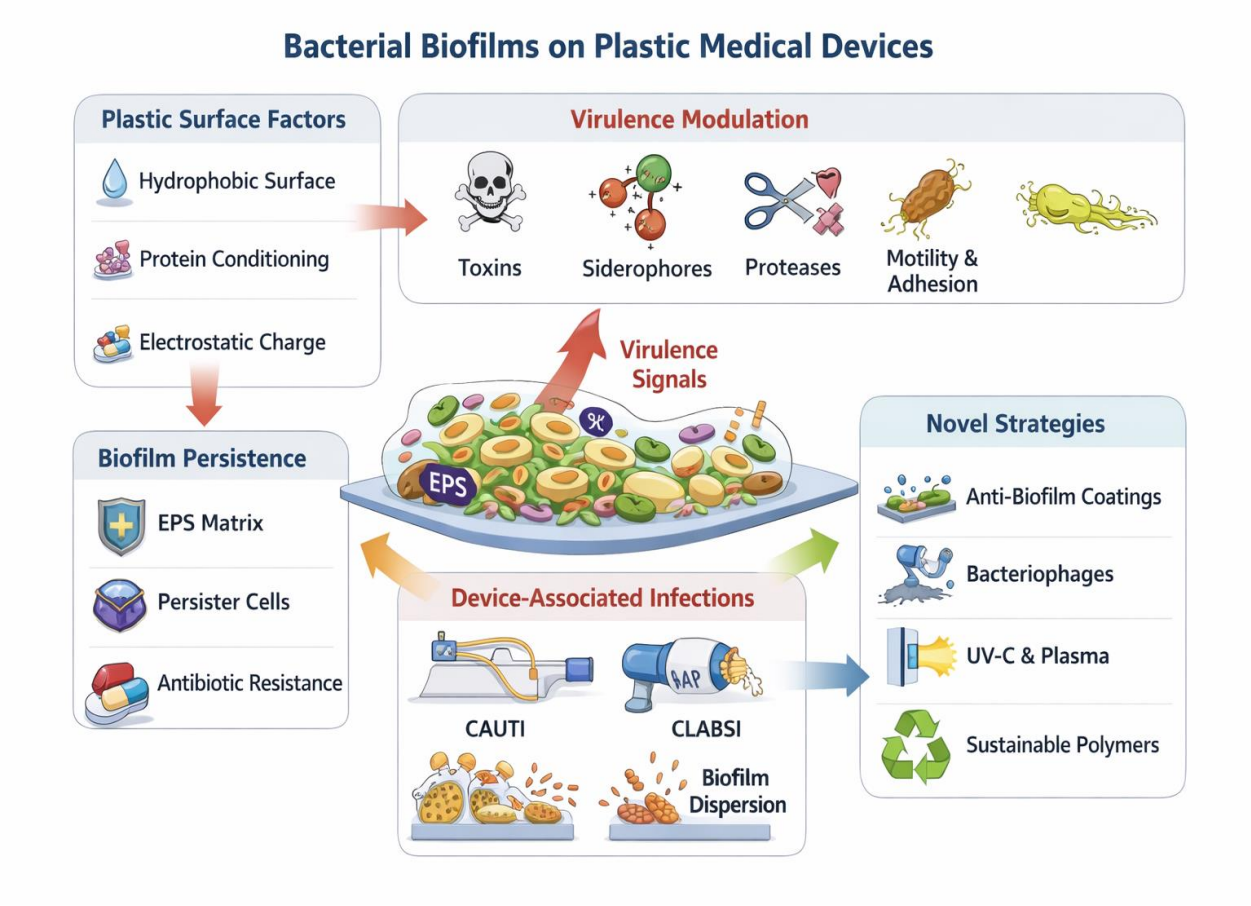
Muhammad Abdul-Razzaq Ali Al-Haidary
Department of Biology/Zoology, Thi-Qar Education Directorate, Thi-Qar, Iraq.

Abstract

The establishment of bacterial biofilms on plastic surfaces has become increasingly recognized as an important, yet understudied, contributor to the persistence of pathogens in clinical and community settings. The wide use of polymers, like polyethylene and polypropylene, in the manufacturing of medical devices and high-contact surfaces inside hospitals creates physicochemical interfaces that promote bacterial adhesion and biofilm development. In contrast to metals or glass, however, plastics contribute to hydrophobic and electrostatic deposition, rapid accumulation of host-derived proteins, and metabolic zonation in biofilm depth with an increased tolerance against antimicrobials while supporting long-term survival in the environment. Surface sensing of polymers is also a heterotypic signal that activates bacterial virulence programs and induces expression of toxins, proteases, hemolysins, siderophores, and motility determinants, which are in turn correlated with phenotypes for chronic infection. Biofilms aged on plastics serve as pathogen dissemination reservoirs by active dispersion, which leads to secondary infections and adds difficulty in infection control. Conventional disinfection modalities are mainly ineffective owing to the inadequate penetration of antimicrobials into EPS matrices and fast resurface recolonization. New developments illustrate the possibility of biofilm-aware interventions such as anti-adhesive materials designs, enzyme/peptide-coated surfaces, quorum-sensing inhibitors, phage-based therapies, UV-C exposure, and plasma treatments, as well as the manufacture of inherently

antimicrobial/biocompatible/sustainable polymer alternatives. This review integrates existing mechanisms, clinical risk, and emergent infection preventive technologies, proposing that the next-generation biosafety guideline should ultimately center on the polymer–microbe–altitude of virulence axis to constrain biofilm resilience and inhibit pathogenic switching event upon plastic medical device surfaces.

