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Developing Biofilms with Pathogenic Bacteria

Rana Talib Mohsen
University of Anbar, Al-Anbar, Iraq
rana2011@uoanbar.edu.iq

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Abstract

Microbial communities that develop on living and nonliving surfaces, such as dental tissues or artificial implants, often form complex, structured assemblies known as biofilms. These biofilms enhance microbial survival by providing protection against environmental stressors, including antimicrobial agents. The formation of biofilms contributes significantly to the antibiotic resistance observed in many bacterial populations. *Bacillus cereus*, a known foodborne pathogen, is capable of forming biofilms and producing toxins that cause gastrointestinal illnesses, including vomiting and diarrhea. Preventing the initial development of biofilms may be more effective than attempting to eliminate mature biofilms, which are notoriously difficult to eradicate. A range of strategies, such as chemical disinfectants, antibiotic therapies, and the application of nanoparticles has been explored to inhibit or disrupt biofilm formation. The significance of microbial biofilms spans various sectors, notably the food and pharmaceutical industries, where contamination and persistent infections pose major concerns. Growing recognition of the link between biofilms and chronic disease has intensified research interest, as bacteria residing in biofilms are often shielded from immune responses and conventional treatments. Current insights into biofilm-associated pathogenesis highlight multiple mechanisms through which biofilms contribute to disease development and persistence.

Keywords: Biofilm; Medical interaction; Biotic surfaces; Pathogenicity; Antimicrobial resistance

Introduction

Originally developed as a defensive mechanism for prokaryotes against excessively severe circumstances, biofilm has attracted a lot of attention because of its greater impact on the food, pharmaceutical, medical, and public health sectors[1,2]. Biofilms were characterized as structured collections of microbial cells that adhered to one another and disintegrated in an extracellular matrix of varying density and composition [3]. Biofilm is regarded as an adaptation mechanism that allows microbial cells to endure harsh environments including UV rays, abrupt pH shifts, and draining [3,4]. The issues brought on by biofilm development in food and medicine are summed up in this overview. Additionally, the stages involved in biofilm production and prevention are emphasized.

The role of biofilm in the food sector

Biofilms are the main source of bacterial buildup on food surfaces, including utensils and equipment, during food preparation, making it a persistent source of food contamination [5,6]. In food production systems, the majority of pathogenic or spoiling bacteria are found on surfaces as adherent or planktonic cells or in community configurations as biofilms [7]. the capacity of Listeria monocytogenes and Staphylococcus aureus to stick to the surfaces of food goods as well as materials that come into touch with food, such as glass, rubber, stainless steel, and polypropylene [8]. Due to their capacity to survive at high temperatures and create biofilms, developing on abiotic or biological surfaces [9]. Microbial biofilms that form on the inside of thermal equipment, including pasteurizers and sterilizers, can cause material degradation and lower heat exchanger performance, which can result in significant losses for the food sector [10,11]. The biofilm generation of harmful and spoilage bacteria in the food business is greatly influenced by variables including temperature, food ingredients' pH, and the availability of their leftovers. These elements may also make associated cells more resistant to disinfection processes, making it more difficult to remove biofilm using standard cleaning techniques [12,13,14].

The medical interaction of biofilm

Antibiotic-resistant biofilms and diseases linked to indwelling medical devices pose the biggest threats to public health [15,16,17, 18]. Bacteria that live in biofilms are less vulnerable to antibiotics; because of changes in cell metabolism and structural characteristics that affect drug permeability [19].

The process by which bacteria become resistant to antibiotics

1- Bacteria can alter their structure to alter the antibiotic's target structure. For example, fluoroquinolone antibiotics board DNA gyrase in Gram-negative organisms, then altering the target location raises resistance and decreases drug binding [20].

2- Bacteria harvest enzymes which canister deactivate antibiotic and become ineffective such as Extended spectrum β-lactamases (ESBLs) include CTXM enzymes originate in Gram-negative type plummeting the effectiveness of cephalosporins, aztreonam then penicillin [21].

3- By increasing the normal level of bioactivity before plummeting the porousness of the active membrane by suppressing the manufacture of purines, bacteria can prevent the bioactivity from reaching the target site [22].

