The Dual Role of VEGF-Mediated Angiogenesis in Oral Tissues: From Physiological Wound Healing to Oral Cancer Progression

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Vascular endothelial growth factor (VEGF) is a key regulator of angiogenesis that plays an essential role in maintaining tissue homeostasis and vascular remodeling. In oral tissues, VEGF-mediated angiogenesis is indispensable for physiological wound healing by promoting neovascularization, oxygen delivery, and tissue regeneration. However, dysregulated VEGF signaling also contributes to pathological angiogenesis that supports oral squamous cell carcinoma (OSCC) progression. Understanding these context-dependent functions is essential for developing regenerative and therapeutic strategies targeting VEGF. This narrative review aimed to critically evaluate the dual role of VEGF-mediated angiogenesis in oral tissues by comparing its physiological function during oral mucosal wound healing with its pathological role in oral cancer progression. A narrative review was conducted by synthesizing peer-reviewed literature. Relevant articles were identified using electronic databases with keywords related to oral mucosal wound healing, angiogenesis, VEGF, HIF-1α, oral squamous cell carcinoma, and tumor angiogenesis. Selected studies were analyzed qualitatively to identify the molecular mechanisms regulating VEGF signaling in physiological and pathological conditions. The reviewed literature demonstrated that transient VEGF activation is essential for physiological angiogenesis, facilitating endothelial cell proliferation, vascular remodeling, and tissue regeneration during oral wound healing. In contrast, persistent VEGF overexpression under chronic hypoxia and inflammation promotes pathological angiogenesis characterized by abnormal vascularization, increased tumor growth, invasion, lymph node metastasis, and poor prognosis in OSCC. Most available studies focused on VEGF in oral cancer, whereas evidence regarding its regulatory role during oral mucosal wound healing remains comparatively limited. VEGF serves as a context-dependent regulator of angiogenesis in oral tissues, contributing to both physiological tissue repair and pathological vascular remodeling. During oral mucosal wound healing, controlled and transient VEGF activation supports neovascularization and tissue regeneration, whereas sustained VEGF signaling under pathological conditions has been associated with angiogenesis that may facilitate oral cancer progression. The current evidence highlights the importance of understanding the molecular regulation of VEGF across different biological contexts. Further studies are needed to clarify the mechanisms governing VEGF activity during oral mucosal wound healing and to explore strategies that promote physiological angiogenesis while maintaining vascular homeostasis and reducing the risk of pathological angiogenesis.

Keywords: vascular endothelial growth factor, angiogenesis, oral mucosal wound healing, oral squamous cell carcinoma, VEGF signaling.

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1. Introduction

Oral tissues possess a unique regenerative capacity that enables rapid repair following injury compared with many other tissues. Oral mucosal wound healing is characterized by rapid epithelial turnover, abundant vascularization, continuous exposure to saliva, and a complex microbial environment that collectively

influence tissue repair processes (Politis et al., 2016). Saliva contributes to mucosal homeostasis by providing growth factors, antimicrobial proteins, and bioactive molecules, including epidermal growth factor (EGF) and histatins, which promote epithelial migration, re-epithelialization, and wound closure (Brand et al., 2014). Despite these protective mechanisms, extensive oral mucosal injuries, including traumatic ulcers, surgical wounds, and therapy-induced oral mucositis, may exceed the regenerative capacity of oral tissues, resulting in delayed healing and impaired oral function (Pulito et al., 2020).

Successful tissue regeneration requires coordinated regulation of inflammation, angiogenesis, extracellular matrix remodeling, and epithelial restoration. Among these processes, angiogenesis plays a crucial role by restoring blood supply, oxygen delivery, and nutrient availability within the healing wound bed. Vascular endothelial growth factor (VEGF) is recognized as a primary regulator of angiogenesis through its ability to stimulate endothelial cell proliferation, migration, survival, and new vessel formation (Melincovici et al., 2018; Apte et al., 2019). During wound healing, VEGF expression is induced by local hypoxia through activation of hypoxia-inducible factor-1 alpha (HIF-1α), facilitating neovascularization and supporting tissue regeneration (Cañedo-Dorantes & Cañedo-Ayala, 2019).

