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Research Article

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The Role of Skin Microbiome Dysbiosis in Orthopedic Implant Failure and Infection

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Abstract

The skin microbiome, a dynamic ecosystem of commensal and pathogenic microorganisms, plays a critical role in maintaining immune homeostasis and barrier integrity at surgical sites. Yet, its disruption has been increasingly implicated in postoperative infections in orthopedic surgery. Dysbiosis, marked by an overrepresentation of opportunistic pathogens such as *Staphylococcus aureus* and *Cutibacterium acnes*, often arises from preoperative antiseptic protocols and prolonged hospital exposures. An imbalance compromises the skin's natural defense mechanisms, facilitating microbial translocation to the implant surface and promoting biofilm formation. Biofilms, composed of extracellular polymeric substances, protect embedded bacteria from immune responses and antimicrobial agents, significantly increasing the risk of chronic periprosthetic infections, and eventual implant failure. Reduced diversity of commensal species, such as *Staphylococcus epidermidis*, further exacerbates the risk, as these organisms typically inhibit pathogen colonization through competitive exclusion and antimicrobial peptide secretion. Emerging evidence suggests that targeted microbiome restoration strategies, including the application of topical probiotics, prebiotics, or bacteriophage therapies, may re-establish microbial equilibrium and reduce infection susceptibility. Additionally, innovations 'such as antimicrobial implant coatings and microbiome-preserving skin preparations' could enhance perioperative protocols by preventing dysbiosis-induced complications. High-resolution microbiome sequencing and predictive microbial modeling are advancing the identification of at-risk patients, enabling personalized approaches to infection prevention. Advancing understanding of the relationship between skin microbiome dysbiosis and implant-associated infections offers a paradigm shift in orthopedic surgery, emphasizing precision microbiome management to optimize surgical outcomes and implant longevity.

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Introduction

The skin microbiome is composed of diverse microorganisms, including bacteria, fungi, viruses, and mites, that are crucial in maintaining a host's homeostasis and barrier integrity. This broad range of microbes interacts with the host's immune system to maintain an intricate balance between commensal and pathogenic organisms, preventing the overgrowth of the pathogenic microbes and still supporting the host's skin barrier and protective functions. The commensals often seen within the skin microbiome include *Staphylococcus epidermidis* (*S. epidermidis*) and *Cutibacterium acnes* (*C. acnes*), which function to produce antimicrobial peptides that inhibit pathogen growth, and modulate immune responses to maintain skin health [1]. The skin microbiome's influence extends to the development and function of the host's immune system, as exemplified by *S. epidermidis'* ability to induce IL-17A production by CD8+ T cells, thereby enhancing innate barrier immunity and limiting pathogen invasion [2]. There are times when this intricate balance becomes disrupted, known as dysbiosis, leading to impaired barrier function, increased susceptibility to infections, and even inflammatory skin disorders. Triggers to disrupted homeostasis can include diet, genetic mutations, trauma, surgeries, and foreign bodies, potentially leading to cutaneous presentations, such as atopic dermatitis, acne, and psoriasis [3]. Understanding the complex interactions between commensal and pathogenic microorganisms and the host's immune system is essential for developing effective strategies to manage skin health and prevent disease, particularly in the context of orthopedic implant-related infections and failures.

Postoperative infections, particularly periprosthetic joint infections (PJIs), occur in approximately 1-2% of all total joint arthroplasty procedures [4]. These infections can significantly impact patient quality of life, morbidity, and mortality. Recent studies have shown a growing link between these infections and dysbiosis of the microbiome [5]. This emphasizes the complex interplay between the host's skin microbiome and surgical success. A significant challenge in managing PJIs is the formation of biofilms on orthopedic implants, which commonly involve skin commensals like *S. epidermidis* [5]. Biofilms create a protective environment for bacteria, preventing the host's immune system from effectively targeting them by limiting

antibiotic penetration. Furthermore, metabolically dormant cells can exist within these biofilms, exhibiting heightened antibiotic resistance, complicating treatment strategies, and increasing the risk of prolonged infection. This underscores the need for innovative strategies to prevent and treat periprosthetic joint infections to enhance patient outcomes and combat antibiotic resistance.