Processes by which biofilms arise and the variables that influence them

Micro colonies are the fundamental structural components of a biofilm; they are rod-shaped and allow water to pass through them [23]. 10–25% of microcolonies are made up of cells, while 79–90% are made up of extracellular polysaccharides (EPS) [24].Complex biofilm production can be stimulated by a variety of stimuli, including certain regulators like c-di-GMP and second messengers. Everyone agrees that the second messenger, c-di-GMP, is a switch molecule that mediates the bacterial change from a planktonic lifestyle to the production of biofilms [25]. It is crucial for the post-translational stimulation of the manufacture of exopolysaccharides [26]. C-di-GMP and biofilm development are strongly correlated; while low c-di-GMP levels promote planktonic growth, high c-di-GMP levels promote biofilm formation [27, 28]. There are several phases in the general biological model of the biofilm formation cycle.

The first stage, known as the adhesion stage, occurs when cells adhere to a surface using hydrophobic effects and weak van der Waals forces [29, 30]. A collection of these cells forms micro colonies during the second stage, known as sessile growth. Since the cells can separate and revert to a planktonic condition, the two processes are reversible [31]. In general, physical characteristics like hydrophobicity, roughness, electrostatic charges, and surface materials like silicon, rubber, stainless steel, and polypropylene may all affect how easily microbial cells adhere to surfaces [32,33]. The end stage of biofilm development is irreversible because cells are encased in a dense, stable, complex bimolecular layer [34]. A three-dimensional tower structure matched the fully developed biofilm. Cells disperse through both active and mechanical mechanisms after a biofilm is fully formed. The fourth step, or dispersion stage, is where these processes take place. Cell-cell-adhesive matrix components, phenol-soluble modulins, proteases, regulators, and nucleases are among the disruptive agents secreted by the cells in the biofilm [35].

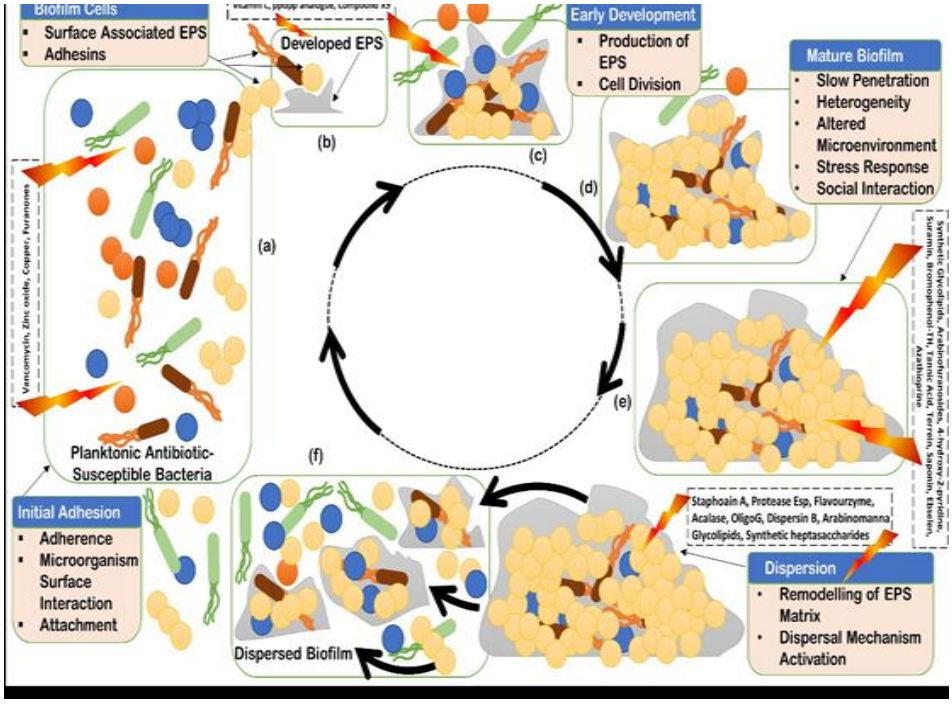

Figure 1. The biofilm life cycle comprises the following stages: initial adhesion (a), early polymerization (b), early EPS production development (c), EPS development (d), mature biofilm (e), and biofilm dispersion (f) [36].

Some microorganisms that create biofilms

According to the Centers for Disease Control, biofilms are the most prevalent clinical barrier of the century [37]. Numerous bacterial species are capable of forming biofilms.

Staphylococci may cling to abiotic materials as well as human tissues like skin [38]. Furthermore, S. epidermidis, which forms biofilms on abiotic surfaces, is responsible for a high proportion of device-associated illnesses. [39,40,41].

