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CIGARETTE SMOKING AS A RISK FACTOR FOR IMPAIRED POSTOPERATIVE WOUND HEALING PROCESS

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ABSTRACT

Background. Proper postoperative wound healing is an essential and ongoing component of effective hospital care. Cigarette smoking might be one of the modifiable risk factors for impaired wound healing, which can lead to prolonged hospitalization and increased healthcare costs.

Objective. This systematic review examined existing literature on the documented impact of cigarette smoking on the incidence and types of postoperative wound-healing complications by comparing smokers with non-smokers and whether preoperative smoking cessation significantly reduces the risk of these complications.

Methods. A structured literature review was conducted using PubMed, Elsevier, ScienceDirect and Scopus were searched between June and August 2026. Studies included systematic reviews, meta-analyses, cohort studies, and review articles published between 2000 and 2025

Results. Smoking cigarettes disrupts the functioning of our body and is associated with numerous disorders and impairs postoperative wound healing. Importantly, these adverse outcomes may be reduced through effective smoking cessation interventions.

Conclusion. Smoking cessation before surgery is an important strategy for reducing postoperative complications, healthcare costs, and mortality. Preferably, smoking should be stopped at least 4–6 weeks before elective surgery and continued for 4–8 weeks postoperatively to maximize the potential benefits. Overall, quitting smoking improves surgical outcomes and can be lifesaving.

KEYWORDS

Wound Healing, Cigarette Smoking, Surgical Wound, Postoperative Care

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Introduction

Skin is the biggest organ in the human body and fulfills many functions. Therefore, its healing requires an exceptional mechanism of cascading cellular functions, which leads to scar formation. Although healing and regeneration processes take place in every part of the human body, this review focuses on the postoperative wound healing processes of the skin and its phases [1]. Wounds have been one of the most prominent pathologies since the beginning of humanity. There are various types that can be classified in several ways, depending on their cause, depth, duration, and healing process, and every one of them requires different care. Surgical wounds are intentionally created under controlled conditions and are generally classified as acute wounds, typically progressing through a predictable sequence of healing. Nevertheless, they remain susceptible to complications such as surgical site infection, wound dehiscence, delayed healing, and abnormal scar formation, and therefore require appropriate postoperative care and monitoring [2]. Over the past few decades, significant improvements in postoperative wound care have been observed, resulting from a broader understanding of the underlying mechanisms and access to more advanced technologies. Surgical wounds are a common aspect of healthcare and often require a multidisciplinary approach to ensure optimal healing. Their healing process might be influenced by numerous factors, including nutritional status, age, genetic predisposition, lifestyle, comorbidities, blood glucose levels, tissue perfusion, alcohol consumption, and cigarette smoking. [3] Therefore, postoperative wound management should be carefully coordinated and individualized to each patient, as inadequate care and control of contributing factors may impair healing and ultimately lead to the development of chronic, non-healing wounds. Chronic wounds represent a significant healthcare challenge, often resulting in substantial consequences for patients while imposing a considerable socioeconomic burden on healthcare systems and society due to their high prevalence, prolonged healing time, and frequent recurrence [3,4]. Resulting in poor health-related quality of life and often preventing them from

returning to work. They are also frequently associated with prolonged hospital stays, which can increase healthcare costs and place additional demands on healthcare resources. Further, it exposes patients to the risk of surgical site infection and furthermore sepsis. Despite advances in our understanding of risk factors and the development of sophisticated wound-treatment strategies, achieving optimal wound healing remains a significant clinical challenge, particularly in the context of the increasing prevalence of metabolic disorders among patients. Therefore, there is currently a need for effective strategies and education focused not only on treatment but also on the prevention of non-healing chronic postoperative wounds. It is important to facilitate faster recovery of functional capacity while reducing the healthcare costs associated with prolonged treatment. Therefore, it is essential to identify, recognize, and proactively address modifiable risk factors for impaired wound healing, particularly cigarette smoking, through appropriate preventive and therapeutic interventions [5].

The Aim of The Study

The aim of this study is to analyze the literature regarding the documented impact of cigarette smoking on the incidence and types of postoperative wound healing complications comparing smokers with non-smokers and to assess whether smoking cessation prior to surgery significantly reduces the risk of postoperative complications.

Methodology

A structured literature review was conducted to identify and evaluate the available evidence concerning the impact of cigarette smoking on postoperative wound healing and associated complications. The databases PubMed, Elsevier, ScienceDirect, and Scopus were systematically searched between June and August 2026. The search included focused on studies published between 2000 and 2025, focusing particularly on those published up to ten years ago allowing for the inclusion of both established evidence and more recent findings. Eligible publications included systematic reviews, meta-analyses, cohort studies, and narrative or comprehensive review articles addressing the relationship between cigarette smoking, smoking cessation, wound healing process, and postoperative complications. Relevant articles were assessed for their applicability to the research question, with particular attention given to evidence comparing smokers with nonsmokers and evaluating the potential benefits of preoperative smoking cessation. Studies that were published before the year 2000 or did not directly address smoking, postoperative wound healing, or surgical outcomes were excluded.

Results

The wound-healing process

Skin consists of three primary layers the epidermis, dermis, and hypodermis; which play essential roles in maintaining tissue integrity and responding to injury. Wound healing is a dynamic and systemic process that occurs through a series of overlapping phases, namely hemostasis, inflammation, proliferation, and tissue remodeling [6,7,8]. Successful healing depends on the coordinated interaction of numerous mediators, including growth factors, cytokines, and chemokines, which regulate the cellular processes involved in tissue repair. The hemostatic phase is the first stage of acute wound healing and begins immediately following tissue injury, including surgical incision. This phase generally occurs within the first few hours and involves the formation of a provisional wound matrix. Vasoconstriction is the initial response to surgical injury and helps limit blood loss by reducing blood flow to the affected area. Blood subsequently enters the wound and forms a clot that helps approximate and stabilize the wound's edges. During this process, fibrinogen is converted into interconnected fibrin strands, forming a meshwork that provides structural support for subsequent cellular activity. As the clot dries at the wound surface, it contributes to the formation of a scab, which provides temporary protection against external contamination and helps maintain the local wound environment. The fibrin matrix also provides a temporary scaffold through which cells can migrate during subsequent stages of healing and ultimately contributes to scar formation. In addition to control bleeding, the hemostatic phase initiates the inflammatory phase of wound healing. Inflammation begins during coagulation and can be broadly divided into an early phase characterized predominantly by the recruitment of neutrophils and a later phase involving the recruitment and differentiation of monocytes into macrophages [1]. The inflammatory phase plays a crucial role in wound healing by delivering essential nutrients and immune components to the site of

