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REVIEW ARTICLE **OPEN ACCESS**

Approaches to prevent and treat bacterial biofilm on Implantable devices

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ABSTRACT
Microbial biofilms are complex communities of microorganisms that live in exopolysaccharide matrices adhering to an abiotic surface. Dispersal of the single and clustered cells implies a significant risk of microbial dissemination within the host and increased risk of infection. Such biofilms have been found to be a cause of a wide range of infections, be it chronic, nosocomial or medical device-related infections. The use of antibiotics has been rendered ineffective by the fact that these biofilms are drug-resistant. Due to the fact that treatment of an already formed biofilm with antibiotics is difficult, there should be concerted efforts to bring about strategies that can help in this regard. Measures such as antimicrobial coating and surface alterations of medical devices provide promising opportunities in the prevention of biofilm formation on medical devices.
Keywords; Biofilm, CAUTIs, Catheter coating, Quorum sensing, Infection and medical device

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INTRODUCTION

The human microbiota contains both unicellular and multicellular organisms which include bacteria, fungi, and viruses. It is estimated that such microbes outnumber human cells in an excess of factor of 10. The microbial flora are mostly found on the skin, in the oral cavity and also in the gastrointestinal tract. The normal flora play a major role including; Vitamin synthesis, ATP production and also play a critical role in innate defense mechanisms against pathogenic microorganisms [1]. Much as these microbes play a monumental role in the human body, their proliferation can be dangerous as they are prone to cause diseases and infections. This has been further compounded by the rapid rise of drug- resistant pathogens especially the bacteria.

Bacteria can exist either in planktonic state or in sessile state [2] both of which have distinct characteristics. This is because bacterial adhesion onto a surface alters expression of genes responsible for exopolysaccharide production and also maturation. Alteration of genes in sessile bacteria commences at initial attachment of bacteria which necessitates the production of a protective exopolysaccharide matrix. The matrix protects the bacteria against the organism's defense mechanism and external agents including antibiotics [3].

In this review, we highlight some approaches and technique which could be useful in handling the problems associated with biofilms especially the infections arising from this and through this, we hope to inspire the development of implants that can be able to adhesion of microbes which subsequently form biofilms.

URINARY CATHETERS

Urinary catheters are mostly used to collect urine during surgery and or prevent urine retention or control urinary incontinence [4]. For catheterized patients, the chances of acquiring a CAUTI increases by up to 10% each passing day. Microbial biofilms develop on both the inner and outer surfaces of urinary

catheters [5], and this makes it difficult to prevent ascending colonization by merely observing hygiene. Even so, steps like using catheters when absolutely necessary, short term catheterization and regular exchange of catheters have been shown to play an important role in the prevention of CAUTIs.

Various sources of the contaminating bacteria have been indicated to be; the normal biota that colonize the periurethral skin, which move into the bladder via the mucoid layer that forms between the epithelial surface or the urethra and the catheter. Bacteria from contaminated urine in urine collection bags can as well be a source of infection in patients [6].

To prevent the ascension of these bacteria, a number of strategies have been used. They include; removal and replacement of the catheter which unfortunately has not been effective due to the frequent disruption of the catheter and replacement which could be a genesis of other complications and shedding of parts of the biofilm from the indwelling device which enables the spread of infecting bacteria to previously uncolonized sites.

The production of urease by some bacteria, particularly *P. mirabilis*, increase the pH of urine. This increase in pH is then responsible for the formation of crystalline biofilms within and without the urinary catheter, around the balloon and catheter tip. This could cause trauma to the bladder and urethral epithelia. Crystalline biofilms could also block the catheter lumen thus preventing urine flow through the catheter [7].

BIOFILM FORMATION

Anthony van Leeuwenhoek in 1684 first observed a surface- associated bacteria. However, the term 'biofilm' was first used and defined in a report by WHO would later on called it a "a structured community of bacterial cells enclosed in a self-produced polymeric matrix, adherent to a surface." Biofilm importance was recognized by The American Society for Microbiology in 1993 [2]. Bacteria can form biofilms on medical implants, Bacteria like *E. faecalis*, *S. aureus*, *S. epidermidis*, *S. viridans*, *E. coli*, *K. pneumoniae*, *P. mirabilis* and *P. aeruginosa* are most common in forming biofilms on such implants [8].