Keywords: Bacterial biofilms, Plastic surface colonization, Polyethylene & polypropylene, Extracellular polymeric substances (EPS), Quorum sensing regulation, Virulence gene activation, Disinfectant tolerance.



Introduction

Nosocomial (hospital-acquired) infections or hospital-associated infections continue to be a significant global public health issue that contributes substantially to morbidity, mortality, and cost of medical care [1,2]. Recent data suggested that 5–10% of hospitalized patients in the world suffer from HAIs; however, rates increase substantially to as high as 20% in the ICU and among patients with invasive medical devices [3,4]. Bacterial pathogens, including Staphylococcus aureus, Pseudomonas aeruginosa, Klebsiella pneumoniae, and Acinetobacter baumannii, play a significant role by frequently developing biofilm communities which promote both persistence and acquisition of virulence genes as well as antibiotic resistance [2,5,6]. Biofilm on hospital surfaces and devices is a concern for infection control, linked with extended hospital stay, repeated antibiotic treatment, and adverse clinical outcome [7,8]. With their versatility, low cost, and simpler sterilization process, plastic materials have become omnipresent in health care settings. Catheters, endotracheal tubes, intravenous lines, surgical instruments, hospital furniture, and disposable tools are medical plastics that have large areas for host bacterial colonization [9, 10]. Introduction The tremendous utilization of plastics in patient care has dramatically changed, however, also unconsciously increased the available surfaces where bacteria can adhere as well as biofilm formation resulting in device-related infections [11]. Plastics have unique physicochemical properties that are different from metals and glass, affecting the attachment of microorganisms. Such properties include hydrophobicity, surface

roughness, and flexibility, as well as microcrevices or the use of polymer additives that typically encourage bacterial adhesion and EPS (extracellular polymeric substances) formation [12,13]. It has been widely shown that biofilms develop faster and last longer on plastic than on stainless steel or glass in comparable conditions [14,15]. Moreover, they may protect bacteria from disinfectants and ecological stresses that can further contribute to their roles as infection reservoirs [16]. This review addresses hospital plastic surfaces as sites for bacterial biofilm growth, providing mechanistic models of attachment and biofilm formation with their associated enhancement in virulence and persistence. We additionally discuss the clinical relevance of plastic biofilms in device-related infections, and summarize novel approaches for the prevention and control of such infections. This review pays a scopic attention to sensitize the clinician and research workers about preventing nosocomial infections due to plastics.

Bacterial Colonizers of Hospital Plastic Surfaces

Plastic materials in hospital environments are reservoirs for a variety of clinically significant nosocomial bacterial pathogens. These may adhere to polymeric surfaces and establish biofilms that may contribute to both survival and antimicrobial resistance. P. aeruginosa is an opportunistic Gram-negative pathogen known to robustly form biofilms on plastic devices, including catheters and ventilator tubing. Its biofilms show high resistant against antibiotics/disinfectants and cause lasting infections in intensive care units/immunocompromised groups [6,17]. S. aureus, including MRSA, is commonly found in the hospital environment on surfaces and medical devices. These surfaces comprised of plastic material (e.g., intravenous lines, bed rails, and wound dressings) are conducive to adhesion and biofilm formation of S. aureus leading to difficulty in the treatment of infection and a production for chronic infections [18,19]. K. pneumoniae, a Gram-negative enteric organism is able to form biofilms on polymeric materials resulting in device-associated infections including catheter associated urinary tract infections (CAUTIs). Its encapsulated nature and exopolysaccharide production promote hospital-plastic adhesion and biofilm persistence [20,21]. Acinetobacter baumannii: A nosocomial pathogen with a role in the wild? It easily attaches to plastic materials, such as respirators and catheters, and produces biofilms that increase resistance to drying out and multiple antimicrobials [22,23]. Enterococcus faecalis is a Gram-positive species frequently associated with catheter-related infections. It can colonise plastic medical devices and create biofilms which are inherently resistant to numerous antibiotics and disinfectants, rendering treatment difficult [24,25]. It is known that clinical strains of *E. coli* are involved in various nosocomial infections and often build a biofilm on medical plastics, particularly in catheter and drain systems. [26,27]. These biofilms help the bacteria to survive in a hostile environment and evade immune detection. These pathogens have been reported to persistently survive on plastic surfaces, and biofilm formation increases long-term survival, despite routine cleaning and disinfection procedures Figure 1. This persistence represents a significant contribution to the risk of ongoing transmission in healthcare settings [17,22,28].