injury, removing bacteria and cellular debris, and initiating signals that promote tissue repair. Immediately following tissue injury, a series of vascular and cellular changes occurs in the tissues surrounding the wound. Vasodilation of local blood vessels increases vascular permeability, allowing plasma and its components—including enzymes, proteins, antibodies, and complement factors to enter the surrounding tissue. This inflammatory response facilitates the recruitment of nutrients, antibodies, chemical mediators such as histamine, serotonin, kinins, prostaglandins, and proteolytic enzymes, as well as inflammatory cells to the surgical site. These mediators promote the recruitment and activation of polymorphonuclear leukocytes (neutrophils), which contribute to the early stages of wound healing. The resulting inflammatory exudate, which is present to varying degrees in all wounds, contains leukocytes, necrotic tissue, and cellular debris. Wound debridement is initiated primarily through the activity of neutrophils. As these cells undergo degeneration, they release enzyme-containing granules that help break down and remove extracellular debris and microorganisms. In relatively clean and uncomplicated wounds, neutrophils are subsequently replaced by monocytes, which migrate into the wound and differentiate into macrophages. Macrophages are essential to both wound debridement and tissue repair. They facilitate the removal of cellular debris through enhanced lysosomal enzyme activity and the secretion of proteolytic enzymes. In addition, macrophages produce inflammatory mediators, including interferons and prostaglandins, and release chemotactic factors that attract mesenchymal cells to the wound site. These factors also contribute to the differentiation of mesenchymal cells into fibroblasts, thereby linking the inflammatory phase to the subsequent proliferative phase of wound healing. The proliferative phase of wound healing begins shortly after surgery and involves several coordinated processes, including epithelialization, fibroplasia, angiogenesis, and the formation of granulation tissue. The primary objective of this phase is to restore the damaged wound surface, replace lost tissue, and re-establish an adequate vascular network to support continued repair. The earliest visible manifestations of tissue repair are the migration and proliferation of epithelial cells. Following injury, basal keratinocytes detach from their connections with the underlying dermis and migrate toward areas of tissue loss. These cells subsequently proliferate through mitosis and differentiate to restore the integrity, structure, and function of the epidermis. At the same time, fibroplasia is becoming increasingly prominent. Fibroblasts begin to migrate into the wound, appearing approximately 48 hours after surgery, and using the fibrin matrix formed during the earlier hemostatic phase as a temporary scaffold to guide their movement. Once within the wound, fibroblasts synthesize ground substance and extracellular matrix components, particularly collagen, which are essential for the development of granulation tissue and provide structural support for the healing tissue. Concurrently, angiogenesis and neovascularization occur as new capillaries sprout from pre-existing blood vessels at the wound margins. These newly formed vessels extend into the wound and establish interconnected vascular networks, supplying the developing tissue with oxygen and nutrients required for continued repair. As the proliferative phase progresses, the formation of granulation tissue is followed by cellular apoptosis, which helps regulate cellularity and contributes to the transition from active tissue proliferation toward wound maturation. The inflammatory and proliferative processes gradually subside, and the newly formed tissue undergoes further remodeling. Ultimately, wound healing results in the formation of a mature scar, which is characterized by reduced cellularity and vascularity compared with normal tissue. The extent and characteristics of scar formation are influenced, in part, by the intensity and duration of the inflammatory response throughout the healing process [1].

Smoking and Postoperative Wound Healing Complications

Smoking has long been recognized as a significant factor that adversely affects wound healing and numerous other biological processes. It is also considered to be associated with the development of several major diseases, including cancer, atherosclerosis, chronic obstructive pulmonary disease, and degenerative disorders [9]. Given the complex molecular and cellular mechanisms involved in tissue repair, it is therefore unsurprising that smoking can negatively influence many of the processes required for effective wound healing [10]. The association between smoking and delayed postoperative wound healing has been well established in numerous studies and demonstrated across a broad range of surgical specialties and procedures [11,12]. Smoking can impair postoperative wound healing through multiple mechanisms. Nicotine induces vasoconstriction, which reduces local blood flow, while carbon monoxide binds competitively to hemoglobin, thereby decreasing the oxygen-carrying capacity of the blood. Consequently, tissue perfusion and oxygenation are compromised, both of which are essential for a proper wound repair. Furthermore, smoking has been identified as an independent risk factor for deep incisional surgical-site infection, wound dehiscence, and the

need for reoperation [5]. Cigarette smoke contains more than 5,000 chemical constituents, including over 250 substances known to be toxic or harmful. Among these, nicotine, carbon monoxide, hydrogen cyanide, and nitric oxide have been particularly implicated in impaired wound healing [12,13]. These compounds can interfere with tissue repair through several interconnected mechanisms, including increased oxidative stress, endothelial dysfunction, thrombogenesis, reduced tissue perfusion, and impaired oxygen delivery. Therefore, the detrimental effects of smoking on postoperative outcomes are complex and cannot be attributed to a single substance or mechanism. Instead, they result from the combined effects of multiple components of cigarette smoke acting through several biological pathways. Overall, smoking can interfere with the wound-healing process at at least three distinct levels:

1. Limited tissue perfusion by vasoconstriction and endothelial dysfunction.
2. Inhibition of new tissue formation with evidence of reduced epidermal regeneration and neovascularization.
3. The initial recruitment of inflammatory cells is inhibited with evidence of reduced neutrophil chemotaxis and migration as well as reduced phagocytic activity of macrophages *in vitro*.

Peripheral vasoconstriction and endothelial dysfunction are considered among the most important mechanisms by which nicotine negatively affects wound healing. These effects can substantially reduce tissue perfusion and occur through both direct and indirect pathways. Directly, nicotine impairs endothelium-dependent arteriolar dilation, an effect that may be mediated by increased production and release of oxygen-derived free radicals [14]. Indirectly, nicotine promotes vasoconstriction by stimulating the release of catecholamines from the adrenal medulla, sympathetic ganglia, and cardiac chromaffin tissue, as well as by enhancing the activity of thromboxane A₂, a potent vasoconstrictive mediator. Furthermore, nicotine may disrupt the physiological balance between thromboxane A₂ and prostacyclin in favor of thromboxane A₂. This imbalance promotes platelet adhesion and aggregation, increasing the risk of thrombotic occlusion within the microvasculature. Consequently, microvascular perfusion and the delivery of oxygen and essential nutrients to the healing tissue may be further compromised. In severe cases, these combined effects on tissue perfusion and oxygenation may contribute to tissue ischemia and necrosis at the surgical site [12].

