Review Article ISSN 2639-944X

Journal of Medical - Clinical Research & Reviews

Cancer Stem Cells in Oral Neoplasms: Implications for Therapy and Recurrence

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Received: 18 Oct 2025; **Accepted:** 30 Nov 2025; **Published:** 10 Dec 2025

Citation: Verena Lengston. Cancer Stem Cells in Oral Neoplasms: Implications for Therapy and Recurrence. J Med - Clin Res & Rev. 2025; 9(12): 1-6.

ABSTRACT

Oral squamous cell carcinoma (OSCC), the predominant malignancy of the oral cavity, is characterized by high rates of local recurrence, metastasis, and therapeutic resistance, contributing to persistently poor survival outcomes. The cancer stem cell (CSC) paradigm offers a compelling biological explanation for these clinical challenges. This paper provides a comprehensive examination of CSCs in oral neoplasms, elucidating their identification, molecular regulation, and pivotal role in tumor initiation, progression, and treatment failure. We detail the characteristic biomarkers used to isolate oral CSCs including CD44, ALDH1, CD133, and epithelial-specific antigen (ESA) and the core signaling pathways (Notch, Hedgehog, Wnt/β-catenin, and Bmi-1) that govern their self-renewal and survival. A critical analysis reveals that CSCs exhibit intrinsic resistance to conventional chemotherapy and radiotherapy through enhanced DNA repair capacity, upregulation of drug efflux pumps, activation of anti-apoptotic pathways, and a quiescent cell-cycle state. These properties enable CSCs to survive initial treatment and subsequently drive tumor recurrence and metastatic dissemination. The clinical implications are profound: a tumor's CSC burden may correlate with advanced stage, nodal metastasis, and worse prognosis. Moving beyond conventional cytotoxic approaches, this paper explores emerging therapeutic strategies specifically targeting the CSC compartment. These include pathway-specific inhibitors, agents targeting the CSC niche, differentiation therapy, and immunotherapeutic approaches leveraging CSC-specific antigens. We conclude that successfully eradicating oral CSCs, or disrupting their maintenance circuits, represents the most promising avenue for achieving durable cures and preventing recurrence in OSCC. Integrating CSC-targeted agents with standard therapies in a rational, biomarker-driven manner may fundamentally transform the therapeutic landscape for this devastating disease.

Keywords

Cancer Stem Cells, Oral Squamous Cell Carcinoma, Tumor Recurrence, Therapeutic Resistance, CD44, ALDH1, Notch Signaling, Metastasis, Targeted Therapy, Minimal Residual Disease.

Introduction: The Persistence of Oral Cancer

Oral squamous cell carcinoma (OSCC) accounts for over 90% of all oral malignancies and remains a significant global health burden with an estimated 377,713 new cases and 177,757 deaths annually. Despite advances in multimodal therapy combining surgery, radiotherapy, and chemotherapy, the 5-year survival rate for advanced-stage disease has stagnated at approximately 40-60%, largely due to high rates of local recurrence, regional metastasis, and the development of distant disease. This clinical recalcitrance suggests that conventional therapies, while effective at debulking the tumor mass, fail to eradicate a critical subpopulation of cells responsible for long-term tumor maintenance and regeneration [1-23].

The cancer stem cell (CSC) hypothesis provides a robust conceptual framework to explain these phenomena. First proposed for hematologic malignancies and later solid tumors, this model posits that tumors are hierarchically organized, mirroring normal tissues. At the apex of this hierarchy reside CSCs (or tumor-initiating cells, TICs): a small, biologically distinct subpopulation endowed with the capacity for self-renewal (giving rise to new CSCs) and differentiation (generating the heterogeneous, non-tumorigenic

progeny that constitute the bulk of the tumor). CSCs are proposed to be the primary drivers of tumor initiation, progression, metastasis, and recurrence due to their unique biological properties, including enhanced survival mechanisms and relative quiescence.

In the context of OSCC, the CSC paradigm offers critical insights into its aggressive behavior. This paper will synthesize current evidence on the identification and characterization of oral CSCs, delineate their molecular underpinnings, and critically analyze their direct contribution to therapeutic resistance and disease relapse. Finally, we will explore the translational implications of this knowledge, surveying novel therapeutic strategies aimed at targeting the CSC compartment to achieve more durable clinical responses.

Identification and Characterization of Oral Cancer Stem Cells

The isolation and study of CSCs rely on the identification of specific cellular markers and functional assays that demonstrate stem-like properties.