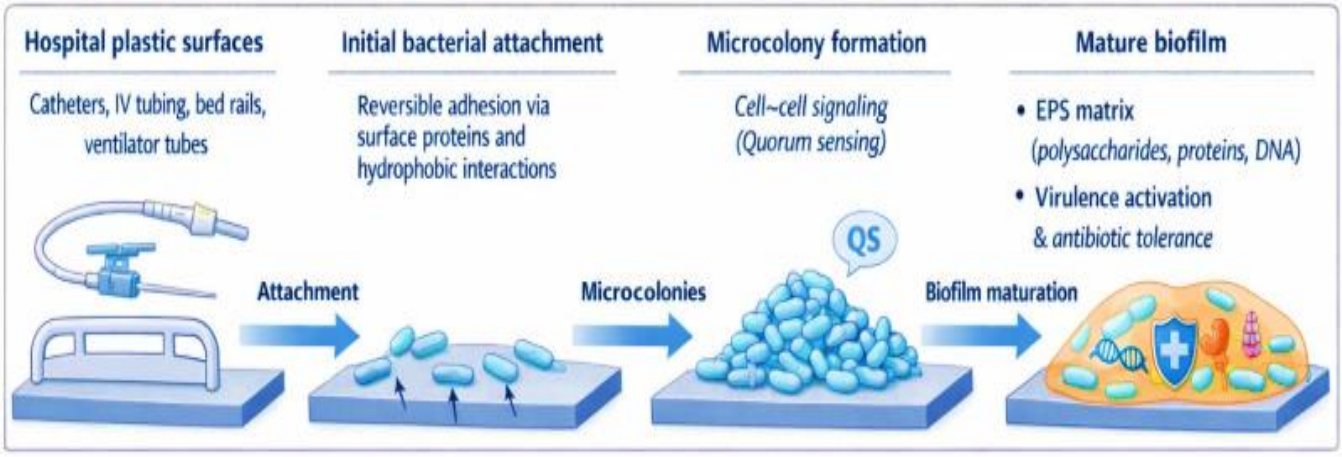


Figure 1. Bacterial colonization and biofilm formation on hospital plastics.

4. Polymer Properties That Promote Bacterial Adhesion

Plastic used in the healthcare industry is an essential part of any medical institution, from hospitals to clinics, from plastic IV to laboratory equipment. Polymers, such as polyethylene (PE), polypropylene (PP), polyurethane, and silicone, are widely adopted in the clinical field because of their lightness, chemical

stability, and low cost [29,30]. But these materials interact with microbes in a fundamentally different way than metal and glass surfaces do. Plastics as a class are generally more hydrophobic, less thermally conductive, and harbor microscopic surface imperfections that promote bacterial adherence, while metals and glass are generally smoother, stiffer, and with greater surface energy to diminish bacterial incorporation over time [31–33]. This physicochemical distinction enables nosocomial pathogens to more effectively colonize plastics and trigger biofilm formation faster when compared with that on stainless steel or glass [31,34]. Bacterial adhesion is even more enhanced by the addition of polymer (plasticizers, stabilizers, antioxidants) and process aids to the polymers and the quick formation of conditioning films in relation to plastics exposed to clinical fluids. These films modify surface charge and energy, resulting in an a biological interface that facilitates microbial attachment [34,36]. High-risk devices, the tubing and IV bags used for intravenous feeding, bladder catheters, central venous catheters, and endotracheal ventilator circuitry surfaces provide large polymeric surface areas in constant contact with nutrients – a nourishing environment not only for formation of biofilms by these opportunistic bacteria but also that are caused by *P. aeruginosa*, *S. aureus*, *K. pneumoniae*, *A.baumannii* (all mentioned references are within current list), *E. faecalis* as well as pathogenic clinical isolates of *E.coli* [34–40]. In these, PE and PP surfaces are highly susceptible to bacterial adhesion thanks to hydrophobic forces and structural flexibility that assist bacteria in withstanding mechanical removal [41]. Upon exposure to human biological fluids, the plastics are quickly coated with host proteins, of which fibrinogen and albumin were identified as being directly involved in microbial colonization. Fibrinogen is a primary ligand for bacterial surface adhesins (eg, MSCRAMMs, especially in G+) and serves as a molecular bridge between bacterial cells and polymer surfaces [42,43]. Albumin is also involved in the modulation of surface heterogeneity and energy, indirectly promoting bacterial adhesion and persistence according to environmental conditions [36,44]. Taken together, the combination of hydrophobicity, electrostatic effects, protein coatings, and polymer microstructure can account for why plastics are a source of biofilm and act as reservoirs for bacterial colonization in the clinical as well as environmental hospital environments [31,34,42,45].