At the soft-tissue level, smoking has been consistently associated with disruption of several stages of the wound-healing process. The early stages of tissue repair rely on the coordinated activity of fibroblasts, growth factors, and an adequate capillary blood supply. Fibroblasts are essential for granulation of tissue formation, responding to growth factors released by macrophages, and producing key extracellular matrix components such as fibronectin, collagen, and elastin. Nicotine, a major constituent of cigarette smoke, has been shown to inhibit fibroblast proliferation and impair their migration into the wound bed. These effects may compromise granulation of tissue formation and subsequently contribute to delayed wound healing [9,15,16]. On the other hand, exposure to nonlethal levels of firsthand cigarette smoke might activate fibroblast survival pathways, prolong cellular lifespan, and promote fibroblast accumulation within tissues. This increased fibroblast activity may lead to excessive deposition of connective tissue, potentially contributing to abnormal tissue repair and organ fibrosis. In contrast, in open wounds, cigarette smoke exposure may impair the migration of fibroblasts into the wound bed, thereby delaying wound closure and potentially affecting normal scar formation. In response to cellular injury or environmental stress, cells activate stress-response pathways and increase the production of stress-response proteins as a protective mechanism to promote survival. However, prolonged exposure to cigarette smoke may result in persistent or excessive activation of these pathways and sustained production of stress-response proteins. Such chronic cellular stress may have detrimental effects and has been associated with the development and progression of pathological conditions, including cancer and tissue fibrosis [9,15].

Cigarette smoke can disrupt immunological homeostasis and contribute to the development of various diseases by exerting complex and sometimes opposing effects on both innate and adaptive immune responses. It may either enhance pathological immune reactions or suppress the normal protective functions of the immune system. Consequently, smoking can reduce host defense against infections while simultaneously promoting autoimmune responses. These alterations may adversely affect the inflammatory phase of wound healing by reducing the chemotactic recruitment of inflammatory cells, impairing their migration, and compromising their oxidative bactericidal activity [10]. The magnitude and severity of these effects may vary according to the complex composition of cigarette smoke and the individual's overall health status. Notably, the effects of smoking on innate immune responses appear to diminish relatively rapidly following smoking cessation, whereas alterations in adaptive immunity may persist for a considerably longer period, potentially

as a result of smoking-induced epigenetic changes [17]. Nevertheless, whether smoking is active or has produced persistent immune alterations, its overall effect on immune function is considered detrimental rather than beneficial. Prolonged impairment of host immunity and persistent infection may potentially contribute to cross-reactive autoimmune responses involving both pathogens and host tissues; however, the precise mechanisms underlying smoking-associated immune dysregulation remain incompletely understood and require further investigation [18]. These alterations in immune function have direct implications for the body's ability to protect the surgical wound from microbial contamination. In particular, impaired phagocytic and bactericidal activity of neutrophils and monocyte–macrophage cells compromise early host defense, reducing the ability to effectively control bacterial contamination at the wound site and consequently increasing the risk of postoperative surgical-site infection [10].

Infection is one of the major threats to successful wound healing and may lead to a range of complications, including suture disruption, abscess and fistula formation, tissue necrosis, cellulitis, and sepsis, which can progress to life-threatening systemic shock. Consequently, effective prevention and management of wound infection are essential components of postoperative care. This includes a thorough assessment of the patient's medical history and risk factors, appropriate perioperative antibiotic prophylaxis, strict adherence to aseptic techniques, removal of necrotic tissue, effective management of wound exudate, and reduction of bacterial colonization [19]. A bacterial burden exceeding 10^5 microorganisms per gram of tissue has traditionally been associated with an increased risk of infection and delayed wound healing. Therefore, an important objective of wound management is to maintain the bacterial load below this critical threshold through appropriate wound preparation, including effective management of dead space and the application of suitable, atraumatic dressings. The clinical and economic consequences of wound infection are particularly evident in surgical site infections (SSIs), which represent a major postoperative complication. SSIs are associated with substantial morbidity and have been reported to contribute, at least in part, to more than one-third of postoperative deaths. Their clinical presentation and severity can vary considerably, ranging from relatively minor wound discharge without further complications to severe, potentially life-threatening infections. In addition to acute complications, SSIs may result in poor cosmetic outcomes, including spreading, hypertrophic, or keloid scarring, as well as persistent pain and itching. They may also restrict mobility and can have a considerable negative impact on patients' emotional well-being. Beyond their clinical consequences, SSIs place a significant burden on healthcare systems. As previously discussed, they can substantially prolong hospitalizations, in some cases doubling the duration of hospital stay [19].

Interestingly, smoking may have an opposing effect on wound contraction. Under normal circumstances, wound contraction begins during the inflammatory phase and progresses concurrently with other repair processes. Studies have demonstrated that incisional wounds in smokers may have a smaller diameter approximately seven days after injury compared with those in never-smokers, suggesting enhanced wound contraction [20]. One possible explanation is increased activity of myofibroblasts, which are responsible for wound contraction. In smokers, increased levels of fibrinogen and fibronectin within the early wound environment may promote the transformation of fibroblasts into contractile myofibroblasts, potentially enhancing wound contraction. However, the precise mechanisms remain unclear, and it is uncertain whether enhanced contraction provides any meaningful benefit when compared with the numerous detrimental effects of smoking on wound healing. Thus, although wound contraction may be increased in smokers, potentially as a result of altered myofibroblast activity, its overall influence on postoperative complications remains uncertain [10,15]. Beyond its effects on inflammation and wound contraction, smoking also interferes with the subsequent proliferative and remodeling phases of tissue repair. Smoking adversely affects these phases by impairing epidermal regeneration and neovascularization. Reduced fibroblast activity, together with limited availability of substrates required for collagen production, results in decreased collagen synthesis and deposition of mature collagen. These alterations may contribute to impaired tissue strength and remodeling and help explain the higher incidence of delayed wound healing, wound dehiscence, and incisional hernia observed among smokers compared with nonsmokers across a range of surgical procedures [10,15,21].