S. epidermidis, which culminates in an LPXTG sorties covalently bound to the cell wall's peptidoglycan [42]. E. coli accountable aimed at extensive spectrum of microbial illnesses in the human body such as UTIs, catheter- related infections, or dental plaques [43]. E. coli may form functional amyloids known as curli. Because they interact with several host proteins during infections, these extracellular fibrils may function as virulence factors and aid in the production of biofilms. Curli fibrils play a crucial role in the extracellular matrix needed for the expansion of biofilms, as well as in bacterial adhesion to surfaces and cell aggregation [44]. Since Curli fibrils interrelate by diversity of crowd proteins and are identified by Toll-like receptors, which trigger the innate immune system, they are also regarded as significant virulence factors and are referred to as pathogen-associated molecular patterns (PAMPs) [45]. Similar to Salmonella entericase ovar Typhimurium, CsgD is a transcriptional controller that governs the formation of biofilms [46]. Genes complicated in the synthesis of curli fibers and the polysaccharide cellulose are found in the CsgD regulon [47, 48]. The curli subunit operon is directly induced by CsgD, whereas the diguanylate cyclase gene adrA is activated by CsgD [49]. The second messenger cyclic-di-GMP, which is shaped via AdrA, stimulates BcsA and cellulose synthase, causing biofilm development [50,51]. A common bacteria that forms endospores in dairy products and diary settings is B. cereus [52]. They can stick to food processing equipment and create biofilms [53]. Two types of toxins are produced by B. cereus: diarrheal, which causes diarrheal syndrome when bacteria grow in the small intestine, and emetic, which causes emetic syndrome when bacteria grow in food [54]. Numerous virulence factors, including collagenase, phospholipase, hemolysin, and metalloprotease, are secreted by B. cereus and contribute to its pathogenicity. Zn-dependent enzymes called metalloprotease, in particular immunological inhibitor A (Inh A), let bacteria pass through the intestinal barrier and avoid being detected by macrophages [55]. The simplest way to combat B. cereus biofilms in industry is to stop them from forming, either by lowering the spore weight in rare

resources or by identifying and eliminating anew formed biofilms as soon as they appear [56]. One organism by itself or a combination of two organisms can produce a biofilm. The majority of microorganisms were naturally found in polymicrobial communities [57]. When fungal or bacterial species work together, they can form cooperative multi-species biofilms. For example, Candida albicans and Streptococcus gordonii interact in the oral cavity when their proteins, Als3 and SspB, directly bind to each other [58], and Candida albicans and S. aureus work together to cause denture stomatitis infections. Prosthetic joint infections can also result from the formation of mixed biofilms by S. aureus and S. epidermidis in vitro [59].

Bacterial biofilm prevention

Inhibition of bacterial biofilms occurs by several methods as follow:

1. A combination of antibiotics Antibiotics with a much higher minimum inhibitory concentration (MIC) in cells within biofilms are administered topically as treatments [60]. Antibiotic side effects are a drawback of combination antibiotic therapy, while medications such sodium salicylate and N-acetylcysteine can be utilized as immune-modulatory and anti-inflammatory drugs, as well as to break up extracellular matrix and eliminate biofilms [61,62].

2- Because of their distinct processes, which vary from those of conventional antibiotic treatment, nanoparticles (NPs)—polymer-, lipid-, and protein-based NPs—have been employed to break through biofilms [63]. Silver nanoparticles interact with intracellular proteins, phosphate residues in DNA, and bacterial membrane proteins to impair cell division and ultimately cause bacterial death, despite the fact that their toxicity is a major problem that limits their usage to certain areas [64].3. Biofilm production can be inhibited by Bdellovibrio and related organisms (BALOs). A number of proteases, hydrolases, and nucleases are secreted by gram-positive bacterial biofilms, causing an intracellular transcriptome response in B. bacteriovorus. This is linked to the degradative activity of BALOs, which ultimately damages the biofilms [65].

4- Because the first and most important stage in the production of biofilms is the adhesion of microbes to a surface, medical equipment' surfaces, such as metal ones, can be modified to remove biofilms. Biofilm development is prevented by applying antimicrobial chemicals

(antiseptics, antibiotics, or metals) to the surface and employing materials that are resistant to microbe adherence [66].