Surface and Intracellular Biomarkers

A combination of markers is typically used to enrich for oral CSC populations, as no single marker is universally specific [24-29].

- CD44: The most widely studied oral CSC marker. CD44, a transmembrane glycoprotein receptor for hyaluronan, is involved in cell adhesion, migration, and signaling. The CD44+ subpopulation in OSCC cell lines and primary tumors demonstrates increased tumorigenicity in immunodeficient mice, enhanced sphere-forming capacity *in vitro*, and resistance to therapy. The variant isoform CD44v6, in particular, is strongly associated with metastatic potential.
- ALDH1 (Aldehyde Dehydrogenase 1): A detoxifying enzyme responsible for oxidizing intracellular aldehydes. High ALDH1 enzymatic activity, measured by the ALDEFLUOR assay, serves as a functional marker for stem cells in various cancers. ALDH1+ cells from OSCC exhibit greater self-renewal, differentiation potential, and tumor initiation capacity compared to ALDH1- cells.
- CD133 (Prominin-1): A pentaspan transmembrane glycoprotein. While its function remains unclear, CD133+ cells in OSCC show enhanced clonogenicity, chemoresistance, and expression of stemness-related genes.
- Combination Markers: Often, a combination such as CD44+CD133+ or CD44high/ALDH1high identifies a more highly enriched tumorigenic population. The Side Population (SP) phenotype, identified via efflux of Hoechst 33342 dye by ABC transporters like ABCG2, also identifies cells with stem-like properties.

Functional Assays for Stemness

- Sphere-Forming Assay: When cultured in ultra-low attachment plates with serum-free media supplemented with growth factors, CSCs form non-adherent three-dimensional colonies called tumorspheres. This capacity for anchorage-independent growth and self-renewal is a hallmark of stem-like cells.
- *In Vivo* Tumorigenicity Assay: The gold-standard functional test. Serial dilution of putative CSC populations (e.g., CD44+ vs. CD44-) implanted into immunodeficient mice (NOD/SCID or NSG) demonstrates that the marker-positive fraction can initiate tumors at very low cell numbers, recapitulating the heterogeneity of the original tumor, while the marker-negative fraction cannot or requires vastly higher numbers.
- Clonogenic Assay: Measures the ability of a single cell to proliferate and form a large colony, indicating self-renewal and proliferative potential [27-35].

Molecular Regulation of the Oral CSC Phenotype

The maintenance of the CSC state is governed by a core set of evolutionarily conserved signaling pathways and transcription factors, often dysregulated in OSCC.

Key Developmental Signaling Pathways

- Notch Signaling: Plays a crucial role in cell fate determination. In OSCC, Notch signaling is frequently dysregulated, with evidence supporting both oncogenic and tumor-suppressive roles depending on context. However, in CSCs, activated Notch (Notch1 intracellular domain, NICD) promotes self-renewal, survival, and epithelial-mesenchymal transition (EMT). Inhibition of Notch signaling depletes the CSC pool and sensitizes cells to chemotherapy.
- Hedgehog (Hh) Signaling: Essential for embryonic patterning and stem cell maintenance. Components like Gli1 and Patched1 are overexpressed in OSCC CSCs. Hh pathway activation promotes sphere formation, proliferation, and contributes to chemoresistance. Cyclopamine, a Hh inhibitor, reduces the CD44+ population and tumorigenicity.
- Wnt/β-Catenin Signaling: A central regulator of stem cells. Nuclear accumulation of β-catenin, a hallmark of pathway activation, is common in OSCC and correlates with poor differentiation and CSC markers. Wnt signaling maintains CSC self-renewal; its inhibition leads to reduced sphere formation and tumor growth.
- Bmi-1 (Polycomb Ring Finger Oncogene): A transcriptional repressor and key regulator of somatic stem cells. Bmi-1 is consistently overexpressed in oral CSCs, where it represses genes like p16INK4a and p14ARF, promoting self-renewal, immortalization, and radioresistance. Knockdown of Bmi-1 impairs CSC properties *in vitro* and *in vivo* [36-46].

The Epithelial-Mesenchymal Transition (EMT) Connection

EMT is a developmental program co-opted by carcinoma cells, conferring migratory, invasive, and stem-like properties. Transcription factors driving EMT (e.g., Snail, Slug, Twist, ZEB1) are often upregulated in oral CSCs. These factors directly repress epithelial markers like E-cadherin and induce a mesenchymal phenotype, facilitating local invasion and the generation of circulating tumor cells with stem cell attributes. Thus, EMT and the CSC state are intimately linked, creating a feed-forward loop that drives metastasis.