Smoking is also associated with several chronic conditions that may further increase the risk of adverse surgical outcomes, particularly respiratory and vascular diseases. Cigarette smoke contains numerous harmful and carcinogenic substances, which may contribute significantly to perioperative morbidity. Cigarette smoking also increases vulnerability to respiratory infections by reducing the effectiveness of airway cilia responsible

for clearing pathogens. It has been causally linked to a higher risk of pneumonia and is also associated with an increased likelihood of *Mycobacterium tuberculosis* infection, progression to active tuberculosis, and tuberculosis-related mortality [22]. In addition, smoking can cause narrowing of the bronchioles, making them more susceptible to airway collapse. These changes can compromise respiratory function and increase the risk of postoperative pulmonary complications and respiratory infections [9].

In addition to its effects on wound healing, cigarette smoking might influence the severity and perception of perioperative pain. Smokers have been reported to experience greater postoperative pain, as reflected by higher pain scores and increased requirements for opioid analgesics. Recognizing the potential for increased postoperative pain in patients who smoke may allow for more effective pain management and contribute to improved postoperative recovery and outcomes [24].

Moreover, active smoking may also influence the decision to proceed with surgical intervention. In cases where the anticipated risks are particularly high, elective surgery may be postponed or, in extreme circumstances, not offered to patients who continue to smoke because of their increased risk of adverse outcomes [23]. Therefore, smoking status may be an important consideration when assessing a patient's suitability for surgery and determining the appropriate timing of the procedure.

Finally, smoking may also contribute to prolonged hospitalization, which is as previously mentioned associated with additional clinical and socioeconomic consequences. Extended hospital stays increase the risk of healthcare-associated infections and may contribute to complications such as muscle wasting, pressure ulcers, venous thrombosis, and deterioration in mental and functional status, particularly among older patients. Prolonged hospitalization may also reduce patients' independence and delay their return to normal activities. In addition, longer hospital stays increase treatment-related costs and place an additional burden on healthcare resources.

Factors affecting wound healing:

Age in wound healing

The capacity for wound healing generally declines with advancing age due to several physiological changes, including reduced collagen density, decreased fibroblast numbers, increased fragmentation of elastin, and slower wound contraction [8]. However, delayed healing and the development of chronic wounds among elderly are often more strongly associated with the presence of comorbidities, which become increasingly prevalent with age. Current evidence suggests that, even in otherwise healthy older individuals, cutaneous wound healing may be impaired compared with younger patients, although on the other hand scar maturation may be more favorable. Nevertheless, advanced age remains an independent risk factor for reduced rates of chronic wound closure [25].

Type and extent of surgery

The impact of smoking on perioperative outcomes varies depending on the type and extent of surgery. Both current smoking and to a lesser extent a history of smoking is associated with poorer treatment outcomes following all procedures, but particularly after cardiac and oncological procedures. Therefore, especially those patients who have been qualified for major surgical procedures should be encouraged to discontinue tobacco use to optimize perioperative and overall procedural outcomes [26]. The extent of the surgical procedure appears to influence the risk of smoking-related wound-healing complications, with more extensive surgeries being associated with a greater likelihood of impaired wound healing and postoperative complications among smokers. Therefore, patients undergoing major surgical procedures should maintain smoking abstinence for a longer period than those undergoing minor surgery [27].

Immune function in wound healing

Whether alterations to immune function are intrinsic or extrinsic, their effect on the inflammatory phase of healing leads to poor wound healing. Immunosuppressed individuals are less capable of fighting infection but also lack proper functioning or quantity of inflammatory cells to properly follow the inflammatory phase. If improper cellular debridement of the wound occurs via these cells, wound healing is delayed and ultimately leads to more scarring [11].

Obesity

Obesity is associated with several complications that can adversely affect wound healing, including surgical-site infection, wound dehiscence, hematoma and seroma formation, as well as pressure and venous ulceration. The increased risk of wound infection among individuals with obesity is well established. The relatively poor vascularity of adipose tissue can result in local tissue hypoxia, which impairs neutrophil function and bacterial clearance and may also reduce the delivery of antibiotics to the wound. In addition, wound dehiscence and the formation of hematomas and seromas are largely attributed to mechanical factors, as increased tension on the wound and the greater difficulty of adequately eliminating dead space can compromise wound integrity. These factors may further increase the susceptibility of the wound to infection [11, 28, 29]. If such patients also smoke cigarettes, the risk of infection is further increased due to the combined impairment of immune system function caused by both excess adipose tissue and cigarette smoke.

Diabetes and wound healing

Impaired wound healing is estimated to affect approximately 25% of individuals with diabetes mellitus [30]. Several factors contribute to the impaired healing of diabetic wounds. Persistent hyperglycemia promotes the formation of biofilms, making these wounds more difficult to manage and treat effectively. In addition, reduced tissue perfusion resulting from accelerated atherosclerosis and diabetes-associated neuropathy increases susceptibility to infection. When combined with impaired immune function, these factors create an environment conducive to prolonged and abnormal wound healing. Patients with diabetes also tend to develop wounds with reduced tensile strength, increasing the risk of wound dehiscence. This may be attributed to impaired collagen synthesis and accumulation, as well as reduced angiogenesis, all of which compromise the structural integrity of the healing tissue. Clinically, appropriate prevention and management of diabetes, together with careful and comprehensive wound care, are essential for improving healing outcomes [30]. Given the increasing prevalence of diabetes and the corresponding rise in diabetic wounds, future therapeutic strategies should address the multiple underlying mechanisms responsible for impaired healing in this patient population [31].

Oxygen delivery in wound healing

Chronic wounds typically exhibit reduced oxygen availability, and tissue hypoxia is closely associated with deterioration of wound condition [32]. This is particularly important because adequate oxygenation is essential for multiple stages of the wound-healing process. Oxygen plays a critical role in inflammation, bactericidal activity, angiogenesis, epithelialization, and collagen deposition. Consequently, adequate tissue perfusion and oxygenation are fundamental requirements for successful wound healing and represent two of the most common factors contributing to impaired or delayed healing. Collagenases function most effectively at oxygen tensions between approximately 20 and 200 mm Hg, whereas oxygen tension in human surgical incisions typically averages only 30–40 mm Hg. Therefore, any further reduction in oxygen delivery, such as that caused by vasoconstriction or hypovolemia—often cigarette smoke impairment, may compromise cellular function and contribute to abnormalities or delays in the wound-healing process [11].