5. Biofilm Formation Stages on Plastic Surfaces

The formation of biofilm on plastic surfaces is a dynamic multi-stage process. In early attachment, bacteria mainly adhere to the polymers by hydrophobic interactions and electrostatic adhesions and thus can easily attach onto surfaces like PE, PP, polyurethane, or silicone (31. 46). Following adhesion, these microorganisms initiate the process of micro-colony formation in which the first EPS deposition is detectable, stabilizing an attached subpopulation and giving rise to clusters that are impervious to low-shear stress (6). The maturing phase involves the transition to a 3D structured biofilm, densification of the EPS matrix, and activation of quorum-sensing (QS) regulatory pathways. QS circuits -las and rhl systems that contribute to *P.aeruginosa* and agr in S. aureus- control cell-to-cell communication, biofilm thicknesses, the metabolic heterogeneity among cells, as well as expression of its virulence-associated genes (47,48,49). The agr QS system in Gram-positive bacteria regulates toxin production, protease secretion, and detachment behavior, while the rhl (RhlI/RhlR) and las (LasI/LasR) systems of *P. aeruginosa* control the levels of elastase, rhamnolipid, pyocyanin, and other biofilm-related virulence factors (47-50). The ending step, biofilm dispersal, leads to the liberation of planktonic pathogenic cells that are capable of initiating secondary colonization or systemic infections and promotes polymer-mediated spread of pathogens in clinical zones (51). An important factor that dictates the persistence of biofilms is the EPS matrix, largely comprised of polysaccharides, eDNA, proteins, and lipids, making up a viscoelastic scaffold to entrap nutrients, impede diffusion of disinfectants, and protect bacteria from oxidative stress, desiccation, and immune assault (6,52-53). EPS serves as a physical as well as biochemical barrier of defense, which allows for long-term survival on the polymers, despite repetitive sanitation exposures (53,54). Concomitantly, QS also governs the EPS biosynthetic genes and surface-active compounds like rhamnolipids (in Gram-negative) and phenol-soluble modulins (PSMs in Gram-positive), which are involved in the structuring of biofilm, as well as detachment from plastics (Gram-positive) (50,55). Collectively, the combination of hydrophobic polymer affinity with early EPS secretion and QS-driven virulence activation rationalizes biofilms' aggressive persistence and pathogenic ability on hospital plastics, informing the ineffectiveness of traditional surface disinfection strategies that do not address QS or EPS disruption (31, 47, 53, 54).

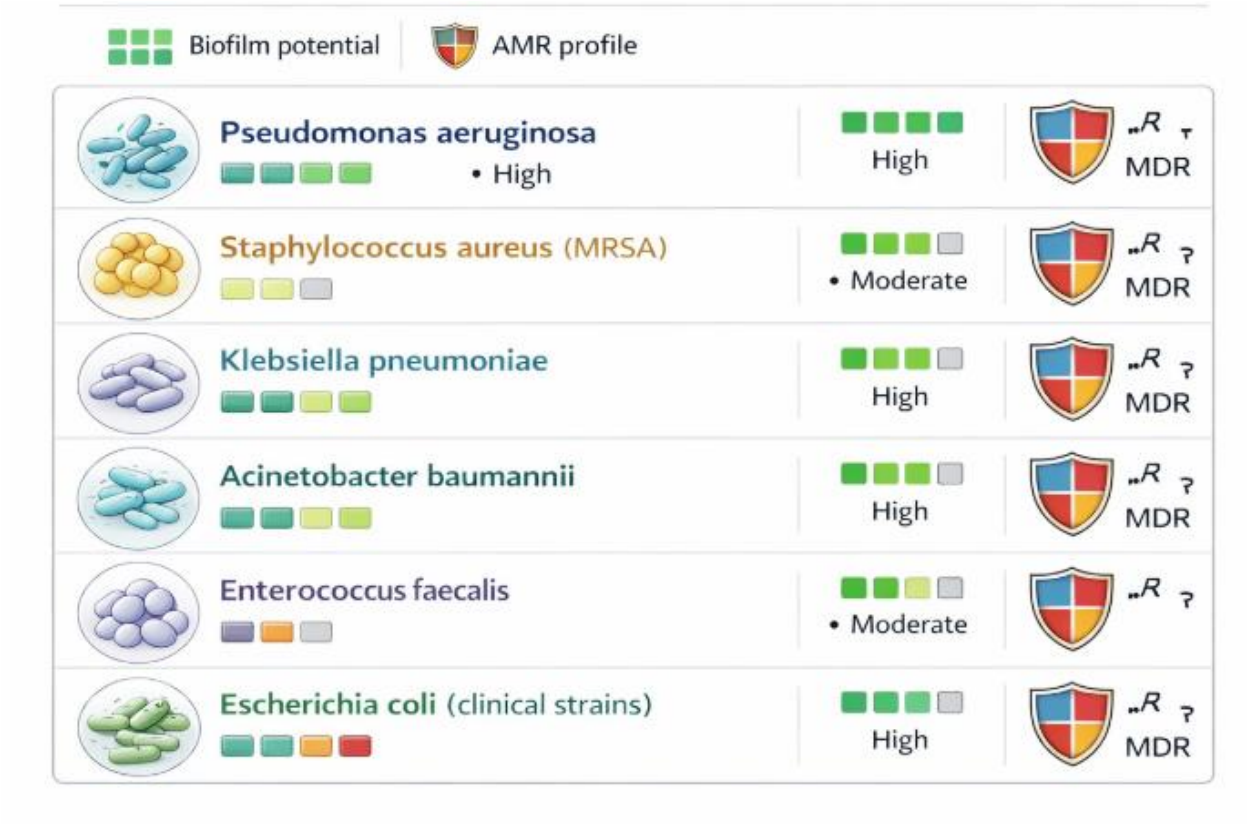


Fig. 2 Common nosocomial bacterial pathogens associated with plastics.