5- The main purpose of medicinal medicines containing copper is to treat malignancies. [64,67]. Coordination compounds containing copper are excellent anticancer, antifungal, antibacterial, antimalarial, and anti-inflammatory medicines because they are less expensive. Numerous proteins and enzymes, such as oxidase, cytochrome C, and Cu/Zn superoxide dismutase, which are involved in DNA synthesis, energy metabolism, and respiration, require copper for their production and proper operation [68]. These are potent medications that can take the place of traditional platinum chemotherapy [69]. Additionally, because sorbates promote the generation of toxins and cause cellular stress, they can decrease the development of bacterial biofilms. Increasing the cell's acidity increases the sorbate's action [70].6- deactivation of enzymes and denaturation of proteins. Dispersin B and deoxyribonuclease are two enzymes that break down the extracellular matrix of the biofilm and may help the biofilm spread [71,72]. Anti-biofilm drugs include fatty acid messenger and cis-2-decenoic acid can promote dispersion and stop the growth of biofilm colonies, therefore chemicals that break down the biofilm matrix can be helpful. Because Pseudomonas aeruginosa secretes cis-2-decenoic acid, a fatty acid messenger, many bacterial species, including the yeast Candida albicans, form cyclo-heteromorphic cells [73].

Detrimental impacts of biofilms in health

Biofilms have also been linked to a extensive range of nosocomial . Biofilm-ecstatic reasons of contagion can comprise catheter surfaces, wound dressings, medical grafts, then numerous additional kinds of medical equipment. Biofilms play an important character in the founding of Mycobacterium tuberculosis contagion then defend exist in bacilli after immune reply then antimycobacterial managers. An rise in illness prevalence due to pathogen presence in the residential environment immediately reflects poor and inefficient disinfection and cleaning measures[74].

Conclusion

Biofilms represent a persistent and complex challenge across food safety and healthcare domains. Their structural resilience and adaptive mechanisms not only allow microbial

survival in harsh environments but also complicate disinfection efforts and contribute to antibiotic resistance. This review has detailed the mechanisms of biofilm formation, the microbial species involved, and the diverse prevention strategies available—from conventional antibiotics to advanced nanotechnologies and biofilm-dispersing enzymes. Moving forward, interdisciplinary research focusing on early detection and targeted inhibition strategies will be critical for managing biofilm-associated risks effectively in both clinical and industrial settings.

References

1. Henrici, A. T. (1936). Studies of freshwater bacteria: III. Quantitative aspects of the direct microscopic method. *Journal of Bacteriology, 32*(3), 265–280. https://doi.org/10.1128/jb.32.3.265-280.1936
2. Baier, R. E. (1984). Initial events in microbial film formation. In J. D. Costlow & R. C. Tipper (Eds.), *Marine biodeterioration: An interdisciplinary approach* (pp. 57–62). E & FN Spon.
3. Moris, V., Lam, M., Amoureux, L., Magallon, A., Guilloteau, A., Maldiney, T., & Neuwirth, C. (2022). What is the best technic to dislodge *Staphylococcus epidermidis* biofilm on medical implants? *BMC Microbiology, 22*(1), 1–14. https://doi.org/10.1186/s12866-022-02443-3
4. Kouzuma, A., Kato, S., & Watanabe, K. (2015). Microbial interspecies interactions: Recent findings in syntrophic consortia. *Frontiers in Microbiology, 6*, 477. https://doi.org/10.3389/fmicb.2015.00477
5. Abdallah, M., Chataigne, G., Ferreira-Theret, P., Benoliel, C., Drider, D., Dhulster, P., & Chihib, N. E. (2014). Effect of growth temperature, surface type and incubation time on the resistance of *Staphylococcus aureus* biofilms to disinfectants. *Applied Microbiology and Biotechnology, 98*, 2597–2607. https://doi.org/10.1007/s00253-013-5419-9
6. Boubendir, S., Arsenault, J., Quessy, S., Thibodeau, A., Fravalo, P., Thériault, W. P., & Gaucher, M. L. (2021). Salmonella contamination of broiler chicken carcasses at critical steps of the slaughter process and in the environment of two slaughter plants: Prevalence, genetic profiles, and association with the final carcass status. *Journal of Food Protection, 84*, 321–332. https://doi.org/10.4315/JFP-20-308
7. Ago, A., Rocha, V., Barros, O., Silva, B., & Tavares, T. (2024). Bacterial biofilm attachment to sustainable carriers as a clean-up strategy for wastewater treatment: A review. *Journal of Water Process Engineering, 63*, 105368. https://doi.org/10.1016/j.jwpe.2024.105368
8. Bridier, A., Sanchez-Vizuete, P., Guilbaud, M., Piard, J. C., Naïtali, M., & Briandet, R. (2015). Biofilm-associated persistence of foodborne pathogens. *Food Microbiology, 45*, 167–178. https://doi.org/10.1016/j.fm.2014.04.015