Identification of the inciting organism(s) in PJIs is not clear even with tissue biopsy and culturing methods, as culture-negative infections continue to rise [7]. This is sometimes attributed to possible biofilms and antimicrobial medication usage prior to culture sampling. It is challenging to accurately detect bacteria embedded within biofilms, which are very common on orthopedic implants, requiring more sophisticated molecular techniques for accurate diagnosis [8]. Unfortunately, these traditional approaches have high failure rates, and patients may require multiple attempts to successfully diagnose and eradicate PJIs. To address these challenges, researchers are exploring emerging strategies to improve outcomes. These strategies include developing anti-biofilm coatings for orthopedic implants that can help prevent bacterial adhesion. Additionally, local delivery of antibiotics can target infections effectively, which new technologies like intraosseous antibiotic delivery methods are exploring. Non-antibiotic methods, such as bacteriophages and quorum-sensing inhibitors, are also being investigated for their potential to disrupt biofilm formation [9]. These findings highlight the urgent need for a comprehensive understanding of the microbiome's role in postoperative infections and improved strategies to manage and prevent complications associated with orthopedic implant surgeries.

This literature review investigates the link between skin microbiome dysbiosis and the failure of orthopedic implants, emphasizing strategies to manage the skin microbiome to reduce postoperative complications. Recent studies have shown that the microbiome plays a crucial role in surgical outcomes, especially in orthopedic implant procedures, where dysbiosis can heighten the risk of infections and implant failures. This review analyzes how beneficial and harmful microorganisms interact on the skin and how these imbalances can lead to complications. It also highlights innovative microbiome management strategies, such as anti-biofilm coatings, localized antibiotic delivery systems, and non-antibiotic approaches like bacteriophages. This review discusses the potential advantages of preoperative microbiome assessments in identifying high-risk patients to enhance orthopedic surgical outcomes. This review aims to provide valuable insights into preventing and managing microbiome-related infections in orthopedic surgeries.

Overview of Skin Microbiome in Orthopedic Implant Failure and Infection

The skin microbiome is a dynamic and intricate ecosystem composed of commensal and opportunistic microorganisms, including bacteria, fungi, and viruses. These microbes play essential roles in maintaining skin health by protecting against pathogenic colonization, modulating immune responses, and supporting barrier integrity. The composition of the skin microbiota varies based on physiological factors, such as sebaceous (oily), moist, or dry regions, which foster the growth of specific microbial communities [10-12]. For example, *C. acnes* thrives in sebaceous environments by metabolizing sebum triglycerides into free fatty acids, shaping the lipid landscape and preventing pathogen overgrowth. However, under dysbiotic conditions, biofilm-forming strains of *C. acnes* can become pathogenic, contributing to chronic inflammation and conditions such as acne vulgaris [13,14]. In contrast, *S. epidermidis*, a key commensal species, enhances colonization resistance through the production of antimicrobial molecules like serine protease glutamyl endopeptidase, which disrupt *Staphylococcus aureus (S. aureus)* biofilms and reduce epithelial adhesion [15]. Beyond pathogen suppression, *S. epidermidis* promotes immune homeostasis by inducing cytokines such as interleukin-1α, which recruit immune cells and strengthen the skin barrier [2]. This balance, however, is fragile; disruptions caused by preoperative antiseptic protocols, prolonged hospital exposure, or skin barrier injury can tip the equilibrium, allowing opportunistic pathogens to dominate.

Before understanding how skin microbes are implicated in orthopedic infections, it is essential to first understand the mechanisms of infection in bacterial biofilms and their ability to evade the immune system. A biofilm is a structured community of bacteria encased in a protective matrix of extracellular polymeric substances (EPS), which includes polysaccharides, proteins, extracellular DNA, and lipids [16,17]. This matrix acts as a physical shield, preventing antibiotics and immune cells from penetrating and effectively targeting the bacteria. Within the biofilm, bacteria exhibit altered metabolic activity, often entering a dormant state that renders them less susceptible to antibiotics, which primarily target actively dividing cells [18]. Additionally, biofilm formation allows bacteria to adhere tightly to surfaces, such as implants or tissues, creating a persistent reservoir for infection [19]. The biofilm environment promotes horizontal gene transfer between bacteria, facilitating the spread of antibiotic resistance genes and further enhancing their survival [20]. To evade immune detection, bacteria within the biofilm can downregulate immunogenic surface proteins and release factors that suppress the host's immune response. As a result, immune cells like neutrophils and macrophages struggle to clear the infection, often leading to prolonged inflammation and tissue damage. This persistent and protective nature of biofilms makes infections extremely difficult to treat and prone to recurrence.

