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WOUND HEALING IN OPEN AURICULAR WOUNDS IN SMOKERS: A NARRATIVE LITERATURE REVIEW

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ABSTRACT

Smoking is associated with impaired tissue repair and a higher risk of postoperative wound complications. Open auricular wounds require particular attention because the pinna contains a thin soft-tissue envelope over cartilage, and injury severity depends on whether cartilage, perichondrium, or the vascularized skin envelope is disrupted. This narrative review discusses the biological effects of cigarette smoking on wound healing and considers how these mechanisms may be relevant to open wounds of the auricle. A focused narrative search was conducted using PubMed-indexed literature and reference-list checking, prioritizing reviews, meta-analyses, clinical studies, and anatomical reports concerning smoking, wound repair, and auricular injury. The evidence indicates that smoking can reduce tissue oxygenation, alter inflammatory-cell function, impair fibroblast migration and proliferation, and reduce collagen synthesis and deposition. Clinical meta-analysis has also associated smoking with increased risks of surgical-site infection, wound complications, delayed healing, and tissue necrosis. Auricular anatomy, however, should not be characterized as uniformly poorly vascularized: the skin receives arterial supply from the posterior auricular and superficial temporal systems with interconnections, whereas auricular cartilage itself is avascular and depends on surrounding tissues for nourishment. Direct clinical evidence specifically comparing healing of open auricular wounds in smokers and nonsmokers remains limited. Therefore, current recommendations for smoking cessation, careful assessment of cartilage involvement, atraumatic wound care, and surveillance for infection are supported mainly by broader wound-healing evidence and established auricular-trauma principles rather than auricle-specific comparative trials.

Keywords: Smoking; Wound Healing; Auricle; Cartilage; Tissue Oxygenation; Wound Complications

1 INTRODUCTION

Wound healing is a coordinated process involving hemostasis, inflammation, cellular proliferation, angiogenesis, extracellular-matrix deposition, and tissue remodeling. The process is influenced by local factors including perfusion, infection, tissue damage, and wound tension and systemic factors such as smoking, diabetes, nutritional status, and medications [1]. Cigarette smoking is a modifiable exposure associated with impaired wound repair and increased postoperative complications [2–5].

The external ear, or auricle, presents distinct anatomical considerations. Its shape is supported largely by elastic cartilage covered by a relatively thin skin and soft-tissue envelope. The auricular skin has an arterial network supplied principally by branches of the posterior auricular artery and superficial temporal artery, with anastomoses between vascular territories [6, 7]. Cartilage itself is avascular; consequently, its viability relies on the integrity of surrounding perichondrial and soft tissues. Lacerations involving cartilage, tissue loss, hematoma, contamination, or compromised skin coverage may require more than routine superficial wound management [8–11].

Although the adverse effects of smoking on surgical wounds and skin repair are well described, studies specifically examining open auricular wounds in smokers are scarce. This creates an important translational gap: evidence from general surgical populations may support biological plausibility and risk counseling, but it cannot quantify the effect of smoking on outcomes after auricular trauma. The contribution of this narrative review is to bring together smoking-related wound biology, clinical evidence on general wound complications, and the distinct anatomy and management considerations of the auricle, while explicitly distinguishing direct evidence from extrapolation.

2 METHODS

This article was prepared as a narrative literature review, not a systematic review. A focused search of PubMed-indexed literature was supplemented by checking reference lists of relevant reviews and primary studies. Search concepts were combined as appropriate and included “smoking,” “cigarette smoke,” “wound healing,” “collagen,” “surgical-site infection,” “auricle,” “auricular injury,” and “external ear.” Sources were considered relevant when they addressed at least one of the following: (1) biological mechanisms linking smoking with impaired tissue repair; (2) clinical associations between smoking and wound complications; (3) vascular anatomy or tissue characteristics of the auricle; or (4) assessment and management of open auricular injuries. Systematic reviews and meta-analyses were prioritized for broad clinical associations, while primary clinical, experimental, anatomical, and trauma-management studies were used to clarify mechanisms and site-specific considerations. The literature was selected purposively for relevance to the review question rather than through a predefined exhaustive screening process. No formal protocol, protocol registration, duplicate independent screening, formal risk-of-bias assessment, or quantitative synthesis was undertaken. The search date range and number of records screened were not prospectively documented; accordingly, this review should be interpreted as a focused narrative synthesis and not as an exhaustive or reproducible systematic search.