Nutrition in wound healing

Providing all necessary nutrients is essential for the proper healing of postoperative wounds. Understanding the specific roles of individual nutrients in tissue repair therefore provides an important basis for nutritional assessment and therapeutic intervention. It should form an integral part of both preoperative preparation and postoperative care, as it may help identify nutritional deficiencies and reduce the risk of impaired or delayed wound healing. Although, deficiencies in any nutrient may negatively affect the healing process, protein plays a particularly important role due to its essential contribution to collagen synthesis and tissue repair. Protein deficiency can impair fibroblast proliferation and the formation of new capillaries, thereby compromising wound healing. Similarly, deficiencies in vitamins and other micronutrients—often observed among smokers may adversely affect collagen production, epithelialization, wound closure, and capillary integrity, potentially increasing tissue fragility and susceptibility to infection. Therefore, maintaining adequate nutritional status and addressing existing deficiencies are essential components of postoperative care and may help minimize the adverse effects of malnutrition on wound healing [1].

Smoking cessation and Wound Healing

Patients who smoke cigarettes may benefit from a period of smoking cessation before and after surgery, as this may reduce the risk of smoking-related postoperative complications. For elective procedures, cessation for at least 4 to 6 weeks before surgery, followed by continued abstinence for an additional 4 to 8 weeks postoperatively, has been suggested to substantially reduce the incidence of wound-related complications, with reductions of up to 50% reported by the American College of Surgeons [8]. Preoperative counselling is therefore an important responsibility of healthcare professionals, who should inform patients about the potential adverse effects of smoking on surgical outcomes and the benefits associated with cessation [9]. Approaches to tobacco dependence treatment have continued to evolve, with advances in pharmacological therapies and behavioral interventions. Current evidence-based strategies increasingly emphasize individualized treatment and the use of different technologies and support systems to facilitate smoking cessation. Available interventions include pharmacotherapy, healthcare-provider advice, individual counselling, structured group programs, online resources, telephone quitlines, social media-based support, and incentive-based interventions. A combination of pharmacological and behavioral approaches is generally recommended to improve the likelihood of successful cessation [33]. Despite the availability of these interventions, many smokers continue to attempt cessation without adequate preparation, or professional support. This unassisted approach is associated with high rates of relapse and failure, with unsuccessful cessation attempts reported in approximately 95–98% of cases [33]. In addition, the cost of treatment and limited access to specialized cessation services remain important barriers to successful smoking cessation. Consequently, systematic identification of patients who smoke and the implementation of effective perioperative cessation programs may provide benefits extending beyond improved short-term surgical outcomes and reduced healthcare costs, potentially contributing to better long-term health outcomes as well [34].

Comparison of smokers and non-smokers

Sorensen et al. studied 78 smokers to evaluate the effects of smoking cessation on wound healing. They found that wound infection occurred more frequently among individuals who continued smoking than among those who remained abstinent. Notably, four weeks of preoperative smoking cessation substantially reduced infection rates, although it did not significantly influence wound dehiscence. The absence of adverse outcomes among participants using nicotine patches further suggested that components of cigarette smoke other than nicotine may contribute substantially to impaired wound healing. These findings support the benefits of smoking cessation before surgery in reducing postoperative wound complications [8,35]. In another study, Sorensen and colleagues also demonstrated that neither smoking nor abstinence affected the process of neovascularization. Smokers exhibited an attenuated inflammatory response within the wound and reduced fibroblast proliferation, indicating delayed healing. Smoking cessation restores the inflammatory response but does not affect proliferation [36]. These findings suggest a pathophysiological mechanism underlying postoperative infections and wound dehiscence in smokers; this may explain why quitting smoking appears to reduce the risk of wound infection but not the risk of dehiscence [9,35]. Sorensen et al. also found that temporary smoking cessation did not fully restore fibroblast cellularity or collagen synthesis to the levels observed in never-smokers. In some cases, these parameters decreased further after cessation, although the effect was less pronounced in participants using transdermal nicotine patches, suggesting that nicotine may have a stimulatory effect on fibroblast activity and collagen synthesis. Although cessation increased the inflammatory response and macrophage presence in wounds, it did not improve fibroblast function or collagen I synthesis. Vitamin C deficiency often associated with long-term cigarette smoking may also contribute to impaired fibroblast function and collagen production, as it is essential for collagen synthesis. Overall, the findings suggest that smoking and smoking cessation may affect the inflammatory and proliferative phases of wound healing through different mechanisms [37].

Comparisons between light smokers (0–10 pack-years) and heavier smokers revealed no significant differences in the rates of superficial or deep incisional surgical-site infections or wound dehiscence according to cumulative smoking exposure. Although unexpected, these findings suggest that smoking may increase the baseline risk of postoperative wound complications, while the extent of smoking exposure may not further amplify this risk. However, further research and longer-term observation might be needed to confirm this hypothesis [5].

Toyoda and colleagues studied that smokers overall had a higher rate of reoperation within 30 days than nonsmokers, and those with ≥ 21 pack-years had more reoperations than light smokers. This increased risk persisted even after adjusting for differences in patient characteristics, suggesting that smoking itself may contribute to postoperative complications. The higher reoperation rate may partly reflect smoking-related wound complications, including deep surgical-site infections and wound dehiscence. However, the incidence of wound complications did not differ significantly according to cumulative smoking exposure. Interestingly, patients with ≥ 31 pack-years had a higher rate of bleeding requiring transfusion, although the underlying mechanism remains unclear [5].