6. Virulence Modulation Triggered by Plastics

Plastics are not passive substrates upon which bacteria adhere and grow, but rather, plastics actively shape bacterial physiology to increase their potential for being pathogenic. Upon adhesion to polymeric surfaces, bacteria encounter environmental signals (e.g., differences in surface energy, hydrophobic interfaces, and lack of nutrients) that act as stress stimuli, leading to the activation of regulatory entities that modulate virulence gene expression [56,6]. These responses comprise increased production of toxins, extracellular proteases, siderophores, and hemolysins, which contribute to tissue injury, iron acquisition, immune evasion, and survival in the host's environment [57, 58, 59]. For instance, in Pseudomonas aeruginosa, contact with the surface induces the production of elastase and pyocyanin, two important virulence factors involved in lung tissue invasion and oxidative stress generation [57], [59]. Staphylococcus aureus exhibits increased expression of hemolysins and adhesins during biofilm formation on hydrophobic plastics, thereby enhancing the invasive potential of these organisms and their resistance to host defense [60,61]. Apart from toxin production, cell attachment to plastics may enhance motility and expression of the adhesive factors (pili, flagella, and surface adhesins), allowing for a deeper colonization within polymeric microstructures and maturation of biofilms [62, 63]. Increased motility also serves to further expand biofilm territories and for its spread on device surfaces. In addition, contact with plastic surfaces has been associated with a shift from acute to chronic infection phenotypes such that biofilm-associated cells are characterized by reduced metabolic activity, increased stress tolerance, and upregulation of antibiotic and immune defense mechanisms [64,65]. Together, these modulatory effects highlight a mechanism that polymer contact uses to template bacterial virulence landscapes, converting environmental surfaces into catalysts for pathogenicity and persistence in health care settings.