9. Hall-Stoodley, L., Costerton, J. W., & Stoodley, P. (2004). Bacterial biofilms: From the natural environment to infectious diseases. *Nature Reviews Microbiology, 2*(2), 95–108. https://doi.org/10.1038/nrmicro821
10. Pogiatzis, T., Vassilis, S., Mergulhão, F., & Wilson, D. (2015). Choosing when to clean and how to clean biofilms in heat exchangers. *Heat Transfer Engineering, 36*, 676–684. https://doi.org/10.1080/01457632.2014.957143
11. Pagedar, A., Singh, J., & Batish, V. K. (2010). Surface hydrophobicity, nutritional contents affect *Staphylococcus aureus* biofilms and temperature influences its survival in preformed biofilms. *Journal of Basic Microbiology, 50*(1), 98–106.
12. Köhler, A. T., Rodloff, A. C., Labahn, M., Reinhardt, M., Truyen, U., & Speck, S. (2019). Evaluation of disinfectant efficacy against multidrug-resistant bacteria: A comprehensive analysis of different methods. *American Journal of Infection Control, 47*(10), 1181–1187.
13. Del Pozo, J. L., Rouse, M. S., & Patel, R. (2008). Bioelectric effect and bacterial biofilms: A systematic review. *International Journal of Artificial Organs, 31*(9), 786–795.
14. Lazar, V. (2011). Quorum sensing in biofilms—How to destroy the bacterial citadels or their cohesion/power? *Anaerobe, 17*(6), 280–285.
15. Nobile, C. J., Solis, N., Myers, C. L., Fay, A. J., Deneault, J.-S., Nantel, A., … Mitchell, A. P. (2012). A recently evolved transcriptional network controls biofilm development in *Candida albicans*. *Cell, 148*(1–2), 126–138. https://doi.org/10.1016/j.cell.2011.10.048
16. Curran, N. (2020). New study first to visually capture biofilm architecture in retrieved implants from live patients. *Spinal News International.*
17. Zhao, Y. M., Patange, A., Sun, D. W., & Tiwari, B. K. (2020). Plasma-activated water: Physicochemical properties and applications in food industry. *Comprehensive Reviews in Food Science and Food Safety, 19*, 3951–3979.
18. Eshed, M., Lellouche, J., Matalon, S., Gedanken, A., & Banin, E. (2012). Sonochemical coatings of ZnO and CuO nanoparticles inhibit *Streptococcus mutans* biofilm formation. *Langmuir, 28*(33), 12288–12295.
19. Jolivet-Gougeon, A., & Bonnaure-Mallet, M. (2014). Biofilms as a mechanism of bacterial resistance. *Drug Discovery Today: Technologies, 11*, 49–56.
20. Redgrave, L. S., Sutton, S. B., Webber, M. A., & Piddock, L. J. (2014). Fluoroquinolone resistance: Mechanisms and role in evolutionary success. *Trends in Microbiology, 22*(8), 438–445.
21. Zhao, F., Wang, W., Lu, W., Xu, L., Yang, S., Cai, X. M., Zhao, M., Lie, M., Ma, M., Xu, H., & Cao, F. (2018). High anticancer potency on tumor cells of dehydroabietyl amine Schiff-base derivatives and a copper (II) complex. *European Journal of Medicinal Chemistry, 146*, 451–459.
22. Høiby, N., Bjarnsholt, T., Givskov, M., Molin, S., & Ciofu, O. (2010). Antibiotic resistance of bacterial biofilms. *International Journal of Antimicrobial Agents, 35*, 322–332. https://doi.org/10.1016/j.ijantimicag.2009.12.011
23. Stoodley, P. (2016). Biofilms: Flow disrupts communication. *Nature Microbiology, 1*(1), 1–150.
24. Allesen-Holm, M., Barken, K. B., Yang, L., Klausen, M., Webb, J. S., Kjelleberg, S., Molin, S., Givskov, M., & Tolker-Nielsen, T. (2006). A characterization of DNA release in *Pseudomonas aeruginosa* cultures and biofilms. *Molecular Microbiology, 59*(4), 1114–1128.