Biofilm formation is mostly a cyclic process involving multiple bacterial species [9]. It is a complex process involving four major steps. Biofilms secrete a sticky extracellular polymeric substances which make up of about 80% of any biofilm. The extracellular polymeric substance is crucial in biofilm formation and comprises of water channels that are necessary for distribution of nutrients and oxygen and also serves to protect the bacteria from from both physical and chemical compounds and also the hosts defense mechanism [3] beside being the basic platform for surface attachment [10].

Biofilms are basically formed as n adaptive mechanism by the microorganisms as a defense mechanism, to help retain nutrients, and to ensure survival [2]. Biofilm formation can be classified into three stages; initial attachment, maturation of microcolonies, and dispersion [11] . For attachment, bacterial adhesins that stick to the surface are produced. As cell to cell adhesion processes aid in maturation, the biofilm degrading enzymes aid in the dispersal [12].

Once planktonic cells are exposed to a conditioning film, they attach by either physical forces or bacterial appendages (pili or flagella) [2,3]. This stage is otherwise known as reversible attachment since the interaction can be reversed due to the fact that the bacteria are attached to the surface by weak interactions [13]. The rate and extent of attachment of the bacteria on a surface is dependent on factors like, nature of the material, temperature and surface properties of the bacterial cell. Attachment of bacterial cells to a surface is referred to as adhesion while cohesion is the process of cell to cell attachment [3]. Biofilms adhere to surfaces by hydrophobic interactions, protein adhesion, electrostatic interactions, and weak Van der Waal forces [14].

Irreversible attachment occurs when attractive forces overpower the repulsion. This causes the reversibly attached cells to remain clamped together and become irreversibly attached, which subsequently leads to the formation of specific, strong adhesion and monolayer formation [3]. Adhesins include covalently anchored cell wall proteins (like SasX), and non-covalently associated proteins and non-protein factors [15]. In a developing biofilm, Planktonic cells convert into the sessile form due to the accumulation of Acylated Homoserine lactones [16]. The biofilm then fully matures to again generate Planktonic microorganisms. The Planktonic cells due to unavailability of nutrients, decreased pH and or accumulation of toxic metabolic byproducts scatter to colonize newer surfaces. The dispersed cells retain some characteristics from the previous biofilms including antimicrobial resistance.

Understanding the process of biofilm formation is the key to unlocking this problem especially on mechanisms to prevent, control or eliminate the biofilms. The possible mechanisms may include; manufacture of biomaterials that can prevent microbial adhesion on their surfaces, strategies to disrupt the signal molecules that modulate biofilm formation and also a way that can disintegrate the already formed matrix [17].

MECHANISMS OF BIOFILM RESISTANCE

Unlike in Planktonic bacteria, sessile microorganisms in the biofilm have developed adaptive mechanisms responsible for the enhanced resistance to external antimicrobial treatments. A number of theories have been advanced to explain this phenomenon. One such theory explains that that this could be as a result of slow or incomplete penetration of antimicrobial compounds through the biofilm's EPS matrix [17]. The EPS is also able to protect the biofilm from external stimuli like UV light and dehydration besides neutralizing the antimicrobial compounds [18]. Khoury *et al.,* (1992) reported that mature biofilms can be resistant to an excessive of up to 5000 times the concentration of bactericidal agents that completely destroy the same organism in a planktonic state [19].

The theorem of incomplete penetration was however rebutted by Singh *et al*., [20] who tested the efficacy of oxacillin, cefotaxime, amikacin, ciprofloxacin and vancomycin against *S. aureus* ATCC 29213 and *S. epidermidis* ATCC 35984 in 48 h laboratory-grown biofilms. According to their results, there was a reported reduction in the penetration of oxacillin, cefotaxime and vancomycin, while amikacin and ciprofloxacin showed no significant reduction in biofilm penetration.

In yet another theory, Hall-Stoodley *et al*., explain that biofilms adapt themselves against the antimicrobials through their slow growth rate. This to them is able to then protect the biofilm as antimicrobials require certain degree of cellular activity so as properly to function [18]. Other adaptive mechanisms aiding in the antimicrobial resistance acquired by certain micro-organisms within biofilms are; presence of efflux pumps, with the expression of several gene-encoding efflux pumps being increased in biofilms [21]. The increased rates of plasmid exchange in biofilms increasing the chances of developing naturally occurring and antimicrobial-induced resistance [22]. Also, an altered micro-environment within a biofilm, such as nutrient depletion and reduced oxygen levels, may also reduce the efficacy of antimicrobials [17].