The CSC Niche (Microenvironment)

CSCs reside in specialized microenvironments or "niches" that provide critical signals for their maintenance and protection. In OSCC, this niche includes cancer-associated fibroblasts (CAFs), tumor-associated macrophages (TAMs), mesenchymal stem cells, and extracellular matrix components. Paracrine signaling from the niche (e.g., via IL-6, TGF-β) activates STAT3, NF-κB, and other pathways in CSCs, promoting survival and therapy resistance. Hypoxia within the tumor core, mediated by HIF-1α, is a potent inducer of stemness, upregulating markers like CD44 and OCT4.

CSCs as Drivers of Therapy Resistance and Recurrence

The clinical gravity of CSCs stems from their intrinsic and acquired mechanisms of resistance to conventional cancer therapies, allowing them to survive as minimal residual disease (MRD) and seed future recurrence.

Resistance to Chemotherapy

- Enhanced Drug Efflux: CSCs overexpress ATP-binding cassette (ABC) transporter proteins like ABCB1 (MDR1/P-glycoprotein) and ABCG2. These pumps actively export a wide range of chemotherapeutic agents (e.g., doxorubicin, paclitaxel) from the cell, reducing intracellular concentrations to sub-lethal levels.
- Increased DNA Repair Capacity: CSCs exhibit elevated activity of DNA repair machinery, particularly mechanisms like homologous recombination (HR). This allows for efficient repair of DNA damage induced by platinum-based agents (cisplatin, carboplatin) and other genotoxic drugs.
- Anti-Apoptotic Mechanisms: Upregulation of anti-apoptotic Bcl-2 family proteins and inhibitors of apoptosis proteins (IAPs), coupled with downregulation of pro-apoptotic signals, creates a high apoptotic threshold.
- Quiescence: A significant proportion of CSCs reside in the G0 phase of the cell cycle. Since most conventional chemotherapies target rapidly dividing cells, these quiescent CSCs are spared from cytotoxic effects, acting as a reservoir for relapse once therapy ceases.

Resistance to Radiotherapy

- Reactive Oxygen Species (ROS) Management: CSCs maintain lower baseline levels of ROS through upregulation of free radical scavengers (e.g., glutathione). As radiation kills cells largely through the generation of ROS, this reduced oxidative state confers radioresistance.
- Enhanced DNA Damage Response: Similar to chemoresistance, CSCs efficiently activate checkpoint kinases (ATM, ATR, CHK1/2) and DNA repair pathways following radiation-induced double-strand breaks.
- Activation of Survival Pathways: Radiation can paradoxically activate pro-survival signaling like the PI3K/AKT and NF-κB pathways in CSCs, further enhancing their resistance [47-57].

The Recurrence and Metastasis Cascade

Following therapy, surviving CSCs constitute the MRD. These cells can:

1. Initiate Local Recurrence: Proliferate and regenerate a tumor at the primary site, often with a more aggressive phenotype.
2. Drive Metastasis: Undergo EMT, intravasate into circulation, and survive as circulating tumor cells (CTCs). At a distant site, a subset of these CTCs with stem-like properties can extravasate, colonize, and establish a metastatic colony, recapitulating the heterogeneity of the primary tumor. The "self-seeding" hypothesis further posits that CTCs can also return to the primary tumor, fueling its regrowth.

Clinical Correlations

Numerous clinical studies have linked the presence of CSCs, as identified by biomarker expression (e.g., high CD44, ALDH1), with worse clinicopathological features in OSCC patients: advanced TNM stage, lymph node metastasis, perineural invasion, poor differentiation, and significantly reduced disease-free and overall survival. This provides direct translational evidence of the CSC's role in aggressive disease biology.

Therapeutic Implications: Targeting the CSC Compartment

Eradicating CSCs is considered essential for achieving durable remission and cure. Strategies are moving beyond broad cytotoxicity to specifically target CSC vulnerabilities.