The opportunistic pathogens *S. aureus*, *C. acnes*, and *Pseudomonas aeruginosa (P. aeruginosa)* are commonly associated with orthopedic infections, particularly following surgical procedures or implant placements. *S. aureus* remains the predominant causative organism in both septic arthritis and osteomyelitis, accounting for up to two-thirds of all pathogens in orthopedic implant infections [21,22]. As a common colonizer of the skin, *S. aureus* can transition into a pathogenic state when the skin barrier is disrupted, especially during surgical interventions. This transition allows the bacterium to efficiently adhere to implant surfaces and form biofilms composed of extracellular polymeric substances [23]. Biofilm formation plays a critical role in infection persistence, as the protective matrix significantly reduces antibiotic penetration and shields the bacteria from immune responses. As a result, *S. aureus* can remain on implant surfaces for prolonged periods, leading to chronic PJIs that often necessitate surgical revision and removal of the infected hardware [24]. In addition to biofilm formation, *S. aureus* possesses immune evasion mechanisms that further complicate treatment. For instance, the bacterium produces protein A, which binds to the Fc region of immunoglobulins, inhibiting phagocytosis and impairing host immune clearance [25]. Additionally, toxin production disrupts

immune cell function, contributing to persistent infection and tissue damage. These combined mechanisms highlight the challenge of eradicating *S. aureus* infections in orthopedic settings, as the biofilm matrix and immune evasion strategies render conventional antibiotics and host defenses largely ineffective.

P. aeruginosa, another opportunistic pathogen, adds another layer of complexity to orthopedic infections by exploiting compromised skin to form robust biofilms that delay wound healing and promote chronic infections [26]. Noted for its environmental adaptability, *P. aeruginosa* rapidly colonizes surgical sites and implants, producing virulence factors such as proteases and elastase, which degrade host tissues and impair recovery [27]. Its quorum-sensing capabilities, which enable cell-to-cell communication within biofilms, allow the bacteria to coordinate biofilm formation, enhancing its resistance to antibiotics and immune clearance. Unlike many other pathogens, *P. aeruginosa* thrives in moist environments and produces dense, complex biofilms that further delay healing and predispose patients to chronic, treatment-resistant infections [28]. Compounding the issue, *P. aeruginosa* can synergize with other bacteria in polymicrobial infections, enhancing biofilm formation and virulence, which significantly complicates treatment outcomes [29]. Similarly, *C. acnes*, despite being a commensal organism, can exacerbate *Saureus* infections by producing coproporphyrin III, a molecule that facilitates *S. aureus* biofilm aggregation and further destabilizes the microbial balance [30]. This imbalance is particularly concerning because the persistence of biofilms and the entrenchment of infections generate sustained inflammation and microbial burden, which not only compromise implant integrity but also interfere with the natural process of osseous healing.

The mechanisms driving these osseous healing failures center on the disruption of the natural balance between bone formation and bone resorption. Persistent infection and biofilm production drive chronic inflammation, which promotes osteoclast activity, leading to bone breakdown and cortical destruction [31]. At the same time, inflammatory mediators suppress osteoblast function, preventing new bone matrix deposition and impairing callus formation [32]. This dysregulation often culminates where bone regeneration is essentially absent, leaving structurally weakened areas vulnerable to additional damage [33]. Furthermore, bacterial pathogens exacerbate these effects through the production of toxins that cause tissue necrosis and compromise blood flow, limiting the delivery of nutrients and antibiotics to the infection site. The vascular impairment creates dead space within the bone, fostering bacterial proliferation and biofilm maturation, which further entrenches infection [34]. In advanced stages, the interaction between bacterial colonization, biofilm protection, and inflammatory damage erodes the bone's ability to support hardware, increasing mechanical stress and driving implant failure. Understanding the intricate role of the skin microbiome in maintaining immune homeostasis highlights how its disruption can predispose to these orthopedic complications. Dysbiosis not only compromises the protective barrier of the skin but also facilitates the invasion and colonization of opportunistic pathogens. These pathogens exploit disrupted tissue environments to drive infection and biofilm formation, ultimately contributing to the osseous healing complications of non-union, malunion, and hardware failure, adding significant challenges to the management of orthopedic infections.