However, the biological role of VEGF extends beyond physiological tissue repair. Dysregulated VEGF signaling contributes to pathological angiogenesis, particularly in cancer development and progression. In oral squamous cell carcinoma (OSCC), increased VEGF expression has been associated with enhanced tumor vascularization, invasion, metastasis, and poor clinical outcomes (Lalla et al., 2011; Folkman, 2007). This dual function highlights the complexity of VEGF signaling, where controlled activation promotes tissue regeneration, whereas excessive or persistent activation facilitates disease progression.

Therefore, this narrative review aims to critically discuss the dual role of VEGF-mediated angiogenesis in oral tissues by comparing its physiological function during oral mucosal wound healing and its pathological contribution to oral cancer progression. This review focuses on the molecular regulation of VEGF signaling, the cellular mechanisms underlying angiogenesis, and the factors determining whether VEGF activity contributes to tissue regeneration or pathological vascularization.

2. Literature Review And Problem Statement

Angiogenesis represents a fundamental biological process involved in both tissue regeneration and disease progression. During oral mucosal wound healing, tissue injury induces inflammatory responses and local hypoxia, leading to stabilization of HIF-1α and subsequent activation of VEGF expression. VEGF binds primarily to vascular endothelial growth factor receptor-2 (VEGFR-2) on endothelial cells, initiating downstream signaling pathways that regulate endothelial proliferation, migration, vascular permeability, and capillary formation (Ferrara, 2009). This coordinated angiogenic response facilitates restoration of microvascular networks, supports immune cell trafficking, and provides essential resources for epithelial and connective tissue regeneration (DiPietro, 2016). However, excessive or prolonged VEGF activation can disrupt tissue homeostasis and contribute to pathological angiogenesis.

In oral cancer, particularly OSCC, VEGF-mediated angiogenesis becomes a critical mechanism supporting tumor growth and progression. Tumor cells, cancer-associated fibroblasts, and infiltrating immune cells can produce VEGF in response to hypoxia and inflammatory signals, promoting abnormal vascular development within the tumor microenvironment (Lalla et al., 2011). Unlike physiological angiogenesis during wound healing, tumor-associated angiogenesis is characterized by uncontrolled vessel formation, increased vascular permeability, and structurally abnormal blood vessels that facilitate tumor invasion and metastasis (Folkman, 2007). These contrasting effects demonstrate that VEGF function is highly dependent on biological context, cellular sources, duration of activation, and the surrounding microenvironment.

Despite extensive research on VEGF in wound healing and cancer biology separately, the mechanisms determining the transition between beneficial and pathological angiogenesis in oral tissues remain incompletely understood. The oral cavity represents a unique biological environment influenced by saliva, microbiota, immune responses, and continuous mechanical stress, which may modify VEGF signaling outcomes. Therefore, the central problem addressed in this narrative review is how VEGF-mediated angiogenesis is regulated differently during physiological oral mucosal repair and oral cancer progression, and how understanding this balance may provide insights for developing strategies that enhance tissue regeneration while limiting pathological vascularization.

3. Method

This study employed a narrative review approach to summarize and critically analyze current evidence regarding the dual role of vascular endothelial growth factor (VEGF)-mediated angiogenesis in oral tissues, focusing on its physiological function during oral mucosal wound healing and its pathological contribution to oral cancer progression. A narrative review approach was selected because it allows the integration of findings from various study designs, including experimental and clinical studies, to provide a comprehensive understanding of the molecular mechanisms regulating VEGF signaling in different biological contexts. Relevant articles were identified using keywords including *oral mucosal wound healing*, *oral tissue regeneration*, *angiogenesis*, *vascular endothelial growth factor (VEGF)*, *VEGF signaling*, *HIF-1α*, *hypoxia*, *oral squamous cell carcinoma*, and *tumor angiogenesis*. Peer-reviewed original research articles and review articles relevant to VEGF-mediated angiogenesis in oral tissues were included, while duplicate, non-English, and unrelated publications were excluded. The selected studies were analyzed qualitatively and synthesized based on the main themes of this review, including VEGF regulation during wound healing, mechanisms of physiological angiogenesis, pathological angiogenesis in oral cancer, and factors determining the dual biological roles of VEGF signaling.