Discussion

Cigarette smoking is one of the leading preventable causes of postoperative morbidity and mortality, with its effects involving multiple organ systems and potentially compromising postoperative recovery. Therefore, a thorough understanding of normal wound healing is essential for healthcare professionals involved in both preoperative and postoperative patient care. Effective perioperative management begins with an appropriate medical history and assessment to identify chronic diseases, smoking and other addictions, and additional risk factors that may interfere with wound healing, as well as to optimize and stabilize the patient's condition before surgery [9]. During the procedure, strict adherence to principles of sterility, antisepsis, and asepsis, together with appropriate prophylactic treatment, is essential for minimizing postoperative complications. Following surgery, effective wound management should focus on maintaining an appropriate moist environment with suitable dressings, protecting the wound from further injury, and ensuring adequate nutritional support to promote optimal healing. Sriram et al report that providing a regular, palatable diet during the immediate postoperative period is essential for achieving adequate protein and energy intake, which supports the wound-healing process. Thus, ensuring that nutritional requirements are met during this period is consistent with best clinical practices and may contribute to optimal postoperative recovery [38]. Within this comprehensive approach to perioperative care, smoking cessation represents an important and potentially modifiable intervention. Patients who smoke may benefit substantially from a period of smoking cessation before and after surgery, as this may help reduce some of the adverse outcomes associated with tobacco use. Physicians therefore have an important role in educating patients before surgery about the potential detrimental effects of smoking and the benefits of cessation [9]. Moreover, a recent literature review by Rice and Stead [18] demonstrated the benefits of nurse-led preoperative smoking cessation counseling for surgical patients. Identifying and addressing smoking as a modifiable risk factor also contributes to preoperative optimization and may help prevent postoperative complications [21]. Furthermore, smoking cessation may provide benefits not only for patients but also for healthcare systems by reducing the costs associated with surgical care. In addition to lowering postoperative complication rates, successful cessation has been associated with reductions in reoperation, readmission, and mortality, thereby potentially reducing the overall healthcare burden of surgical treatment [34,39].

Preoperative smoking has been associated with an increased risk of various postoperative complications, including overall morbidity, wound healing complications, infections, pulmonary and neurological complications, and intensive care unit admission [40]. Despite generally being younger and having fewer underlying health conditions, current smokers experience a higher incidence of perioperative complications, particularly those affecting the respiratory system. Hawn et al. were among the first to demonstrate the adverse effects of tobacco smoking on surgical outcomes in a large cohort of patients undergoing noncardiac surgery. Their findings showed that patients who regularly smoked before surgery were nearly twice as likely to experience adverse events, including respiratory complications and mortality [34]. These findings were subsequently supported by Musallam et al. [41], who also reported that former smokers had a lower risk of cardiopulmonary complications and death. These and other studies have used large datasets to evaluate the overall relationship between smoking and surgical outcomes [42]. However, evidence suggests that the impact of smoking varies according to the type of surgical procedure and may depend largely on the interaction between smoking-related comorbidities and the postoperative recovery associated with a particular intervention.

Shimd et al. studied the effects of smoking status across 16 major cancer and noncancer procedures, including orthopedic and cardiovascular surgeries. Their findings confirmed the detrimental effects of active smoking on outcomes following major surgery. After adjustment for relevant factors, current smokers had a higher risk of cardiovascular, pulmonary, neurological, inflammatory complications, including sepsis, as well as wound-related complications, consistent with previous research [34,41,42]. In contrast, smoking was not associated with an increased need for perioperative blood transfusion, with the highest transfusion rates observed among nonsmokers. This finding may be related to the higher hematocrit levels typically observed in active smokers, which may represent a compensatory response to impaired tissue oxygen delivery [43]. Importantly, the study also demonstrated that the effect of smoking was procedure-dependent. Smoking appeared to have a more limited influence on adverse outcomes following orthopedic surgery, with a significant association observed only between active smoking and wound complications in patients undergoing knee replacement, consistent with previous studies [26, 43].

Sorensen et al. [9] reported that a minimum of four weeks of smoking cessation was required to reduce oxidative stress and circulating neutrophil counts, while also improving the adverse effects of smoking on neutrophil and macrophage function and their oxidative bactericidal activity. However, prior to major surgeries, smoking cessation for at least one year appears to effectively eliminate the increased risk of postoperative mortality associated with current smoking and substantially reduce the risk of arterial and respiratory complications [41]. These findings highlight the importance of smoking cessation interventions before major surgical procedures [41]. Giving up smoking for approximately four weeks also restores the disrupted tissue environment, regenerative cells, and inflammatory functions; however, proliferative activity might still remain impaired. Conversely, nicotine replacement therapy may offer limited anti-inflammatory and proliferative benefits, because nicotine appears to have an inhibitory effect on wound inflammation and a stimulatory effect on wound proliferation [10]. Our findings demonstrate disturbed wound healing in smokers, a limited effect of abstinence from smoking, and a complex effect of nicotine suggesting a set of pathophysiologic mechanisms, which may explain postoperative wound healing complications in smokers and abstinent smokers. [36].

Finally, active smoking may influence the operator's decision to proceed with surgery due to the increased risk of numerous probable postoperative complications. In some cases, particularly when the anticipated risks are substantial, elective surgery may be deferred or not offered at all to patients who continue to smoke [23]. Declining to perform a surgical procedure is dictated by the patient's well-being and protects them from potential postoperative complications. On the other hand, however, postponing surgery may also carry risks, including disease progression and the persistence or worsening of symptoms associated with delayed surgical treatment. Consequently, smoking status may be an important consideration when assessing a patient's suitability for surgical intervention and determining the optimal timing of the procedure.

Conclusions

Smoking is a leading preventable cause of morbidity and mortality after surgery.

It is most optimal to wait at least 4-6 weeks after smoking cessation before elective surgical interventions and an additional 4-8 weeks postoperatively.

Smoking cessation interventions may help reduce both the frequency and healthcare costs associated with these complications.

Quitting smoking saves lives and prevents potential surgical complications.