Mechanisms of Dysbiosis Development

Failure to control the skin microbiome is a well-established contributor to surgical site infections (SSIs) [35]. While preoperative antiseptics are essential for preventing infections, they also disrupt the delicate balance of skin microbiota. Among commonly used antiseptics, chlorhexidine (CHX) stands out for its longer-lasting effects and broad-spectrum antibacterial activity, often making it the preferred choice over povidone-iodine (PVI) in many surgical settings [36]. However, there is growing concern over the potential for microbial resistance to antiseptics, necessitating further investigation into their long-term efficacy. Studies have highlighted that PVI significantly disrupts bacterial communities at surgical sites, reducing commensal organisms such as *S. epidermidis* [37,38]. Although beneficial for sterility, this reduction in microbial diversity raises concerns among clinicians, particularly in orthopedic procedures where maintaining a balanced microbiota is essential to reduce susceptibility to opportunistic pathogens. Recent evidence supports CHX as an effective preoperative antiseptic, with no increased risk of SSIs associated with its use [39]. Understanding the interplay between antiseptic use and microbial diversity remains vital for optimizing infection control strategies.

Prolonged hospital exposure is also a key contributor to microbiome imbalance, largely due to the heightened prevalence of drug-resistant organisms in these settings. The overuse of antibiotics, particularly broad-spectrum agents, significantly reduces the abundance of normal skin flora such as *S. epidermidis* and *C. acnes*, while simultaneously increasing susceptibility to antibiotic-resistant pathogens [40]. These commensal organisms play a crucial role in maintaining microbial balance, and their depletion creates an environment vulnerable to dysbiosis. Nosocomial factors further exacerbate this disruption by exposing patients to opportunistic pathogens, such as *S. aureus* (including methicillin-resistant *S. aureus* [MRSA]) and *P. aeruginosa*, which are frequently encountered in hospitals [41-43]. Moreover, the intrinsic and acquired resistance mechanisms of these organisms further complicate treatment, placing additional strain on infection prevention efforts. These pathogens often colonize immunocompromised patients and those with pre-existing conditions, exploiting the disrupted microbiome to increase the risk of infections [44]. For orthopedic surgery patients requiring inpatient care, the dual burden of prolonged hospital exposure and antibiotic use increases their postoperative risk of developing SSIs, highlighting the need for infection control strategies, especially for immunocompromised patients.

Operating room sterilization protocols and air circulation systems are critical in maintaining microbial equilibrium and reducing SSIs, with advanced High-Efficiency Particulate Air (HEPA) filtration systems demonstrating significant efficacy in removing airborne microorganisms [45]. Unidirectional or laminar airflow systems, particularly those with flow stabilizers, have shown remarkable potential in minimizing intraoperative bacterial contamination by creating a continuous clean airflow over the surgical field [46]. Having a unidirectional current over the surgical field would minimize a patient getting continuously exposed to the same pathogen, decreasing a patient's risk for post-surgical complications and implant rejection. Single large diffuser systems have been empirically proven to outperform multi-diffuser arrays in removing microbes [47]. This highlights the importance of strategic air delivery mechanisms in

maintaining surgical sterility. The complex interplay between air circulation design, sterilization techniques, and microbial control represents a crucial frontier in preventing orthopedic implant-associated infections. Emerging research continues to refine our understanding of these intricate microbiological dynamics, underscoring the need for adaptive and evidence-based approaches to operating room sterilization.

Patient-related factors, such as diabetes and rheumatologic diseases, can significantly influence the microbiome and impact outcomes in orthopedic surgery. Diabetes alters the microbiome and immune response through impaired immune function caused by elevated blood glucose levels, which weaken the body's ability to combat bacteria at surgical sites. Complications, like peripheral vascular disease and peripheral neuropathy, further hinder wound healing and increase infection risk. This can often be seen in foot and ankle surgeries, where pre-existing foot ulcers and neuropathy elevate the likelihood of postoperative infections [48]. Obesity, another critical factor, is associated with increased SSIs in procedures, such as hip and knee arthroplasty, due to poor blood supply to adipose tissue, which delays wound healing [49]. Similarly, autoimmune diseases like rheumatoid arthritis and lupus, heighten the risk of infection after surgery, as the compromised immune system struggles to fight off bacteria [50]. This risk is exacerbated by immunosuppressive medications often used to manage autoimmune conditions. Collectively, these causes and triggers create a challenging surgical environment by altering the microbiome, impairing healing, and increasing susceptibility to infections.