25. Dahlstrom, K. M., Collins, A. J., Doing, G., Taroni, J. N., Gauvin, T. J., Greene, C. S., Hogan, D. A., & O'Toole, G. A. (2018). A multimodal strategy used by a large c-di-GMP network. *Journal of Bacteriology, 200*(8), e00703-17.
26. Poulin, M. B., & Kuperman, L. L. (2021). Regulation of biofilm exopolysaccharide production by cyclic di-guanosine monophosphate. *Frontiers in Microbiology, 12*, 730980.
27. Valentini, M., & Filloux, A. (2016). Biofilms and cyclic di-GMP signaling: Lessons from *Pseudomonas aeruginosa* and other bacteria. *Journal of Biological Chemistry, 291*(24), 12547–12555.
28. Juan, J., et al. (2022). Anti-biofilm activities of Chinese poplar propolis essential oil against *Streptococcus mutans*. *Nutrients, 14*, 3290.
29. Briandet, R., Herry, J., & Bellon-Fontaine, M. (2001). Determination of van der Waals, electron donor, and electron acceptor surface tension components of static Gram-positive microbial biofilms. *Colloids and Surfaces B: Biointerfaces, 21*(4), 299–310.
30. Di Bonaventura, G., Piccolomini, R., Paludi, D., D'Orio, V., Vergara, A., Conter, M., & Ianieri, A. (2008). Influence of temperature on biofilm formation by *Listeria monocytogenes*. *Journal of Applied Microbiology, 104*, 1552–1561.
31. Kumar, A., Alam, A., Rani, M., Ehtesham, N. Z., & Hasnain, S. E. (2017). Biofilms: Survival and defense strategy for pathogens. *International Journal of Medical Microbiology, 307*(8), 481–489.
32. Teh, K., Flint, S., Palmer, J., Andrewes, P., Bremer, P., & Lindsay, D. (2014). Biofilm: An unrecognised source of spoilage enzymes in dairy products? *International Dairy Journal, 34*, 32–40.
33. Feng, Q., Ahator, S. D., Zhou, T., Liu, Z., Lin, Q., Liu, Y., Huang, J., Zhou, L., & Zhang, L. H. (2020). Regulation of exopolysaccharide production by ProE, a cyclic-di-GMP phosphodiesterase in *Pseudomonas aeruginosa*. *Frontiers in Microbiology, 11*, 1226.
34. Roy, R., Tiwari, M., Donelli, G., & Tiwari, V. (2018). Strategies for combating bacterial biofilms: A focus on anti-biofilm agents. *Virulence, 9*(1), 522–554.
35. Sued-Karam, B. R., Olivella, J. G. B., Cabral-Oliveira, G. G., & Pereira-Ribeiro, P. M. A. (2024). Resistance and virulence factors of *Staphylococcus aureus*: A brief review. *Seven Editora*. https://doi.org/10.56238/sevened2024.013-005
36. Dutt, Y., Dhiman, R., Singh, T., Vibhuti, A., Gupta, A., Pandey, R. P., Raj, S. V., Chang, C., & Priyadarshini, A. (2022). The association between biofilm formation and antimicrobial resistance. *Antibiotics, 11*(7), 930.
37. Blackledge, M. S., Worthington, R. J., & Melander, C. (2013). Biologically inspired strategies for combating bacterial biofilms. *Current Opinion in Pharmacology, 13*(5), 699–706.
38. Vlassova, N., Han, A., Zenilman, J. M., James, G., & Lazarus, G. S. (2011). Biofilms in dermatological disease. *British Journal of Dermatology, 165*(4), 751–758.
39. Otto, M. (2009). *Staphylococcus epidermidis*—The 'accidental' pathogen. *Nature Reviews Microbiology, 7*(8), 555–567.
40. Geoghegan, J. A., Monk, I. R. O., O'Gara, J. P., & Foster, T. J. (2013). Subdomains N2N3 of fibronectin binding protein A mediate *Staphylococcus aureus* biofilm formation. *Journal of Bacteriology, 195*(11), 2675–2683.
41. O'Grady, N. P., Alexander, M., Burns, L. A., Dellinger, E. P., Garland, J., Heard, S. O., … Mermel, L. A. (2011). Guidelines for the prevention of intravascular catheter-related infections. *American Journal of Infection Control, 39*(Suppl. 1), S1–S34. https://doi.org/10.1016/j.ajic.2011.01.003