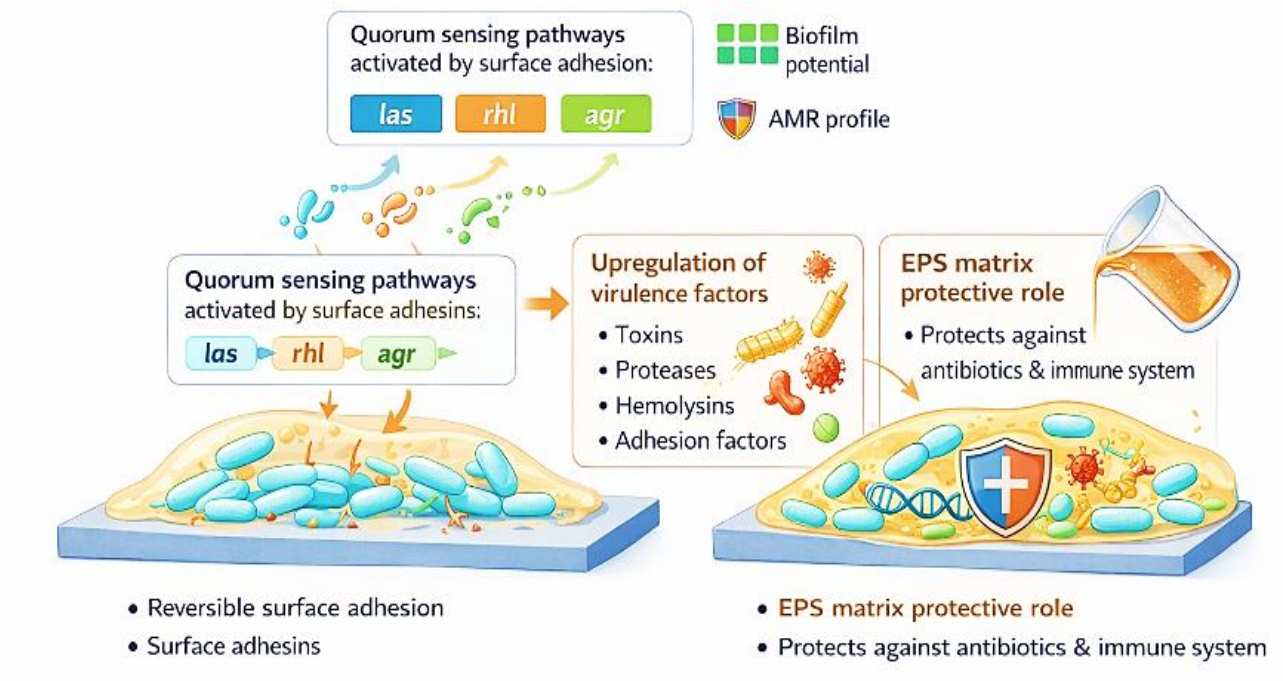


Fig. 3 Mechanisms enhancing bacterial virulence on plastic surfaces

7. Persistence and Resistance Mechanisms in Plastic-Associated Biofilms

Biofilms that smear on plastics are highly persistent and resistant, which creates frequent challenges for infection control and increases the risk of chronic or recurrent hospital infections. One particular difference in biofilm resistance terms is the distinction between tolerance and genetic resistance. In contrast to HGT or mutations causing transmissible AMR, which are genetic resistances, this resistance- and/or tolerance-form in biofilms is mainly phenotypic, nonheritable forms of resistance. The tolerance is the consequence of a limited penetration of antibiotics into dense extracellular polymeric substance (EPS) matrix, but also the result of slow-growth states or modified metabolic activity that allows bacteria to survive in the presence of lethal concentrations of drugs without harboring classical resistance mechanisms [66,56]. In biofilms, a subpopulation of phenotypically dormant cells termed persister cells is able to survive an otherwise lethal challenge of antimicrobials. These cells are metabolically quiescent and antibiotic-penetrant resistant, persisting under drug pressure before repopulating the biofilm when treatment ceases [67,68]. Furthermore, the estrangement of antibiotics from the bacterial cell ensures that biofilm-associated bacteria exploit promiscuous efflux pumps, membrane proteins that extrude toxic molecules from cells, which are upregulated in most known human pathogens [69-70], leading not only to a decrease in intracellular antibiotic concentration but also broad-spectrum tolerance. Other environmental pressures exerted by plastics, such as nutrient deprivation and oxidative stress, further contribute to persistence. Nutrient gradients within the biofilm also contribute to metabolic heterogeneity, with cells in the depth of the biofilm entering a state of slow or non-growth, which is known to render persister/slow-growing stationary cells slightly less susceptible to antibiotics, and their outer layers can generate protective stress responses [71, 72]. For biofilm bacteria, enzymes and stress regulators (e.g., an antioxidant defense) are also expressed in order to moderate the effects of oxidatively damaging agents; therefore, this assists in their survival against immune attack or disinfection [73]. Of particular concern is co-resistance to both disinfectants and antibiotics. Running at low levels of hospital disinfectants, which is typical for regularly cleaned plastics, can select for tolerance features similar to antibiotic resistance mechanisms, including induction of efflux systems, membrane-strengthening, and response networks to stress. This co-selection promotes the selection of MDR strains within biofilms on plastic surfaces, and acts as a reservoir for persistent pathogens that are not eliminated by normal hygiene in daily practice [74,75]. Clarification of these persistence and resistance networks is crucial for the advancement of specific approaches to dismantle EPS matrices, disrupt efflux systems, and deplete persister populations to enhance infection resolution in clinical settings where polymeric surfaces play a critical role.

Fig. 4 Plastic surface properties that promote biofilm formation.

8. Infection Risks Linked to Plastic Medical Devices

Medical devices made of plastic used in hospitals are the basic places where bacterial biofilm is constructed, and they are also associated with several serious device-related infections. Notably, both CAUTI continues to be one of the most prevalent healthcare-associated infections globally (aged 19-55). Catheterized urine drainages offer a steady polymer cover, allowing bacteria (e.g., *E. coli*, *E. faecalis*, and *P. aeruginosa*) to adhere easily and form biofilm in urine indwelling tubes, leading to chronic infection as well as rising UTIs with enhanced antibiotics' application [76,77,78]. Ventilator-associated pneumonia (VAP) is the other serious condition attributable biofilm biofilm-coated plastics. Mechanical ventilation, notably endotracheal tubes, is quickly colonized by oral and environmental flora in highly elaborate biofilm populations that can disseminate distally to the lower respiratory tract. These biofilms act as long-lasting reservoirs for pathogens, like *S. aureus*, *P. aeruginosa*, and MDR Acinetobacter baumannii bacteria, to dramatically escalate morbidity and mortality in the ICU [79,80]. USE: Central-line-associated bloodstream infections (CLABSIs) occur when pathogens travel through a catheter's biofilm to the central circulation. Biofilms shield bacteria from both host defenses and antibiotics, thus causing pathogens to be intermittently seeded into circulation, which may result in septicemia, endocarditis, and organ failure [81,82]. Biofilm dispersion is a major aspect of biofilm biology that may be pertinent to these device-related infections. In dispersion, cells separate from the mature biofilm structures and are released into the circulation or neighboring tissues as planktonic, highly virulent organisms that can cause secondary infections at distant sites and make treatment outcomes difficult to predict [83,84]. For instance, dispersion from catheter biofilms is thought to be responsible for recurrent urinary infections, and release from endotracheal tube biofilms has been related to repeated pneumonia episodes in ventilated patients [83,85]. Epidemiological trends have also been illuminated by the financial burden imposed by polymicrobial device infections on healthcare systems. Rates of CAUTI have been consistently among the highest reported HAI types in many monitoring networks, and frequently range between 30-40% of all reports in acute care hospitals. VAP prevalence has been reported to be between 5 and 40 episodes per 1,000 ventilator days, with a crude mortality as high as 20–50% in some populations. CLABSIs, while less frequent due to the implementation of bundle prevention strategies, continue to occur at a sustained rate, especially in high-risk units, and are related to prolonged length of stay (LOS) and subsequent extended treatment cost [86,87,88].

In the light of such concepts, medical plastics cannot be considered merely as passive materials in a clinical setting, but they can rather be considered involved directly in nosocomial infections epidemiology; for this reason, tailored prevention strategies aimed at disturbing biofilm development and reducing the risk of device-related infections are necessary.