Conflicts of Interest: No conflicts of interest to declare

REFERENCES

1. Reinke JM, Sorg H. Wound repair and regeneration. *Eur Surg Res.* 2012;49(1):35-43. doi: 10.1159/000339613. Epub 2012 Jul 11. PMID: 22797712.
2. Teot L, Ohura N. Challenges and Management in Wound Care. *Plast Reconstr Surg.* 2021 Jan 1;147(1S-1):9S-15S. doi: 10.1097/PRS.0000000000007628. PMID: 33347058.
3. Ghaly P, Iliopoulos J, Ahmad M. The role of nutrition in wound healing: an overview. *Br J Nurs.* 2021 Mar 11;30(5):S38-S42. doi: 10.12968/bjon.2021.30.5.S38. PMID: 33733851.
4. Olsson M, Järbrink K, Divakar U, Bajpai R, Upton Z, Schmidtchen A, Car J. The humanistic and economic burden of chronic wounds: A systematic review. *Wound Repair Regen.* 2019 Jan;27(1):114-125. doi: 10.1111/wrr.12683. Epub 2018 Dec 2. PMID: 30362646.
5. Toyoda Y, Fu RH, Li L, Otterburn DM, Rohde CH. Smoking as an Independent Risk Factor for Postoperative Complications in Plastic Surgical Procedures: A Propensity Score-Matched Analysis of 36,454 Patients from the NSQIP Database from 2005 to 2014. *Plast Reconstr Surg.* 2018 Jan;141(1):226-236. doi: 10.1097/PRS.0000000000003963. PMID: 29280887.
6. Barrientos S, Stojadinovic O, Golinko MS, Brem H, Tomic-Canic M. Growth factors and cytokines in wound healing. *Wound Repair Regen.* 2008 Sep-Oct;16(5):585-601. doi: 10.1111/j.1524-475X.2008.00410.x. PMID: 19128254.
7. Phillips SJ. Physiology of wound healing and surgical wound care. *ASAIO J.* 2000 Nov-Dec;46(6):S2-5. doi: 10.1097/00002480-200011000-00029. PMID: 11110286.
8. Gantwerker EA, Hom DB. Skin: histology and physiology of wound healing. *Facial Plast Surg Clin North Am.* 2011 Aug;19(3):441-53. doi: 10.1016/j.fsc.2011.06.009. PMID: 21856533.
9. Tarakji B, Cil A, Butin RE, Bernhardt M. Adverse Effects of Smoking on Musculoskeletal Health. *Mo Med.* 2017 Jul-Aug;114(4):268-271. PMID: 30228609; PMCID: PMC6140077.
10. Sørensen LT. Wound healing and infection in surgery: the pathophysiological impact of smoking, smoking cessation, and nicotine replacement therapy: a systematic review. *Ann Surg.* 2012 Jun;255(6):1069-79. doi: 10.1097/SLA.0b013e31824f632d. PMID: 22566015.
11. Beyene RT, Derryberry SL Jr, Barbul A. The Effect of Comorbidities on Wound Healing. *Surg Clin North Am.* 2020 Aug;100(4):695-705. doi: 10.1016/j.suc.2020.05.002. Epub 2020 Jun 17. PMID: 32681870.
12. Rinker B. The evils of nicotine: an evidence-based guide to smoking and plastic surgery. *Ann Plast Surg.* 2013 May;70(5):599-605. doi: 10.1097/SAP.0b013e3182764fcd. PMID: 23542839.
13. Kim Y, Chen TC. Smoking and Nicotine Effects on Surgery: Is Nicotine Replacement Therapy (NRT) a Safe Option? *Ann Surg.* 2021 Apr 1;273(4):e139-e141. doi: 10.1097/SLA.0000000000004522. PMID: 33214436.
14. Hanna ST. Nicotine effect on cardiovascular system and ion channels. *J Cardiovasc Pharmacol.* 2006 Mar;47(3):348-58. doi: 10.1097/01.fjc.0000205984.13395.9e. PMID: 16633075.
15. Wong LS, Martins-Green M. Firsthand cigarette smoke alters fibroblast migration and survival: implications for impaired healing. *Wound Repair Regen.* 2004 Jul-Aug;12(4):471-84. doi: 10.1111/j.1067-1927.2004.12403.x. PMID: 15260813.
16. Semlali A, Chakir J, Rouabhia M. Effects of whole cigarette smoke on human gingival fibroblast adhesion, growth, and migration. *J Toxicol Environ Health A.* 2011;74(13):848-62. doi: 10.1080/15287394.2011.570230. PMID: 21598170.
17. Saint-André V, Charbit B, Biton A, Rouilly V, Possémé C, Bertrand A, Rotival M, Bergstedt J, Patin E, Albert ML, Quintana-Murci L, Duffy D; Milieu Intérieur Consortium. Smoking changes adaptive immunity with persistent effects. *Nature.* 2024 Feb;626(8000):827-835. doi: 10.1038/s41586-023-06968-8. Epub 2024 Feb 14. PMID: 38355791; PMCID: PMC10881394.