ANTIBACTERIAL COATINGS

Catheter coating with antibacterial agents is an approach that can suppress the initial attachment of free living bacteria on the catheter implant surface. Even though it is a promising procedure, initially there were concerns due to the menace of rise of antibiotic resistance. However, this has led to the evolution better strategies aimed at coating the implants using natural and synthetic materials [14].

Veerachamy *et al*., [14] reported that widely used Hydroxyapatite based coatings which can be altered by the use of surface adsorbed antimicrobials by soaking them in antibiotic solutions. Some other strategies to inhibit bacterial attachment on catheters include; hydrophilic polymers such as hyaluronic acid, hydrogel and heparin coatings [8].

Silane and nanoplasma trimethyl silane coatings were much effective on hydrophobic surfaces against biofilms formed by *S. epidermidis* [3]. Moreover, commercially available covalently coupled quaternary ammonium silane coatings are in use. However, because of time constraints in their use and also due to their specific, their use has not been a great success [23]. Antimicrobial peptides produced by animals, plants and various microorganisms have been documented as being effective in destroying LL37 peptide when grafted on titanium surfaces [24].

Kannan, 2015 [25] reported that to achieve long-term antifouling on biomaterials, they could be coated with calcium phosphate and other biodegradable polymers, and talking of polymers, [26] stated that use of anti-adhesive high-density polymers were able to inhibit bacterial adhesion due to the presence of steric repulsion. Coating the implants with nanomaterials and nanofilms is also gaining traction . By the fact that they have been able to eliminate biofilm forming microorganisms, and due to their biocompatible nature, silver nanoparticles that have been predominantly used. The nanoparticles are easily be destroyed by the mammalian cells hence reducing chances of toxicity and free radical damage. The silver nanoparticles are able to bind to the microorganisms' cell walls and subsequently destroying it and as such inhibit biofilm formation [14]. Through accumulation of peroxides that oxidise the cell walls, disruption of the bacteria's respiratory chain, cell disruption via hydroxyl radicals and reactive oxygen species [27], silver nanoparticles are able to eradicate bacterial cells.

Unfortunately, due to the silver nanoparticles being biocompatible, their use is only limited as they cannot be used in long term as they can be a cause of surgical site infections [28]. Success in Biomaterial coating using gold, diamond and titanium has been reported as being able to inhibit microbial adhesion, proliferation and biofilm growth [14].

USE OF ANTIBIOTIC ENHANCERS

Autoinducers are the signal molecules responsible for the communication between Gram-positive and gram-negative bacteria. The autoinducers in Gram positive bacteria are oligopeptides and N-acyl homoserine lactones in gram-negative bacteria [17]. The use of antibiotics enhancers to inhibit biofilm

formation is a milestone in the fight against this menace. Jia *et al*., [29] illustrated that D-tyrosine which is an antimicrobial enhancer enhanced ciprofloxacin and successfully prevented *P. aeruginosa* biofilm grown on C1018 carbon steel, while also reducing the ciprofloxacin dosage.

QS plays a significant role in biofilm survival [30]. For instance, Las QS system which is regulated by rpoS gene is responsible for antibiotic tolerance in *P. aeruginosa* [31]. To prevent QS, simply inhibit signal molecules from binding to the receptor through analogues of signal molecules [32].

In order to prevent QS, signaling molecules involved in binding to the receptors on the bacterial cell surface must be curtaileds. This can be achieved through the application of analogues of signal molecules that compete for binding sites [17]. Bjarnsholt, 2013 reported that through prevention of aggregate formation or dissolution of the EPS, exposed bacterial cells can be rendered vulnerable to therapies again [2]. QS inhibitors and antagonists such as halogenated furanone for Gram negative [33] and RNAIII inhibiting peptide (RIP) are promising therapeutic tools for the treatment of biofilm-based infections.