9. Current and Emerging Infection Control Strategies

Despite well-documented cleaning regimens, standard infection control technologies are frequently inadequate to eliminate biofilms on plastic surfaces in hospital premises. The poor penetration of disinfectants into the EPS matrix that harbors active agents, and reduces their effective concentration at the surface of the bacterial cells is a major issue. This barrier function even enables resident bacteria in deeper biofilm layers to survive disinfection challenges that would eliminate planktonic cells [64,65]. Furthermore, even when the impact of surface treatments is successful in reducing initial microbial inocula, there appears to be re-colonization, possibly due to remaining adherent cells or environmental contamination and exposure to new pathogens leading to recurrent cycling of biofilm establishment upon commonly touched plastics (invasive medical devices) such as catheters, ventilator circuits, (intravenous) IV tubing [89, 90]. To address these limitations, newer strategies have been created to prevent biofilm formation and persistence in a more targeted manner. Promising approaches in this sense are the prevention of initial bacterial attachment and the removal of mature biofilms by anti-biofilm surface coatings. Substrates functionalized with silver nanoparticles, chitosan, antimicrobial peptides, and enzymes have shown great decreases in adhesion to polymeric surfaces or time of formation and the biomass of biofilms on polymer materials, through impairing cell integrity, quorum-sensing, and/or simply obstructing structure stability (EPS) [91,6]. For instance, chitosan coatings also possess antimicrobial activity per se and can disturb EPS generation, whilst engineered peptides can inhibit crucial bacterial adhesion receptors [91,92]. An additional novel approach is that of bacteriophage therapy, as well as the use of quorum-sensing inhibitors (QSIs). They combine biofilm matrix penetration with the ability to selectively and specifically lyse target pathogens, being highly specific without causing any harm to host tissues or commensal microbiota [93, 94]. These compounds, such as furanone derivatives or synthetic analogs, interfere with bacterial cell-to-cell communication systems that control virulence and biofilm maturation, which might act to attenuate pathogenic behaviors and make biofilm communities more susceptible to immune clearance and antimicrobial agents [95,96]. Surface modification to minimize hydrophobic adherence also presents considerable prospects. Modifying the hydrophilicity of polymeric surfaces, introducing micro- and nano-patterned topographies, or incorporating antimicrobial agents can reduce bacterial adherence on subsequent biofilm phases [97,98]. This can be especially powerful when attached to coatings that are time-released with antimicrobial agents. Advanced physical sterilization techniques UV-C irradiation, and plasma, are also booming. UV-C light could affect the microbial DNA and disrupt the biofilm architecture non-chemically, whereas cold plasma induces reactive agents that inactivate the surface microbes and alter the surface chemistry of polymers to decrease bacterial attachment [99,100]. At the same time, new AMBs that are intrinsically resistant to colonization and may release biocides in a controlled manner will be developed so as to provide permanent protection without depending on external disinfectants [101]. Taken together, these innovative approaches target the biofilm prevention and disruption on plastic medical surfaces, representing a multi-pronged strategy of infection control urgently needed in healthcare settings where HAIs remain as ongoing threats.

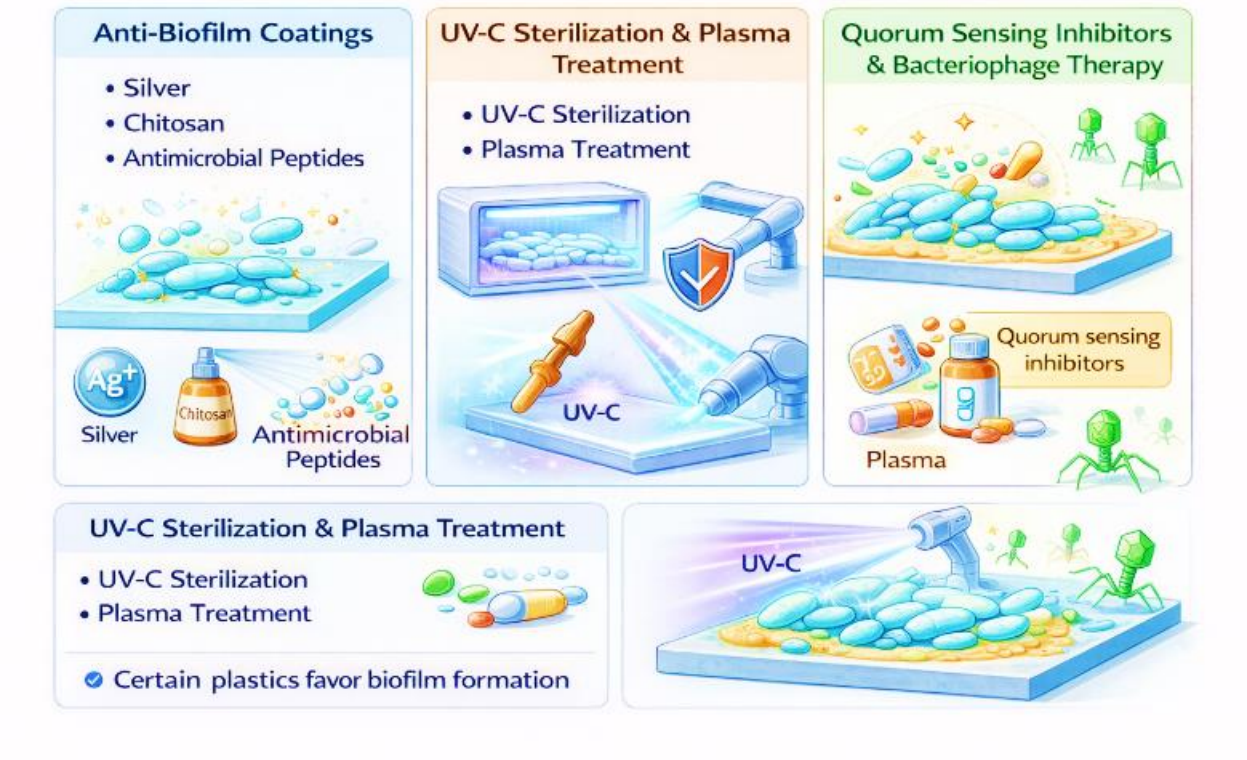


Fig. 5 Emerging strategies to control biofilm-associated infections on plastic medical surfaces