Targeting CSC-Specific Signaling Pathways

- Notch Inhibitors: Gamma-secretase inhibitors (GSIs, e.g., DAPT) block the release of NICD. Clinical trials of GSIs in other cancers have shown promise, though with on-target gastrointestinal toxicity. More specific anti-Notch antibodies are in development.
- Hedgehog Inhibitors: Smoothened antagonists like vismodegib and sonidegib are FDA-approved for basal cell carcinoma. Their efficacy in OSCC, particularly in combination therapy, is under investigation.
- Wnt/β-Catenin Inhibitors: Agents targeting Wnt secretion (porcupine inhibitors), receptor complexes, or β-catenin/TCF interaction are in preclinical and early clinical development. Challenges include on-target bone toxicity.

Disrupting the CSC Niche

- Targeting Hypoxia: HIF-1α inhibitors or hypoxic cell sensitizers may disrupt the protective hypoxic niche.
- Modulating the Immune Niche: Repolarizing TAMs from a pro-tumor (M2) to an anti-tumor (M1) phenotype or inhibiting CAF-derived signals (e.g., targeting IL-6/STAT3) can indirectly target CSCs.

Differentiation Therapy

Forcing CSCs to differentiate into non-tumorigenic, therapy-sensitive progeny. Retinoic acid derivatives, PPARγ agonists, and BMPs have shown potential in preclinical models to reduce stemness markers and tumorigenicity.

Immunotherapy Approaches

CSCs may express unique tumor-associated antigens or overexpress certain antigens.

- CSC-Directed CAR T-cells: Engineering T-cells to target CSC markers like CD44v6 or EGFRvIII (a variant expressed in some CSCs).
- Cancer Vaccines: Vaccines targeting antigens expressed on CSCs (e.g., ALDH1) aim to elicit an immune response against the tumor-initiating population.

Overcoming Specific Resistance Mechanisms

- ABC Transporter Inhibitors: Although early-generation inhibitors failed clinically due to toxicity and pharmacokinetic interactions, newer, more specific agents are being explored.
- DNA Repair Inhibition: Combining chemotherapy with PARP inhibitors (in tumors with HR deficiency) or CHK1 inhibitors can sensitize CSCs to DNA damage.
- Inducing CSC Proliferation: Using cytokines or growth factors to force quiescent CSCs into the cell cycle before administering cycle-specific chemotherapy.

The Combination Paradigm

The most promising strategy is the rational combination of CSC-targeting agents with conventional therapies. The goal is a two-pronged attack: conventional therapy debulks the differentiated tumor mass, while the CSC-targeted agent eliminates the regenerative reservoir, thereby preventing recurrence. This approach requires biomarker-driven patient selection to identify tumors dependent on specific CSC pathways.

Future Directions and Challenges

- Intratumoral Heterogeneity: CSC populations themselves may be heterogeneous, with different subpopulations responsible for initiation, metastasis, or specific resistance mechanisms. Single-cell RNA sequencing is shedding light on this complexity.
- Dynamic Plasticity: Non-CSCs can dedifferentiate and re-acquire stem-like properties under stress (e.g., therapy, hypoxia), blurring the hierarchical model. Targeting this plasticity is a new frontier.
- Liquid Biopsies for CSC Monitoring: Detecting CSC-derived circulating tumor cells (CTCs) or CSC-specific biomarkers in ctDNA could enable real-time monitoring of MRD and early detection of relapse, guiding adaptive therapy.
- Improved Preclinical Models: Patient-derived xenograft (PDX) models that better preserve tumor heterogeneity and the human stromal niche are essential for testing CSC-targeting strategies.

Conclusion

The cancer stem cell paradigm has fundamentally reshaped our understanding of oral squamous cell carcinoma. CSCs are not merely a biological curiosity but the central architects of tumor persistence, therapeutic failure, and lethal recurrence. Their identification through markers like CD44 and ALDH1, and their regulation by Notch, Hedgehog, and Wnt pathways, provides a molecular roadmap for intervention. Their formidable resistance to conventional therapy, rooted in quiescence, efficient DNA repair, and drug efflux, explains why current treatments often fall short of a cure.

The translational imperative is clear: to improve outcomes in OSCC, therapeutic strategies must evolve to incorporate direct targeting of the CSC compartment. While significant challenges remain including CSC plasticity, niche interactions, and intratumoral heterogeneity the development of pathway inhibitors, differentiation agents, and immunotherapies directed against CSCs holds immense promise. The future of OSCC management likely lies in combinatorial regimens that simultaneously attack the bulk tumor and its stem cell foundation. By focusing our therapeutic sights on this resilient cellular subpopulation, we move closer to the goal of transforming OSCC from a recurrent, life-threatening disease into a durable, or even curable, condition.

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