RIP coated CVCs reduced bacterial load and enhanced the effect of tested antibiotics in the treatment of CVC-associated S. aureus infections in a rat model. Through the use of RIP, the S. aureus are as susceptible to antibiotics as planktonic cells [34]. They also reported about antibiotic enhancers such as D-amino acids, ethylenediaminetetraacetic acid, ethylenediaminedisuccinate, norspermidine and bacteriophages. The enhancers are necessary especially to enhance both antibiotics and biocides in order to inhibit and treat industrial biofilm. The same can also be adopted in medical biofilms as well. Polystyrene, glass and mica surfaces treated with two benzoate derivatives prevent the adhesion of S. epidermidis. Further, Qin *et al*. stated that carboximide derivatives reacted bacterial cells to inhibit both adhesion and cell division. Much as both were able to prevent initial attachment of the bacteria, they were however not able to destroy the sessile bacteria [35].

By disrupting the c-di-GMP biosynthesis, sulphathiazole was able to prevent biofilm formation by *E. coli* [3]. A combination of several polymers with lipid II, ginger extracts, Chinese medicinal plants, and some proteases [36], have been able to disrupt QS pathways and hinder biofilms in several species like *E. coli*, *S. epidermidis*, *S. aureus*, *S. mutans* and *P. aeruginosa* [3]. DNase degrades eDNA secreted by *S. aureus* thus hindering biofilm formation. Surfaces adsorbed with albumin can also inhibit bacterial attachment to polymers, and a range of metal surfaces [37]. The use of anticoagulants and antimicrobial agents on catheters also disrupts bacterial growth [38]. A mixture of some antibiotics and D-amino acids secreted by some bacteria prevent biofilm formation by *P. aeruginosa* [29,39].

BIOACOUSTIC EFFECT

Eradication of biofilms using antibiotic therapy is simply "mission impossible! It is because of this reason that research has focused on ways to try and redeem us from this problem. Bioacoustic effect is the use of ultrasound to enhance the effectiveness of antibiotics by increasing antibiotic transport across the biofilms through cell membrane disruption caused by high pressure, high shear stress and or cavitation [40].

A combination of ultrasound waves and gentamycin in bone cements prevented up to 70% of biofilm formation in an in vivo rabbit model [41]. In an indwelling catheter model, low frequency acoustic waves combined with gentamicin successfully inhibited an excess of 85% of *E. coli*, *S. epidermidis* and *P. aeruginosa* biofilms [42]. Ultrasonication mediated microbubbles methods integrated with vancomycin, aminoglycoside or gentamicin were also able to prevent biofilm formation by *S. epidermidis*, *E. coli*, and *P. aeruginosa* [43, 44].

Surface modification of biomaterials

As the prevention of biofilm is easier than elimination, safer and also cheaper, surface modification of the implants to prevent biofilm formation at a later stage is a technique that can also be used [45]. Microbial adhesion on devices can be prevented by coating the surface with hydrophilic polymers. This is because the microbial surfaces are hydrophobic nature in nature [46].

Biomaterial properties like surface area, texture and hydrophilicity play a vital role in microbial attachment on surfaces as they can serve to either increase or decrease protein adsorption [47]. According to Delaviz *et al*., [48] the adhesion of S. epidermidis and E. coli on the material was partly due to the stiffness.

It is such correlations that can be helpful to determine the possible modificationsand optimize biomaterial surfaces to prevent adhesion leading to biofilms [25, 26]. Polymers like as hyaluronic acid [49] (and poly-N-vinylpyrrolidone, have been reported to reduce the adhesion of *S. epidermidis.* The antiadhesive property of heparin is able to reduce CAUTIs [50]. Making the catheter surface negatively charged using heparin, fibronectin deposition is inhibited which reduces microbial adhesion [51].

Antimicrobial photodynamic therapies

Photodynamic therapy is based on the technique in which Reactive Oxygen Species (ROS) is produced when photosensitizers or light absorbing compounds react with light and oxygen and this can be helpful in the eradication of sessile bacterial cells thus reffered to as antimicrobial photodynamic therapy [17]. Light sources such as yttrium aluminum garnet (YAG) lasers [52], potassium yttriumtungstate (KYW) lasers, and femtosecond [53] and near-infrared lasers [54] have been used to control biofilms in both *in vitro* and *in vivo* studies.

This technique has been used to destroy biofilms using diode lasers and different photosensitizers on various surfaces like acrylic resin and glass some of which acted in synergistic with antibiotic treatment [52]. A combination of antibiotics with a broad range of light in the electromagnetic spectrum has an ability of destroying bacteria [55].