3 DISCUSSION

3.1 Normal wound-healing phases and relevant tissue biology

Wound healing is commonly described in overlapping phases. Hemostasis limits blood loss and provides a provisional matrix. During inflammation, neutrophils and macrophages remove contaminants and coordinate repair. In the proliferative phase, keratinocytes migrate to restore the epidermal barrier, endothelial cells support new-vessel formation, and fibroblasts produce extracellular matrix, including collagen. Remodeling reorganizes the matrix and gradually changes the mechanical properties of the healed tissue [1, 12, 13]. These phases overlap and their timing varies with wound depth, tissue type, perfusion, infection, and host factors.

An open auricular wound may involve skin alone or extend to perichondrium and cartilage. The presence of exposed or injured cartilage changes the clinical problem because cartilage lacks intrinsic vascularity. Nevertheless, the auricle's soft tissues have a defined arterial supply and interconnected vessels; it would be inaccurate to describe the whole auricle as inherently poorly vascularized [6, 7, 11]. The risk to healing depends on the location and extent of injury, preservation of the soft-tissue envelope, contamination, hematoma, and the quality of wound management.

3.2 Mechanisms by which smoking can impair wound healing

Cigarette smoke contains carbon monoxide, oxidants, and numerous toxic compounds. Carbon monoxide binds hemoglobin and reduces the amount of oxygen available for tissue delivery, while smoke-related vasoconstrictive and endothelial effects can further compromise microcirculatory function. Tissue oxygen is important for oxidative bacterial

killing, cellular metabolism, collagen hydroxylation, and other processes required for repair [2, 14, 15]. The magnitude and duration of these effects vary with smoking intensity, timing of exposure, comorbidities, and the type of wound.

Smoking is also associated with changes in the inflammatory response. Sørensen's pathophysiological review described reductions in inflammatory-cell chemotaxis, migration, and oxidative bactericidal function among the mechanisms potentially linking smoking with impaired host defense [2]. These changes may make contaminated wounds more vulnerable to infection, although the absolute risk for an individual auricular wound cannot be inferred from these mechanisms alone.

During the proliferative phase, fibroblasts migrate into the wound, proliferate, and synthesize extracellular matrix. Experimental and clinical literature indicates that cigarette smoke exposure can impair fibroblast migration and survival and reduce collagen synthesis or deposition [2, 16–18]. Jørgensen and colleagues reported lower collagen production in smokers, supporting a biological pathway through which smoking may weaken repair [16]. Collagen is essential to the developing wound matrix; however, the effects on final strength and scar quality depend on multiple interacting factors and should not be reduced to collagen alone.

Angiogenesis and matrix remodeling are likewise regulated processes. Effective healing requires adequate vascular support, controlled protease activity, and balanced deposition and degradation of extracellular matrix [1, 12, 13]. Smoking-related hypoxia and altered cellular function may disrupt this balance. These mechanisms provide a plausible rationale for concern in auricular injuries, especially when the soft-tissue envelope is damaged, but direct auricle-specific mechanistic data remain insufficient.

3.3 Clinical evidence linking smoking with wound complications

Clinical evidence is strongest for surgical wounds and postoperative complications generally, rather than traumatic auricular wounds specifically. A systematic review and meta-analysis of 140 cohort studies involving 479,150 patients reported higher adjusted odds among smokers for tissue necrosis (OR 3.60, 95% CI 2.62–4.93), delayed healing or dehiscence (OR 2.07, 95% CI 1.53–2.81), surgical-site infection (OR 1.79, 95% CI 1.57–2.04), and overall wound complications (OR 2.27, 95% CI 1.82–2.84) [3]. These are pooled odds ratios from heterogeneous surgical settings, not absolute risks and not estimates specific to traumatic ear wounds. They support the general association between smoking and wound complications, but differences in wound mechanism, contamination, tissue loss, cartilage involvement, and treatment limit direct transfer of these effect estimates to auricular trauma.