10. Future Directions and Research Gaps

The emerging realization that plastics are proper ecological niches for pathogenic biofilms leads to several immediate research priorities. Yet prevailing hospital hygiene concepts are largely material-neutral, with polymers and their surfaces featuring distinct physico-chemical/biological responses pointing to the imperative of polymer-specific cleaning/disinfection protocols based on hydrophobicity/surface aging/additive leaching profiles rather than classic metal-derived sterilization/sterilizability considerations. It is argued throughout that surface-dependent biofilm tolerance can differ significantly between polymer classes, so that hygiene measures must be directed by substrate. [56,64]. One rising gap is the lack of active surveillance for microbial aerosol within the plastic surfaces. Progress in biosensing indicates that fast abiotic pathogen detection can be achieved by electrochemical and optical sensing platforms, yet these methods are rarely translated into clinical use. A real-time biosensor that could detect polymer contamination would help to intervene early before mature biofilm and pathogen dissemination meta-stable state development, both in materials where they are used as support and food-based contact material. [102,103]. There remains a critical lack of understanding of polymer-mediated bacterial virulence gene regulation. More recently, it has emerged that abiotic surface contact can serve as a regulatory signal for biofilm behavior and virulence gene activation; however, the mechanisms on medical polymers remain relatively uncharted. Understanding the signals by which polymer surfaces act on transcriptional networks for toxins, efflux systems, and chronic-infection phenotypes is necessary to develop counter-strategies that inhibit virulence at genetic and signaling levels [104,63]. Lastly, sustainable thinking into next-generation infection control strategies is required, with research aimed at greener antimicrobial plastic substitutes. Natural and synthetic antimicrobial polymers (including bio-based and biodegradable composites) are being developed as safer alternatives to conventional polypropylene and polyethylene. Sustained Antimicrobial plasticsAriskPlasticsOver the long term, Infection riskShareReductionDInfection environmental pathogen persistence. Sustainable antimicrobial plastics have an additional advantage over regular medical devices that they can reduce infection risk and simultaneously limit environmental pathogen persistence [105,6].

Conclusion

Hospital plastics are not inert surfaces, but alive with pathogens forming biofilm. The combination of hydrophobic polymer chemistry, micro-roughness of the surface, diversity in

electrostatic charge, and fast adsorption of human conditioning (adhesive) proteins provides permissive interfaces for bacterial attachment and biofilm development. Compared to metal and glass, polymers prefer an unsymmetrical bugged micro-niches arrangement, allowing bacteria to shift towards slow-growth and highly tolerant phenotypes in which the EPS houses antiseptic penetration (slide resistance), carpet survival chemically. The biofilm architecture also promotes metabolic heterogeneity and deep-layer survival, and specialized subpopulations (e.g., persister cells) maintain the viability of the biofilm and contribute to infection relapse when treatment pressure is cleared. In addition to being a physical block to phagocytosis, adhesive contact with polymers can also act as a physiological signal that activates virulence regulatory networks controlling the overproduction of toxins, proteases, siderophore-mediated iron acquisition systems, adhesins, and motility factors (such as fimbriae and R-type pili) that together redirect host-pathogen interactions toward chronic infection and recalcitrant phenotypes. These relationships lead not just to device-related infections, but also to infection cascades induced by biofilm dispersion; this leads to free-swimming planktonic cells, which then spread through the host. New infection control approaches, even if not yet practical solutions, underscore that effective prevention will require a material-informed biofilm-aware multilayered strategy comprised of anti-biofilm bed designs, novel intrinsically antimicrobial and biocompatible polymer designs for healthcare products (i.e., HAI-proof pipes21), bacteriophage- and quorum-sensing-based biocontrol technologies combined with advanced physical sterilization technologies (UV-C; cold plasma). Simultaneous advances in green polymer science: driving a critical need for sustainable antimicrobial alternatives. Plastics that mitigate colonization while minimizing environmental reservoirs of multidrug-resistant pathogens. Overall, the future clinical biosafety rests on decoding and, where possible, overcoming the polymer-microbe-virulence axis, permitting design of next-generation medical plastics and infection-control strategies that inhibit adhesion, disrupt biofilm firmness, and diminish virulence induction to improve patient outcomes and enhance global infection control.

CRediT Authorship Contribution Statement

Abbas J. Abed: Conceptualization; methodology; investigation; data curation; formal analysis; data analysis & interpretation; visualization; writing – original draft preparation; **Muhammad Abdul-Razzaq** writing – review & editing; validation; project administration.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data Availability

No new data were generated or analyzed in this study. Data sharing does not apply to this article.

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