Early diagnosis of biofilm formation using biosensors

The first step towards diagnosis, prevention and control of biofilms is early diagnosis of infections. The detection can be through the use of biosensors and hyperspectral imaging methods [56]. As mentioned earlier, treatment of a bacterial biofilm is almost a mission impossible. This has not been made any easier by the fact that even an effective diagnosis of a biofilm is a monumental task.

Some of the most recent diagnostic techniques include Scanning Electron Microscopy (SEM), C-T scans, MRIs and Atomic Force Microscopy among others [57]. However, even with such sophisticated methods, biofilm detection is still an uphill task [58]. To overcome this challenge, much effort must be directed to improving and developing new diagnosis/quantification techniques such as improved CFU counting, various types of microscopy, FT-IR spectroscopy, contact angle measurements, and Qpcr [57].

For molecular sensing, Surface-enhanced Raman spectroscopy (SERS) contains several sharp peaks corresponding to specific molecular vibrational frequencies which indicate the presence of specific molecules in samples. Through SERS, detection of *Salmonella*, *E. coli*, *S. epidermidis* and *Bacillus*, through synthesis of silver nanoparticle on bacterial cell walls has been enhanced [59]. Prevention is better that cure, so goes the old adage. It is easier and cheaper to prevent biofilm than to try eradicate an already formed biofilm. It is then for this reason that more focus should be directed to developing and improving sensing/detection technologies.

Impregnation of catheters with actinobacteria extracts

Antibiofilm compounds could be potentially sourced from actinobacteria metabolic compounds. Previous research has been able to indicate actinobacteria as playing a pivotal role in the development of antibiofilm compounds [60]. In our previous research, *Strptomyces sp.* ABK07 isolated from Kolli hills was able to inhibit biofilm formation by the UTI pathogens (*Escherichia coli*, *Proteus*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Klebsiella*, and *Candida albicans* on urinary catheters [61].

CONCLUSION

It absolutely clear that biofilm eradication is a huge challenge. In this review article, we have outlined the various stages of bacterial formation on implants and the potential technologies that can be used with an intent to prevent and/or treat biofilms. Where bacterial biofilms in vivo models are involved, Utmost care must be observed because bacteria behave differently when compared to *in vitro* models. The struggle against biofilm is however faced with several obstacles ranging from the rise of antibacterial resistance to phagocytes to recurrence of infections after treatment. To overcome the issue resistance and the unnecessary surgeries, the aforementioned technologies are under study both in in vitro and in vivo models. The success of these technologies will be a new dawn in the treatment and eradication of biofilms.

REFERENCES

1. Cochis, A., Azzimonti, B., Della Valle, C., De Giglio, E., Bloise, N., Visai, L., Chiesa, R. (2016). The effect of silver or gallium doped titanium against the multidrug resistant Acinetobacter baumannii. *Biomaterials*, *80*, 80-95.
2. T. Bjarnsholt (2011). Introduction to biofilms, in: T. Bjarnsholt, P.Ø. Jensen, C. Moser, N. Høiby (Eds.), Biofilm Infections, Springer, New York, NY, pp. 1-9.
3. Gupta, P., Sarkar, S., Das, B., Bhattacharjee, S., Tribedi, P. (2016). Biofilm, pathogenesis and prevention e a journey to break the wall: a review, Arch. Microbiol. 198 (1) 1-15.
4. Kaye, D., & Hessen, M. T. (1994). Infections associated with foreign bodies in the urinary tract. *Infections associated with indwelling medical devices. 2nd ed. Washington: American Society for Microbiology*, 291-307.
5. Donlan, R. M. (2001). Biofilms and device-associated infections. Emerg Infect Dis 7, 277–281.
6. Stickler, D. J. (2008). Bacterial biofilms in patients with indwelling urinary catheters. *Nature clinical practice urology*, *5*(11), 598-608.
7. Percival, S. L., Sabbuba, N. A., Kite, P. & Stickler, D. J. (2009). The effect of EDTA instillations on the rate of development of encrustation and biofilms in Foley catheters. Urol Res 37(4), 205–209.