Strategies for Preventing and Managing Microbiome Dysbiosis

Microbiome restoration through probiotics, prebiotics, and bacteriophage therapies represents a promising approach for mitigating dysbiosis. Probiotics, which introduce beneficial microbes, have shown the potential to reduce pathogen colonization by restoring commensal populations. Topical probiotics containing *Lactobacillus* species exhibit antimicrobial activity against *S. aureus* and *P. aeruginosa* while promoting skin barrier function [51,52]. Prebiotics, which provide substrates for beneficial bacteria, enhance microbial diversity and facilitate microbiome recovery. Novel strategies are utilizing nanocarriers to optimize the topical application of probiotics and prebiotics [53]. These nanocarriers, such as nanoparticles or nanoemulsions, increase skin penetration, maximizing efficacy to help establish a protective barrier. In addition, bacteriophage therapies offer a highly targeted method for eradicating biofilm-associated pathogens, while sparing commensal microbes. Phages engineered to target *S. aureus* biofilms have demonstrated efficacy in preclinical models for treating wound infection, suggesting possible utility in microbiome-preserving interventions [54]. These advancements underscore the potential of microbiome restoration therapies as a cornerstone for combating dysbiosis while preserving the integrity of commensal microbial ecosystems.

Biofilms are structured communities of bacteria encased in extracellular polymeric substances, which enhance bacterial survival by protecting against immune defenses and antimicrobial agents. This creates a persistent infection environment, promoting implant failure and nonunion, particularly when complete debridement or hardware removal is not achieved [55]. Nonunion, characterized by the failure of a fractured bone to heal within a typical timeframe, is exacerbated by persistent biofilm infections. This property of bacteria impairs osteogenesis and induces chronic inflammation at the fracture site [56]. To combat the challenges of biofilm-associated infections and their role in surgical failures, orthopedic advancements have focused on both innovative surgical techniques and preventative measures. These efforts have extended to the development of antimicrobial technologies, such as implant coatings and microbiome-preserving skin preparations.

Antimicrobial innovations, including implant coatings and microbiome-preserving skin preparations, are critical in preventing pathogen colonization without disrupting commensal balance. Antimicrobial coatings, such as release-based systems on orthopedic implants have been developed to inhibit antibacterial activity. These implants are coated with antibiotics or antimicrobial peptides that can be released in a sustained manner to maintain high concentrations of antimicrobial substances around the implants [57]. Moreover, specific components, such as polyethylene oxide, can be used on the surface of implants. Polyethylene oxide prevents bacterial attachment and biofilm development, repelling bacterial agents from implant surfaces [58]. This is one of many substances developed in the orthopedic field to prevent biofilm formation on implants, with the goal of reducing SSI and hardware failure. Additionally, microbiome-preserving skin preparations are designed to maintain the delicate balance of the skin's microbial ecosystem, focusing on formulations that minimize disruption to commensal organisms while targeting pathogens. Microbiome-derived ingredients, such as prebiotics and postbiotics, are being explored to support skin homeostasis, by fostering beneficial microbial interactions and minimizing the disruption caused by external factors [59]. These innovations offer a dual benefit, reducing infection risk while preserving the protective functions of the native microbiome.

Other emerging prevention strategies include metagenomic sequencing, a high-resolution tool for analyzing microbial communities that holds promise for developing individualized care plans for patients prone to microbiome dysbiosis. By examining the DNA profiles of all organisms in a sample, this technology enables researchers to assess the microbial composition of a given environment. Early identification of dysbiosis, often seen in inflammatory skin conditions, allows for timely interventions to preserve the natural skin microbiome and reduce the risk of associated disorders. Patterns of dysbiosis linked to inflammatory skin diseases are increasingly associated with functional and strain-level variations identified through metagenomics [60]. Metagenomics can also precisely characterize microbial communities at wound sites. Recent studies have identified specific strains of *S. aureus* that correlate with poor wound healing outcomes [61]. In orthopedic surgery, metagenomic sequencing holds significant potential for developing personalized care plans tailored to patients at risk of SSIs due to skin dysbiosis, potentially reducing SSI rates. Despite some metagenomic sequencing studies demonstrating the stability of skin microbial communities at the strain and species level, even amidst environmental exposures [62], further research is needed to fully explore and validate the role of this technology in personalized care strategies for at-risk individuals within surgical settings.