A separate meta-analysis of preoperative smoking status also reported associations between smoking and postoperative complications across surgical populations [4]. Intervention studies suggest that perioperative smoking cessation can reduce some complications, although effects vary by outcome, intervention intensity, and the time available before surgery [3, 5, 20]. These findings strengthen the rationale for offering cessation support, but they do not show that cessation guarantees uncomplicated healing or establish the optimal cessation interval for acute auricular injuries. Reviews in plastic surgery likewise emphasize smoking history, risk communication, and cessation around reconstructive procedures [19]. Taken together, the clinical literature is directionally consistent with the proposed biological mechanisms; however, most evidence is observational or arises from planned surgical settings. Confounding by comorbidities and differences in procedure type may affect the observed associations, while acute traumatic wounds introduce additional factors such as contamination, crush injury, tissue loss, delayed presentation, and limited opportunity for pre-injury cessation. The evidence therefore supports cautious clinical counseling rather than a precise auricle-specific risk estimate.

3.4 Clinical considerations for open auricular wounds in smokers

Assessment should first establish the mechanism of injury, timing, contamination, tissue loss, tetanus status, comorbidities, medication use, and smoking history. Examination should document wound dimensions and location, bleeding, skin viability, hematoma, cartilage exposure or disruption, and signs of infection. A careful assessment of the wound's vascularized soft-tissue coverage is important because cartilage does not have its own blood supply [6–11].

Management depends on injury pattern and should follow accepted trauma and reconstructive principles. Relevant considerations include gentle cleansing and irrigation, removal of clearly devitalized tissue when indicated, appropriate closure of viable tissue, management of auricular hematoma, and specialist evaluation when there is significant cartilage involvement, avulsion, tissue loss, or uncertainty about viability. The literature on auricular trauma describes several approaches according to the extent of cartilage injury and the amount of remaining soft-tissue attachment; no single technique is appropriate for every injury [8–11]. This review does not replace local clinical protocols or individualized surgical judgment.

For patients who smoke, cessation counseling should be initiated as early as feasible and continued during recovery. Clinicians should explain that stopping smoking may improve tissue oxygenation and reduce certain wound

complications, while avoiding promises that cessation will guarantee uncomplicated healing [2, 3, 5, 19]. Support may include brief advice, behavioral assistance, and evidence-based cessation treatment according to local guidance. Follow-up should assess pain, erythema, swelling, drainage, wound separation, skin discoloration, and other signs of infection or tissue compromise. A low threshold for reassessment is reasonable when cartilage is exposed or the soft-tissue envelope is compromised.

3.5 Evidence gaps and implications for future research

The main evidence gap is the lack of direct comparative studies evaluating open auricular wound outcomes in smokers and nonsmokers. The review's principal strength is its integration of three evidence domains—wound-healing mechanisms, broader clinical outcome data, and auricular anatomy/trauma management—while making the limits of extrapolation explicit. Its limitations follow from the narrative design: the search was not exhaustive, search dates and record counts were not documented, selection was not performed independently by multiple reviewers, and no formal critical appraisal or risk-of-bias assessment was conducted. Consequently, the review cannot establish causality, calculate an auricle-specific effect size, or determine whether smoking independently predicts outcomes such as infection, cartilage exposure, perichondrial compromise, tissue necrosis, delayed closure, or reconstructive failure. These limitations should be considered when interpreting the synthesis.

Future studies should define smoking exposure clearly, including current use, cigarettes per day, cumulative exposure, second-hand smoke where relevant, and time since cessation. Outcomes should be standardized and should account for wound mechanism, contamination, cartilage involvement, injury severity, diabetes, vascular disease, and treatment approach. Prospective multicenter cohorts may be more feasible than randomized exposure studies and could clarify whether smoking independently predicts complications after auricular trauma. Until such data are available, clinicians should transparently distinguish direct evidence from extrapolation.

4 CONCLUSION

Smoking is associated with impaired wound-healing biology and increased postoperative wound complications, with plausible mechanisms involving reduced tissue oxygenation, altered inflammatory responses, impaired fibroblast activity, and reduced collagen synthesis. These findings are relevant when caring for smokers with open auricular wounds, but direct evidence specific to this injury population remains limited. Auricular management should be guided by the injury pattern, cartilage and perichondrial involvement, preservation of the vascularized soft-tissue envelope, and established trauma principles. Smoking cessation counseling and close wound follow-up are reasonable components of care, while further auricle-specific research is needed to quantify risk and determine outcomes.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

The authors declare no conflict of interest.

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