8. Chen, M., Yu, Q., Sun, H. (2013). Novel strategies for the prevention and treatment of biofilm related infections, Int. J. Mol. Sci. 14 (9) 18488-18501.
9. Al-Ahmad, A., Kürschner, A., Weckesser, S., Wittmer, A., Rauberger, H., Jakob, T., ... & Waidner, B. (2012). Is Helicobacter pylori resident or transient in the human oral cavity?. *Journal of medical microbiology*, *61*(8), 1146-1152.
10. Chang, C. (2017). Surface sensing for biofilm formation in Pseudomonas aeruginosa, Front. Microbiol. 8.
11. Jamal, M., Ahmad, W., Andleeb, S., Jalil, F., Imran, M. (2018). Bacterial biofilm and associated infections, J. Chin. Med. Assoc. 81 (1) 7-11.
12. Boles, BR.., Horswill, A.R. (2011). Staphylococcal biofilm disassembly, Trends Microbiol. 19 (9) 449-455.
13. Floyd, K.A., Eberly, A.R., Hadjifrangiskou, M. (2017). Adhesion of Bacteria to Surfaces and Biofilm Formation on Medical Devices, Biofilms and Implantable Medical Devices, pp. 47-95.
14. Veerachamy, S., Yarlagadda, T., Manivasagam, G. (2014). Bacterial adherence and biofilm formation on medical implants: a review, Proc. Inst. Mech. Eng. H 228 (10) 1083-1099.
15. Paharik, A.E., Horswill, A.R. (2016). The staphylococcal biofilm: adhesins, regulation, and host response, Microbiol. Spectr. 4 (2).
16. Davies, D.G., Parsek, M.R., Pearson, J.P., Iglewski, B.H., Costerton, J., Greenberg, E. (1998) The involvement of cell-to-cell signals in the development of a bacterial biofilm. Science. 280(5361):295-8.
17. Francolini, I., Donelli, G. (2010). Prevention and control of biofilm-based medical-device- related infections, FEMS Immunol. Med. Microbiol. 59 (3) 227-238.
18. Stoodley, P., Hall-Stoodley, L., Costerton, B., DeMeo, P., Shirtliff, M., Gawalt, E. (2013). Biofilms, Biomaterials, and Device-Related Infections, Handbook of Polymer Applications in Medicine and Medical Devices, pp. 77-101.
19. Khoury, A., Lam, E., Ellis, K., Costerton, J. W. (1992). Prevention and control of bacterial infections associated with medical devices. ASAIO J 38, M174–M178.
20. Singh, R., Ray, P., Das, A. & Sharma, M. (2010). Penetration of antibiotics through Staphylococcus aureus and Staphylococcus epidermidis biofilms. J Antimicrob Chemother 65, 1955–1958.
21. Soto, S. M. (2013). Role of efflux pumps in the antibiotic resistance of bacteria embedded in a biofilm. Virulence 4, 223–229.
22. Hausner, M., & Wuertz, S. (1999). High rates of conjugation in bacterial biofilms as determined by quantitative in situ analysis. Appl Environ Microbiol 65, 3710–3713.
23. Rodrigues, L., Banat, I.M., Teixeira, J., Oliveira, R. (2007). Strategies for the prevention of microbial biofilm formation on silicone rubber voice prostheses, J. Biomed. Mater. Res. B 81 (2) 358-370.
24. Barnes L., Cooper I.R. Elsevier; 2015. Biomaterials and Medical Device – Associated Infections.