Clinical Implications and Outcomes

Surgical site infections are a common complication, underscoring the critical importance of integrating microbiome preservation into perioperative protocols. Most infections are attributed to bacteria already present in the patient's microbiome rather than from external sources introduced during surgery [63]. One theory about the cause of perioperative infections is the endogenous translocation of strains can occur after surgery [64]. The Trojan Horse Hypothesis of SSIs states that pathogens from areas such as the gums, teeth, and gastrointestinal tract can be taken up by immune cells, such as neutrophils and macrophages, and can bring the bacteria to the wound site after a procedure [64]. This reveals the importance of also preserving the microbiome in places other than the surgical site, specifically the importance of dental hygiene. Current protocols to prevent infection during a procedure often focus on exogenous sources of bacteria [64]. Preserving the microbiome has been shown to enhance wound healing, reduce risk of infection, and improve long-term outcomes. Minimizing dysbiosis, by maintaining the balance of the patient's own microbiota, reduces the risks of SSIs and further complications [65]. Physicians can minimize the use of unnecessary broad-spectrum antibiotics, by implementing targeted prophylaxis regimes, and incorporating perioperative probiotic supplementation strategies [65]. Young Khadaroo (2014) also reports that by maintaining the microbiome, the risk of biofilm formation is minimized, leading to improved implant longevity.

Challenges and Limitations

There are gaps in understanding the balance between preventative care and microbiome preservation. By giving prophylactic antibiotics before surgery, the healthcare team is risking the collapse of the "healthy" microbiome, which includes commensal bacteria. Any major disruption to the microbiome can lead to the colonization of pathogenic bacteria [66]. A study by Krezaleck (2016) found that mice that were given probiotics to help maintain a healthy microbiome had a faster wound healing time than the control mice who did not receive probiotics. Damage to the microbiota has been shown to stimulate the production of overactive neutrophils, potentially resulting in excessive tissue injury [66]. Even though prophylactic antibiotics can prevent hospital transmission of infection, they can also harm the patient by disrupting their natural flora. There is also limited clinical data on the efficacy of microbiome-targeted interventions. There is little research on virulence factors and how they impact the likelihood of SSIs [63]. With limited research into this topic, clinicians who practice evidence-based medicine, will be less likely to employ these new techniques. Randomized trials that look into the effectiveness of targeted prophylaxis and perioperative probiotic supplementation in reducing the risk of infection need to be continued.

Metagenomics and RNA sequencing can help sequence variants to be monitored. A map of the migration of microbes can be made including the original site to the surgical wound [64]. Correct identification of the affecting bacteria is necessary for appropriate treatment. Bacteria within biofilms are more difficult to identify. Diagnostic methods, such as tissue sampling and culture, have led to false negative results [67]. Misdiagnosis and delayed results can lead to worsening of the infection or incorrect treatment regimes. These advanced diagnostic tools, such as microbial modeling, specifically BioSolve, can provide a high degree of transparency [68]. The cost of modeling technology varies depending on the microbe it is analyzing [68]. Depending on the specific microbiome, the user may wish to use different techniques. This makes it harder to come up with a standardized way for clinicians to find information and implement protocols.

Future Directions

The growing understanding of the role of the skin microbiome in orthopedic implant outcomes opens new avenues for research and clinical innovation. Future studies should aim to explain the precise mechanisms that skin microbiome dysbiosis contributes to SSIs and implant failures. Advanced molecular tools, such as metagenomic sequencing and single-cell RNA sequencing, can help identify the specific microbial strains and gene expression patterns associated with biofilm formation and chronic infections. Additionally, longitudinal studies tracking microbiome dynamics pre- and post-surgery could provide critical insights into how antiseptic protocols, perioperative antibiotics, and environmental factors affect microbial balance. After these initial microbiome studies are conducted, precision medicine approaches should also be explored, leveraging predictive microbial modeling to identify high-risk patients and tailor perioperative protocols accordingly. Incorporating microbiome assessments into preoperative risk evaluations may allow for the implementation of personalized infection prevention strategies, ultimately improving patient outcomes in orthopedic surgery.

Conclusion

The interplay between the skin microbiome and orthopedic surgical outcomes represents a critical, yet underappreciated, frontier in modern medicine. Dysbiosis of the skin microbiome contributes to biofilm formation, chronic infections, and implant failures, underscoring the importance of preserving microbial homeostasis throughout the perioperative period [19, 55]. Emerging strategies, such as microbiome-preserving antiseptics, antimicrobial implant coatings, and advanced therapeutics like bacteriophages and probiotics, offer exciting opportunities to mitigate these risks [9,51,53]. A multidisciplinary approach that integrates microbiome science, material innovation, and clinical expertise is paramount for advancing infection prevention in orthopedic surgery. While challenges remain, including the need for robust diagnostic tools and the translation of novel therapies into widespread clinical use, ongoing research is steadily paving the way for transformative changes in surgical care. By prioritizing microbiome health, clinicians can enhance patient outcomes, reduce healthcare costs, and ensure the long-term success of orthopedic implants.

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