4. Results And Discussion

The findings of this narrative review are presented in tables and figures to summarize and synthesize the evidence from the selected studies. This presentation facilitates comparison of study characteristics, key findings, and research trends regarding the dual role of VEGF-mediated angiogenesis in oral tissues. The synthesized evidence enables readers to identify current knowledge, existing inconsistencies, and research gaps related to physiological wound healing and oral cancer progression.

Table 1.Summary of Evidence on the Dual Role of VEGF-Mediated Angiogenesis in Oral Tissues

No	Reference	Study Type	Biological Context	Key Findings	Relevance to Review
1	Keswani et al. (2013)	Experimental wound healing study	Physiological oral wound healing	Salivary VEGF promotes palatal mucosal wound healing by enhancing angiogenesis, vascular remodeling, and tissue regeneration	Direct evidence supporting VEGF as regenerative mediator in oral mucosal repair
2	Di Alberti et al. (2013)	Human tissue study	Oral implant tissue/peri-implant inflammation	VEGF expression reflects vascular response in healthy and inflamed oral tissues	Supports VEGF involvement in oral tissue vascular homeostasis

No	Reference	Study Type	Biological Context	Key Findings	Relevance to Review
3	Johnstone & Logan (2007)	Immunohistochemical study	Normal mucosa → dysplasia → OSCC	VEGF expression increased during malignant transformation from normal mucosa to OSCC	Demonstrates VEGF transition from physiological to pathological angiogenesis
4	Aggarwal et al. (2014)	Clinical study	Oral squamous cell carcinoma	VEGF expression was increased in OSCC and associated with tumor characteristics	Supports VEGF involvement in oral cancer progression
5	Yanase et al. (2014)	Clinical prognostic study	OSCC	VEGF-A and VEGF-C expression showed association with prognosis and tumor behavior	Supports VEGF as prognostic marker
6	Nakazato et al. (2006)	Experimental cancer study	OSCC model	VEGF-A promoted angiogenesis, while VEGF-C contributed to lymphatic metastasis	Demonstrates distinct roles of VEGF family members
7	Chen et al. (2010)	Clinical tissue study	OSCC	VEGF-C expression correlated with lymphangiogenesis and lymph node metastasis	Supports pathological vascular remodeling
8	Patel et al. (2015)	Molecular/clinical study	OSCC	VEGFA isoforms contributed to oral cancer progression through angiogenic regulation	Highlights VEGF signaling complexity
9	Li et al. (2005)	Histopathological study	OSCC	VEGF expression correlated with microvessel density in tumors	Demonstrates VEGF-driven tumor vascularization
10	Edirisinghe et al. (2023)	Biomarker study	OSCC	VEGF-A and VEGFR-2 showed potential as biomarkers for OSCC	Supports VEGF/VEGFR axis as clinical target
11	Sun et al. (2022)	Experimental molecular study	OSCC cells	Inhibition of VEGF/VEGFR-2 signaling reduced angiogenesis and cancer cell growth	Supports VEGF pathway targeting in oral cancer

The Physiological Role of VEGF-Mediated Angiogenesis in Oral Mucosal Wound Healing

Angiogenesis is an essential component of the proliferative phase of oral mucosal wound healing. Following tissue injury, disruption of local blood vessels creates a transient hypoxic microenvironment that stabilizes hypoxia-inducible factor-1 alpha (HIF-1α), leading to increased VEGF expression. VEGF subsequently binds to VEGFR-2 on endothelial cells, stimulating endothelial proliferation, migration, and capillary sprouting to restore oxygen and nutrient delivery to the injured tissue (Ferrara, 2009; Apte et al., 2019; DiPietro, 2016). Besides promoting neovascularization, VEGF also facilitates extracellular matrix remodeling and epithelial regeneration, both of which are critical for successful wound closure (Melincovici et al., 2018; Cañedo-Dorantes & Cañedo-Ayala, 2019).