25. Kannan, M.B. (2015). Biodegradable Polymeric Coatings for Surface Modification of Magnesium-Based Biomaterials, Surface Modification of Magnesium and its Alloys for Biomedical Applications, pp. 355-376.
26. Neoh, K.G., Wang, R., Kang, E.T (2015). Surface Nanoengineering for Combating Biomaterials Infections, Biomaterials and Medical Device e Associated Infections, pp. 133-161.
27. Griffith, M., Udekwu, K.I., Gkotzis, S. (2015). Anti-microbiological and anti-infective activities of silver, in: E.I. Alarcon, M. Griffith, K.I. Udekwu (Eds.), Silver Nanoparticle Applications: In the Fabrication and Design of Medical and Biosensing Devices, Springer International Publishing, pp. 127-146.
28. Khare,M.D., Bukhari, S.S., Swann, A., Spiers, P., Laren, J. (2007). Myers, Reduction of catheter-related colonisation by the use of a silver zeoliteimpregnated central vascular catheter in adult critical care, J. Infect. 54 (2) 146-150.
29. Jia, R., Yang, D., Xu, D., Gu, T. (2017). Mitigation of a nitrate reducing Pseudomonas aeruginosa biofilm and anaerobic biocorrosion using ciprofloxacin enhanced by D-tyrosine, Sci. Rep. 7 (1) 6946.
30. Kiran, M.D., Giacometti, A., Cirioni, O., Balaban, N. (2008) Suppression of biofilm related, device-associated infections by staphylococcal quorum sensing inhibitors. Int J Artif Organs 31: 761–770.
31. Kayama, S., Murakami, K., Ono, T., Ushimaru, M., Yamamoto, A., Hirota, K., Miyake, Y. (2009) The role of rpoS gene and quorum-sensing system in ofloxacin tolerance in Pseudomonas aeruginosa. FEMS Microbiol Lett 298: 184–192.
32. Gonzalez, J.E., Keshavan, N.D. (2006) Messing with bacterial quorum sensing. Microbiol Mol Biol R 70: 859–875.
33. Givskov, M., de, N.R., Manefield, M., Gram, L., Maximilien, R., Eberl, L., Molin, S., Steinberg, P.D., Kjelleberg, S. (1996) Eukaryotic interference with homoserine lactone-mediated prokaryotic signalling. J Bacteriol 178: 6618–6622.
34. Cirioni, O., Giacometti, A., Ghiselli, R. (2006) RNAIIIinhibiting peptide significantly reduces bacterial load and enhances the effect of antibiotics in the treatment of central venous catheter-associated Staphylococcus aureus infections. J Infect Dis 193: 180–186.
35. Qin, Z., Zhang, J., Hu, Y., Chi, Q., Ulstrup, J. (2009). Organic compounds inhibiting S. epidermidis adhesion and biofilm formation, Ultramicroscopy 109 (8) 881-888.
36. Chaignon, P., Sadovskaya, I., Ragunah, C., Ramasubbu, N., Jabbouri, S. (2007). Susceptibility of staphylococcal biofilms to enzymatic treatments depends on their chemical composition, Appl. Microbiol. Biotechnol. 75 (1) 125-132.
37. Katsikogianni, M., Missirlis, Y.F. (2004). Concise review of mechanisms of bacterial Adhesion to biomaterials and of techniques used in estimating bacteriamaterial interactions, Eur. Cell Mater. 8 37-57.
38. Zhang, L., Gowardman, J., Rickard, C.M. (2011). Impact of microbial attachment on intravascular catheter-related infections, Int. J. Antimicrob. Agents 38 (1) 9-15.