42. Hussain, M., Herrmann, M., von Eiff, C., Perdreau-Remington, F., & Peters, G. (1997). A 140-kDa extracellular protein is essential for *Staphylococcus epidermidis* surface accumulation. *Infection and Immunity, 65*(2), 519–524.
43. Tambyah, P. A. (2004). Catheter-associated urinary tract infections: Diagnosis and prophylaxis. *International Journal of Antimicrobial Agents, 24*, 44–48.
44. Saldana, Z., Xicohtencatl-Cortes, J., Avelino, F., Phillips, A. D., Kaper, J. B., Puente, J. L., & Girón, J. A. (2009). Synergistic role of curli and cellulose in cell adherence and biofilm formation of *Escherichia coli*. *Environmental Microbiology, 11*(4), 992–1006.
45. Olsen, A., Herwald, H., Wikström, M., Persson, K., Mattsson, E., & Bjorck, L. (2002). Functional regions of curli, a virulence determinant of *Escherichia coli*. *Journal of Biological Chemistry, 277*(37), 34568–34572.
46. Hadi, N., Yang, Q., Barnett, T. C., Tabei, S. M. B., & Kirov, S. M. (2012). Bundle-forming pilus locus of *Aeromonas veronii* bv. *Sobria*. *Infection and Immunity, 80*, 1351–1360.
47. Hammar, M., Arnqvist, A., Bian, Z., Olsen, A., & Normark, S. (1995). Expression of csg operons in *E. coli* for Congo red-binding curli production. *Molecular Microbiology, 18*, 661–670.
48. Chapman, M. R., Robinson, L. S., Pinkner, J. S., Roth, R., Heuser, J., Hammar, M., Normark, S., & Hultgren, S. J. (2002). *E. coli* curli operons direct amyloid fiber formation. *Science, 295*, 851–855.
49. Römling, U., Rohde, M., Olsen, A., Normark, S., & Reinköster, J. (2000). AgfD regulates multicellular behavior in *Salmonella typhimurium*. *Molecular Microbiology, 36*(1), 10–23.
50. Zogaj, X., Nimtz, M., Rohde, M., Bokranz, W., & Römling, U. (2001). *Salmonella* and *E. coli* produce cellulose in extracellular matrix. *Molecular Microbiology, 39*(6), 1452–1463.
51. Römling, U., Galperin, M. Y., & Gomelsky, M. (2005). c-di-GMP: The dawning of a novel bacterial signaling system. *Molecular Microbiology, 57*, 629–639.
52. Salustiano, V. C., Andrade, N. J., Ribeiro Junior, J. I., Fernandes, P. E., Lopes, J. P., Bernardes, P. C., & Portugal, J. G. (2010). Controlling *Bacillus cereus* adherence to stainless steel with cleaning procedures in dairy plants. *Arquivo Brasileiro de Medicina Veterinária e Zootecnia, 62*, 1478–1483.
53. van Dulmen, M. H., Klipfel, K. M., Mata, A. D., Schinka, K. C., Claxton, S. E., Swahn, M. H., & Bossarte, R. M. (2012). Cross-lagged effects between intimate partner violence victimization and suicidality from adolescence into adulthood. *Journal of Adolescent Health, 51*, 510–516.
54. Caro-Astorga, J., Pérez-García, A., de Vicente, A., & Romero, D. (2015). Genomic region involved in the formation of adhesin fibers in *Bacillus cereus* biofilms. *Frontiers in Microbiology, 5*, Article 745.
55. Chon, W., Kim, J. H., Lee, S. J., Hyeon, J. Y., & Seo, K. H. (2012). Toxin profile, antibiotic resistance, and characterization of *Bacillus cereus* in Sunsik. *Food Microbiology, 32*, 217–222.
56. Simões, M., Simões, L. C., & Vieira, M. J. (2010). A review of current and emergent biofilm control strategies. *LWT - Food Science and Technology, 43*(4), 573–583.
57. Peters, B. M., Ovchinnikova, E. S., Krom, B. P., Schlecht, L. M., Zhou, H., Hoyer, L. L., Busscher, H. J., van der Mei, H. C., Jabra-Rizk, M. A., & Shirtliff, M. E. (2012). *Staphylococcus aureus* adherence to *Candida albicans* hyphae is mediated by the hyphal adhesin Als3p. *Microbiology, 158*(12), 2975–2986.