18. Qiu F, Liang CL, Liu H, Zeng YQ, Hou S, Huang S, Lai X, Dai Z. Impacts of cigarette smoking on immune responsiveness: Up and down or upside down? *Oncotarget*. 2017 Jan 3;8(1):268-284. doi: 10.18632/oncotarget.13613. PMID: 27902485; PMCID: PMC5352117.
19. National Collaborating Centre for Women's and Children's Health (UK). *Surgical Site Infection: Prevention and Treatment of Surgical Site Infection*. London: RCOG Press; 2008 Oct. PMID: 21698848.
20. Sørensen LT, Toft BG, Rygaard J, Ladelund S, Paddon M, James T, Taylor R, Gottrup F. Effect of smoking, smoking cessation, and nicotine patch on wound dimension, vitamin C, and systemic markers of collagen metabolism. *Surgery*. 2010 Nov;148(5):982-90. doi: 10.1016/j.surg.2010.02.005. Epub 2010 Mar 29. PMID: 20347467.
21. Sørensen LT, Hemmingsen U, Kallehave F, Wille-Jørgensen P, Kjaergaard J, Møller LN, Jørgensen T. Risk factors for tissue and wound complications in gastrointestinal surgery. *Ann Surg*. 2005 Apr;241(4):654-8. doi: 10.1097/01.sla.0000157131.84130.12. PMID: 15798468; PMCID: PMC1357070.
22. Committee on the Public Health Implications of Raising the Minimum Age for Purchasing Tobacco Products; Board on Population Health and Public Health Practice; Institute of Medicine. *Public Health Implications of Raising the Minimum Age of Legal Access to Tobacco Products*. Bonnie RJ, Stratton K, Kwan LY, editors. Washington (DC): National Academies Press (US); 2015 Jul 23. PMID: 26269869.
23. Gibb C, Fitridge R. Smoking and the surgeon: no more excuses. *ANZ J Surg*. 2018 Sep;88(9):815-816. doi: 10.1111/ans.14774. PMID: 30182406.
24. Chow R, Lans Z, Cohen SP, Lin HM, Zhao J, Rajput K. Smoking and Postoperative Pain: A Systematic Review and Meta-Analysis. *Pain Pract*. 2025 Nov;25(8):e70083. doi: 10.1111/papr.70083. PMID: 40977634.
25. Sgonc R, Gruber J. Age-related aspects of cutaneous wound healing: a mini-review. *Gerontology*. 2013;59(2):159-64. doi: 10.1159/000342344. Epub 2012 Oct 27. PMID: 23108154.
26. Schmid M, Sood A, Campbell L, Kapoor V, Dalela D, Klett DE, Chun FK, Kibel AS, Sammon JD, Menon M, Fisch M, Trinh QD. Impact of smoking on perioperative outcomes after major surgery. *Am J Surg*. 2015 Aug;210(2):221-229.e6. doi: 10.1016/j.amjsurg.2014.12.045. Epub 2015 Apr 23. PMID: 25980408.
27. Fan Chiang YH, Lee YW, Lam F, Liao CC, Chang CC, Lin CS. Smoking increases the risk of postoperative wound complications: A propensity score-matched cohort study. *Int Wound J*. 2023 Feb;20(2):391-402. doi: 10.1111/iwj.13887. Epub 2022 Jul 9. PMID: 35808947; PMCID: PMC9885463.
28. Wilson JA, Clark JJ. Obesity: impediment to postsurgical wound healing. *Adv Skin Wound Care*. 2004 Oct;17(8):426-35. doi: 10.1097/00129334-200410000-00013. PMID: 15492679.
29. Franz S, Ertel A, Engel KM, Simon JC, Saalbach A. Overexpression of S100A9 in obesity impairs macrophage differentiation via TLR4-NFkB-signaling worsening inflammation and wound healing. *Theranostics*. 2022 Jan 16;12(4):1659-1682. doi: 10.7150/thno.67174. PMID: 35198063; PMCID: PMC8825590.
30. Brydges HT, McDonnell G, Nasr HY, Chaya BF, Onuh OC, Alfonso AR, Ceradini DJ. The impact of youth-onset type 2 diabetes on postoperative wound healing complications. *Wound Repair Regen*. 2023 Mar;31(2):187-192. doi: 10.1111/wrr.13064. Epub 2023 Jan 6. PMID: 36515565.
31. Burgess JL, Wyant WA, Abdo Abujamra B, Kirsner RS, Jozic I. Diabetic Wound-Healing Science. *Medicina (Kaunas)*. 2021 Oct 8;57(10):1072. doi: 10.3390/medicina57101072. PMID: 34684109; PMCID: PMC8539411.
32. Gupta S, Mujawdiya P, Maheshwari G, Sagar S. Dynamic Role of Oxygen in Wound Healing: A Microbial, Immunological, and Biochemical Perspective. *Arch Razi Inst*. 2022 Apr 30;77(2):513-523. doi: 10.22092/ARI.2022.357230.2003. PMID: 36284982; PMCID: PMC9548270.
33. Prochaska JJ, Benowitz NL. The Past, Present, and Future of Nicotine Addiction Therapy. *Annu Rev Med*. 2016;67:467-86. doi: 10.1146/annurev-med-111314-033712. Epub 2015 Aug 26. PMID: 26332005; PMCID: PMC5117107.
34. Hawn MT, Houston TK, Campagna EJ, Graham LA, Singh J, Bishop M, Henderson WG. The attributable risk of smoking on surgical complications. *Ann Surg*. 2011 Dec;254(6):914-20. doi: 10.1097/SLA.0b013e31822d7f81. PMID: 21869677.

35. Sorensen LT, Karlsmark T, Gottrup F. Abstinence from smoking reduces incisional wound infection: a randomized controlled trial. *Ann Surg.* 2003 Jul;238(1):1-5. doi: 10.1097/01.SLA.0000074980.39700.31. PMID: 12832959; PMCID: PMC1422652.
36. Sørensen LT, Toft B, Rygaard J, Ladelund S, Teisner B, Gottrup F. Smoking attenuates wound inflammation and proliferation while smoking cessation restores inflammation but not proliferation. *Wound Repair Regen.* 2010 Mar-Apr;18(2):186-92. doi: 10.1111/j.1524-475X.2010.00569.x. Epub 2010 Feb 16. PMID: 20163570.
37. Sørensen LT, Jorgensen LN, Zillmer R, Vange J, Hemmingsen U, Gottrup F. Transdermal nicotine patch enhances type I collagen synthesis in abstinent smokers. *Wound Repair Regen.* 2006 May-Jun;14(3):247-51. doi: 10.1111/j.1743-6109.2006.00118.x. PMID: 16808802.
38. Sriram K, Ramasubramanian V, Meguid MM. Special postoperative diet orders: Irrational, obsolete, and imprudent. *Nutrition.* 2016 Apr;32(4):498-502. doi: 10.1016/j.nut.2015.10.017. Epub 2015 Nov 21. PMID: 26746678.
39. DeLancey JO, Blay E Jr, Hewitt DB, Engelhardt K, Bilimoria KY, Holl JL, Odell DD, Yang AD, Stulberg JJ. The effect of smoking on 30-day outcomes in elective hernia repair. *Am J Surg.* 2018 Sep;216(3):471-474. doi: 10.1016/j.amjsurg.2018.03.004. Epub 2018 Mar 6. PMID: 29559083; PMCID: PMC6637424.
40. Grønkjær M, Eliassen M, Skov-Ettrup LS, Tolstrup JS, Christiansen AH, Mikkelsen SS, Becker U, Flensburg-Madsen T. Preoperative smoking status and postoperative complications: a systematic review and meta-analysis. *Ann Surg.* 2014 Jan;259(1):52-71. doi: 10.1097/SLA.0b013e3182911913. PMID: 23799418.
41. Musallam KM, Rosendaal FR, Zaatari G, Soweid A, Hoballah JJ, Sfeir PM, Zeineldine S, Tamim HM, Richards T, Spahn DR, Lotta LA, Peyvandi F, Jamali FR. Smoking and the risk of mortality and vascular and respiratory events in patients undergoing major surgery. *JAMA Surg.* 2013 Aug;148(8):755-62. doi: 10.1001/jamasurg.2013.2360. PMID: 23784299.
42. Turan A, Mascha EJ, Roberman D, Turner PL, You J, Kurz A, Sessler DI, Saager L. Smoking and perioperative outcomes. *Anesthesiology.* 2011 Apr;114(4):837-46. doi: 10.1097/ALN.0b013e318210f560. PMID: 21372682.
43. Li GQ, Guo FF, Ou Y, Dong GW, Zhou W. Epidemiology and outcomes of surgical site infections following orthopedic surgery. *Am J Infect Control.* 2013 Dec;41(12):1268-71. doi: 10.1016/j.ajic.2013.03.305. Epub 2013 Jul 25. PMID: 23890741.