39. Kolodkin-Gal, I., Romero, D., Cao, S., Clardy, J., Kolter, R., Losick, R. (20100 Damino acids trigger biofilm disassembly, Science 328 (5978) 627-629.
40. Drago, L., Toscano, M. (2017). Biofilm Formation and the Biological Response, Management of Periprosthetic Joint Infections (PJIs), pp. 25-39.
41. Ensing, G.T., Roeder, B.L., Nelson, J.L., Pitt, W.G. (2005). Effect of pulsed ultrasound in combination with gentamicin on bacterial viability in biofilms on bone cements in vivo, J. Appl. Microbiol. 99 (3) 443-448.
42. M. Kopel, E. Degtyar, E. Banin, Surface acoustic waves increase the susceptibility of Pseudomonas aeruginosa biofilms to antibiotic treatment, Biofouling 27 (7) (2011) 701-710.
43. Dong, Y., Chen, S., Wang, Z., Peng, N., Yu, J. (2013). Synergy of ultrasound microbubbles and vancomycin against Staphylococcus epidermidis biofilm, J. Antimicrob. Chemother. 68 (4) 816-826.
44. Cai, Y., Wang, J., Liu, X., Wang, R., Xia, L. (2017) A review of the combination therapy of low frequency ultrasound with antibiotics, BioMed Res. Int. 2317846.
45. Puiu, R.A., Dolete, G., Ene, A.M., Nicoara, B., Vlasceanu, G.M., Bolocan, A. (2017). Properties of Biofilms Developed on Medical Devices, Biofilms and Implantable Medical Devices, pp. 25-46.
46. Van der Mei, H.C., Leonard, A.J., Weerkamp, A.H., Rouxhet, P.G., Busscher, H.J (1988) Surface properties of Streptococcus salivarius HB and nonfibrillar mutants: measurement of zeta potential and elemental composition with X-ray photoelectron spectroscopy. J Bacteriol 170: 2462–2466.
47. Moriarty, T.F., Zaat, S.J.J., Busscher, H.J. Biomaterials Associated Infection: Immunological Aspects and Antimicrobial Strategies, Springer New York, New York, NY, 2013.
48. Delaviz, Y., Santerre, J.P., Cvitkovitch, D.G. (2015). Infection resistant biomaterials, in: L. Barnes, I.R. Cooper (Eds.), Biomaterials and Medical Device e Associated Infections, Woodhead Publishing, Oxford, pp. 223-254.
49. Cassinelli, C., Morra M., Pavesio, A., Renier, D. (2000) Evaluation of interfacial properties of hyaluronan coated poly(methylmethacrylate) intraocular lenses. J Biomat Sci- Polym E 11: 961–977.
50. Abdelkefi, A., Achour, W., Ben, O.T., Ladeb, S., Torjman, L., Lakhal, A., Ben, H.A., Hsairi, M., Ben, A.A. (2007) Use of heparin-coated central venous lines to prevent catheter-related bloodstream infection. J Support Oncol 5: 273–278.
51. Russell, P.B., Kline, J., Yoder, M.C., Polin, R.A. (1987) Staphylococcal adherence to polyvinyl chloride and heparin-bonded polyurethane catheters is species dependent and enhanced by fibronectin. J Clin Microbiol 25: 1083–1087.
52. Leblebicioglu, B., Eick, S., Meier, I., Sculean, A. (2017). In vitro-activity of Er:YAG laser in comparison with other treatment modalities on biofilm ablation from implant and tooth surfaces, PLoS One 12 (1).
53. Doll, K., Fadeeva, E., Schaeske, J., Ehmke, T., Winkel, A., Heisterkamp, A., Chichkov, B.N., Stiesch, M., Stumpp, N.S. (2017). Development of laser-structured liquid-infused titanium with strong biofilm-repellent properties, ACS Appl. Mater. Interf. 9 (11) 9359-9368.
54. Damestani, Y., De Howitt, N., Halaney, D.L., Garay, J.E., Aguilar, G. (2016). Evaluation of laser bacterial anti-fouling of transparent nanocrystalline yttriastabilized- zirconia cranial implant, Lasers Surg. Med. 48 (8) 782-789.
55. Yin, R., Dai, T., Avci, P., Jorge, A.E., de Melo, W.C., Vecchio, D., Huang, Y.Y., Gupta, A., Hamblin, M.R. (2013). Light based anti-infectives: ultraviolet C irradiation, photodynamic therapy, blue light, and beyond, Curr. Opin. Pharmacol. 13 (5) 731-762.
56. Raghavachari, R., Kim, D.H., Kim, M.S., Hwang, J., Liang, R. Monitoring of biofilm formation on different material surfaces of medical devices using hyperspectral imaging method, Design Qual. Biomed. Technol. V (2012).
57. Ribeiro, M., Monteiro, F.J., Ferraz, M.P. (2012). Infection of orthopedic implants with emphasis on bacterial adhesion process and techniques used in studying bacterial-material interactions, Biomatter 2 (4) 176-194.
58. Zimmerli, W., Trampuz, A., Ochsner, P.E. (2004). Prosthetic joint infections, N. Engl. J. Med. 351 (16) 1645-1654.
59. Khetani, A., Momenpour, a., Tiwari, V.S., Anis, H. Surface enhanced Raman Scattering (SERS) using nanoparticles, in: E.I. Alarcon, M. Griffith, K.I. Udekwu (Eds.), Silver Nanoparticle Applications: In the Fabrication and Design of Medical and Biosensing Devices, Springer International Publishing, Cham, 2015, pp. 47-70.
60. Venugopal, G., Raasaiyah, P., Thangavel, S., Manikkam, R. and Ramasamy, B. (2013). Bioprospecting of Actinobacteria from mangrove and estuarine sediments for antifouling compounds. Intl. J. Innov. Res. Sci. Engg. Techn., 2(7): 2726-2735.
61. Kingsley, Angima., Usha, Rajamanickam. (2018). Modification of urinary catheters using antimicrobials from Streptomyces sp. ABK 07 for urinary tract infection resistance. Asian Journal of Pharmaceutical and Clinical Research. Pp. 158-62

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