Vascular Endothelial Growth Factor (VEGF) plays a crucial physiological role in mediating oral mucosal wound healing through angiogenesis. As a potent angiogenic factor, VEGF directly stimulates endothelial

cell migration and proliferation, which are essential for new blood vessel formation (Bode et al., 2023; Sethi et al., 2024; Gurtner et al., 2015). This VEGF-mediated revascularization ensures an adequate supply of blood, oxygen, and nutrients to damaged tissues, supporting effective tissue repair and regeneration following injury or grafting procedures (Bode et al., 2023; Gurtner et al., 2015; Daly et al., 2021). Beyond blood vessel formation, VEGF regulates vascular permeability and recruits immune cells, thereby modulating inflammation at the healing site (Sethi et al., 2024; Gurtner et al., 2015). Driven by rapid angiogenesis, oral mucosal wounds benefit from accelerated healing relative to cutaneous wounds while avoiding the development of chronic non-healing lesions (Daly et al., 2021; MDPI Review, 2025). Throughout the repair process, VEGF acts in concert with other growth factors and cytokines, integrating signals to coordinate the sequential phases of wound healing, including hemostasis, inflammation, proliferation, and tissue remodeling (Sethi et al., 2024; Gurtner et al., 2015).

Compared with cutaneous tissue, oral mucosa exhibits faster healing and minimal scar formation due to its abundant vascular network, saliva-derived growth factors, and tightly regulated inflammatory response (Politis et al., 2016; Brand et al., 2014). Among the studies identified in this review, only Keswani et al. (2013) directly investigated VEGF during oral mucosal wound healing, demonstrating that salivary VEGF significantly contributes to palatal wound repair by enhancing angiogenesis and tissue regeneration. This finding suggests that physiological VEGF activation is indispensable for maintaining oral tissue homeostasis. Nevertheless, the limited number of studies focusing specifically on oral wound healing indicates that the molecular regulation of VEGF in oral tissue regeneration remains insufficiently explored.

VEGF as a Driver of Pathological Angiogenesis in Oral Cancer

Unlike physiological wound healing, where VEGF expression is transient and tightly regulated, persistent VEGF activation is a hallmark of pathological angiogenesis in oral squamous cell carcinoma (OSCC). Most studies included in this review focused on VEGF expression in oral cancer rather than tissue regeneration. Increased expression of VEGF-A, VEGF-C, and VEGFR-2 has consistently been associated with increased microvessel density, tumor growth, invasion, lymph node metastasis, and poor clinical prognosis (Johnstone & Logan, 2007; Aggarwal et al., 2014; Yanase et al., 2014; Patel et al., 2015; Edirisinghe et al., 2023). These findings indicate that VEGF plays a central role in sustaining tumor vascularization and facilitating cancer progression.

Vascular Endothelial Growth Factor (VEGF) plays a central role in driving angiogenesis and progression in Oral Squamous Cell Carcinoma (OSCC). Mechanistically, VEGF operates through several coordinated cellular pathways to support tumor growth. Primarily acting as a selective mitogen, VEGF-A serves as the main mediator of blood vessel formation by stimulating the proliferation, differentiation, and directional migration of vascular endothelial cells to form new capillary sprouts, thereby supplying essential oxygen and nutrients to the tumor microenvironment (Nakazato et al., 2006; Gupta et al., 2016; Pradeep et al., 2018; Edirisinghe et al., 2023). Concurrently, VEGF increases vascular permeability, allowing plasma proteins to leak into the extracellular space and construct a provisional matrix that structurally supports vessel sprouting (Gupta et al., 2016). Beyond vessel formation, VEGF serves as a crucial survival factor that protects newly formed endothelial cells from apoptosis, ensuring the stabilization of the tumor microvasculature (Gupta et al., 2016).