58. Bamford, C. V., d'Mello, A., Nobbs, A. H., Dutton, L. C., Vickerman, M. M., & Jenkinson, H. F. (2009). *Streptococcus gordonii* modulates *Candida albicans* biofilm formation through intergeneric communication. *Infection and Immunity, 77*(9), 3696–3704.
59. Jia, K., Fang, T., Wang, X., Liu, Y., Sun, W., Wang, Y., Ding, T., Wang, J., Li, C., Xu, D., et al. (2020). Antibiotic resistance patterns of *Staphylococcus aureus* isolates from retail foods in mainland China: A meta-analysis. *Foodborne Pathogens and Disease, 17*, 296–307.
60. Olivares, E., Badel-Berchoux, S., Provot, C., Prévost, G., Bernardi, T., & Jehl, F. (2020). Clinical impact of antibiotics for the treatment of *Pseudomonas aeruginosa* biofilm infections. *Frontiers in Microbiology, 10*, 2894.
61. Ciofu, O., Rojo-Molinero, E., Macià, M. D., & Oliver, A. (2017). Antibiotic treatment of biofilm infections. *APMIS, 125*, 304–319.
62. Bernal-Mercado, A. T., Gutierrez-Pacheco, M. M., Encinas-Basurto, D., Mata-Haro, V., Lopez-Zavala, A. A., Ayala-Zavala, J. F., & Islas-Osuna, M. A. (2020). Synergistic inhibition of *E. coli* adhesion using catechin, vanillic and protocatechuic acids. *Journal of Applied Microbiology, 128*(2), 387–400.
63. Wolfram, J., Zhu, M., Yang, Y., Shen, J., Gentile, E., Paolino, D., Fresta, M., Nie, G., Chen, C., Shen, H., & Ferrari, M. (2015). Safety of nanoparticles in medicine. *Current Drug Targets, 16*(14), 1671–1681.
64. Pinto, R. M., Lopes-De-Campos, D., Martins, M. C. L., van Dijck, P., Nunes, C., & Reis, S. (2019). Impact of nanosystems in *Staphylococcus aureus* biofilms treatment. *FEMS Microbiology Reviews, 43*, 622–641.
65. Bratanis, E., Andersson, T., Lood, R., & Bukowska-Faniband, E. (2020). Biotechnological potential of *Bdellovibrio*-like organisms and their secreted enzymes. *Frontiers in Microbiology, 11*, 662.
66. Banerjee, S., Vishakha, K., Das, S., Dutta, M., Mukherjee, D., Mondal, J., & Ganguli, A. (2020). Antibacterial, anti-biofilm activity of pancreatin-doped ZnO nanoparticles against MRSA. *Colloids and Surfaces B: Biointerfaces, 190*, 110921.
67. Zhang, S., Xiong, J., Lou, W., Ning, Z., Zhang, D., & Yang, J. (2019). Antimicrobial activity of triglycerol monolaurate on common foodborne pathogens. *Food Control, 98*, 113–119.
68. Zhao, F., Wang, W., Lu, W., Xu, L., Yang, S., Cai, X. M., Zhao, M., Lie, M., Ma, M., Xu, H., & Cao, F. (2018). High anticancer potency of dehydroabietyl amine Schiff-base derivatives and a copper (II) complex. *European Journal of Medicinal Chemistry, 146*, 451–459.
69. Krasnovskaya, O., Naumov, A., Guk, D., Gorelkin, P., Erofeev, A., Beloglazkina, E., & Majouga, A. (2020). Copper coordination compounds as biologically active agents. *International Journal of Molecular Sciences, 21*(11), 3965.
70. Fodil, S., Delgado, J., Varvaro, L., Yaseen, T., & Rodríguez, A. (2018). Effect of potassium sorbate (E-202) and the antifungal PgAFP protein on *Aspergillus carbonarius*. *Journal of the Science of Food and Agriculture, 98*, 5785–5794.
71. Fine, D. H., Goncharoff, P., Schreiner, H., Chang, K. M., Furgang, D., & Figurski, D. H. (2001). Colonization of *Actinobacillus actinomycetemcomitans* variants in rat mouths. *Archives of Oral Biology, 46*, 1065–1078.
72. Izano, E. A., Amarante, M. A., Kher, W. B., & Kaplan, J. B. (2008). Differential roles of polysaccharides and DNA in biofilms of *Staphylococcus aureus* and *S. epidermidis*. *Applied and Environmental Microbiology, 74*(2), 470–476.

73. Davies, D. G., & Marques, C. N. (2009). A fatty acid messenger induces dispersion in microbial biofilms. *Journal of Bacteriology, 191*(5), 1393–1403.
74. Khawary, M., Rakshit, R., Bahl, A., et al. (2023). M. tb-Rv2462c of *Mycobacterium tuberculosis* shows chaperone-like activity and modulates immunity. *Biology, 12*(1), 69. https://doi.org/10.3390/biology12010069