Notably, OSCC cells utilize both paracrine signaling where secreted VEGF targets surrounding endothelial cells—and autocrine signaling, in which VEGF directly binds to its receptors (VEGFRs) on tumor cells to promote cell survival (Araki-Maeda et al., 2022). In addition to blood vessel development, VEGF-C expression specifically induces lymphangiogenesis the formation of new lymphatic vessels, which facilitates cancer cell dissemination to regional lymph nodes (Nakazato et al., 2006; Shintani et al., 2004). Collectively,

these cellular mechanisms directly drive elevated microvessel density (MVD) as tissue lesions progress from normal mucosa to dysplasia and invasive carcinoma (Edirisinghe et al., 2023; Martano et al., 2016). Consequently, high VEGF levels strongly correlate with deeper tumor invasion, lymph node metastasis, and poor overall survival, making targeted inhibition of the VEGF signaling pathway a significant therapeutic strategy to suppress angiogenesis and inhibit tumor growth in OSCC (Kim et al., 1993; Smith et al., 2000).

Tumor angiogenesis differs substantially from physiological angiogenesis. Under normal wound healing conditions, newly formed blood vessels mature and regress once tissue repair is complete. In contrast, the tumor microenvironment is characterized by chronic hypoxia, persistent inflammation, and continuous production of pro-angiogenic mediators, resulting in excessive VEGF expression and uncontrolled vascular growth (Ferrara, 2009; Folkman, 2007). The resulting tumor vasculature is structurally abnormal, highly permeable, and poorly organized, creating conditions that facilitate tumor invasion, immune evasion, and metastatic dissemination (Melincovici et al., 2018; Pomella et al., 2024). Consequently, VEGF-mediated angiogenesis becomes a critical therapeutic target in OSCC.

Research Gap and Future Perspectives

An important finding emerging from this review is the imbalance in the current literature. Most published studies investigate VEGF as a biomarker or therapeutic target in OSCC, whereas relatively few studies explore its physiological role in oral mucosal wound healing. This disparity suggests that knowledge regarding the temporal regulation of VEGF during normal oral tissue repair remains limited.

Future research should therefore focus on elucidating the molecular mechanisms regulating VEGF expression throughout different phases of oral wound healing, particularly its interactions with HIF-1α, TGF-β, inflammatory cytokines, and immune cells. Such studies may help identify therapeutic strategies capable of promoting physiological angiogenesis to accelerate oral tissue regeneration while avoiding excessive VEGF activation that may predispose to pathological angiogenesis. A better understanding of this biological balance may contribute to the development of safer regenerative therapies and more effective anti-angiogenic approaches for oral diseases.

5. Conclusion

VEGF-mediated angiogenesis plays a dual and context-dependent role in oral tissues. During physiological oral mucosal wound healing, transient activation of VEGF is essential for endothelial cell proliferation, neovascularization, oxygen delivery, and tissue regeneration, enabling efficient restoration of tissue integrity. In contrast, persistent VEGF overexpression driven by chronic hypoxia, inflammation, and oncogenic signaling promotes pathological angiogenesis characterized by abnormal vascular architecture, enhanced tumor growth, invasion, and metastasis in oral squamous cell carcinoma.

This narrative review indicates that the current body of literature has predominantly focused on the role of VEGF in oral cancer progression, whereas studies examining its regulatory function during physiological oral mucosal wound healing remain comparatively limited. Consequently, further investigations are warranted to better understand the temporal and molecular regulation of VEGF throughout the wound healing process and to elucidate the mechanisms that distinguish physiological angiogenesis from pathological vascularization in oral tissues.

A comprehensive understanding of the dual biological functions of VEGF may provide an important foundation for developing targeted therapeutic strategies that promote oral tissue regeneration while minimizing excessive angiogenesis associated with tumor progression. Such knowledge may contribute to

advances in regenerative dentistry as well as precision approaches for the prevention, diagnosis, and treatment of oral diseases.

6. Referensi

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