Review Article

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EXPRESSION OF STEM CELL MARKERS IN ODONTOGENIC CYSTS AND TUMORS: A NARRATIVE REVIEW

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ABSTRACT

This review aims to explore the literature on the presence and participation of stem cells in the pathogenesis and evolution of odontogenic lesions. This narrative review was carried out by searching the PubMed and Medline databases. Articles published between 2010 and 2026 that evaluated the expression of stem cell markers in different odontogenic cysts and tumors were retrieved. In most studies, the expression of stem cell markers was documented by immunohistochemistry and/or immunofluorescence, with OCT4, CD44 and SOX2 being the most commonly studied. The association of stem cells with the development and biological behavior of these pathologies remains uncertain. However, the high immunoexpression of these proteins seems to be related to the maintenance of cells with a stem-like phenotype, morphological diversity of the lesions, tumor initiation and progression, sustained growth, and the aggressiveness of odontogenic lesions.

Keywords: Odontogenic Tumor. Odontogenic Cyst. Stem Cell. Marker.

Statement of Clinical Significance

The expression of stem cell markers in odontogenic lesions strongly correlates with aggressive behavior and recurrence. Identifying these cell populations provides crucial prognostic value for surgical planning. Furthermore, stem cells represent promising targets for novel individualized therapies, optimizing patient care and reducing treatment-related morbidity.

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INTRODUCTION

Odontogenic cysts and tumors exhibit diverse clinical behaviors and morphological appearances, ranging from slow, asymptomatic growth to extensive tissue destruction. Understanding the complex development of these lesions, which arise from abnormal odontogenesis, is essential to clarify their distinct biological behaviors [1–4].

Although the mechanisms underlying odontogenic lesion development remain partially elucidated, epithelial and mesenchymal stem cells appear to be directly involved [1,5]. Since odontogenic lesions originate from embryonic epithelial and mesenchymal remnants - tissues known to be rich in stem cells - it is postulated that these cells play a critical role in their pathogenesis [5–7]. However, their specific contribution remains poorly understood, and studies addressing this topic are scarce[1,8].

Stem cells possess self-renewal and multilineage differentiation capabilities, acting as permanent resident cells that proliferate throughout life [5]. They are fundamental for regenerative medicine due to their multipotency [9]. To identify these cells in oral tissues, studies frequently analyze the immunoexpression profile of markers such as CD133, BMI1, ABCG2, CD44, SOX2, CD90, P75NTR, OCT4, NOTCH, NANOG, ALDH1 and CK5 [3,6,10].

Physiologically, stem cells reside in various dental tissues, including the pulp of permanent and deciduous teeth [11], periodontal ligament [12], gingival tissues [13], and dental follicle [14]. In pathological states, epithelial stem cells have been detected in benign odontogenic lesions [5], while mesenchymal stem cells are found in

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inflammatory conditions like periapical cysts, contributing to tissue regeneration and local immune response regulation [12,15,16].

In tumor biology, stem cell research has become crucial for understanding tumor growth, angiogenesis, progression, infiltration, and invasiveness [10,17].While some odontogenic lesions have been indicated as possible sources of stem cells, the true contribution of these cells to lesion initiation and progression requires further clarification. Therefore, this narrative review aims to explore the literature regarding the presence and role of stem cells in the pathogenesis and evolution of odontogenic lesions.

LITERATURE REVIEW

This narrative review of the literature was carried out by searching the PubMed and Medline databases. Articles published in English between 2010 and 2026 that evaluated the expression of stem cell markers in different odontogenic cysts and tumors were retrieved. This was a period of significant methodological advancements and standardization regarding the identification of stem cell markers in odontogenic lesions. Only original research articles reporting primary data on marker expression were included in the analysis, ensuring that the discussion was based exclusively on experimental and observational findings. The articles were selected using the following keywords: ‘stem cell’, ‘multipotent stem cell’, ‘odontogenic lesions’, ‘odontogenic cyst’, and ‘odontogenic tumors’.

Stem cells residing in epithelial and ectomesenchymal remnants of the tooth-forming apparatus may contribute to the development of odontogenic lesions [18–20].

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Although their presence has been documented, their differentiation potential and precise contribution to lesion progression and biological behavior remain poorly understood [5,19]. Their self-renewal and differentiation capacities may underlie the morphological diversity and sustained growth observed in some odontogenic lesions, potentially influencing their biological aggressiveness. In addition, López de Andrés et al. [21] (2020) emphasized that stem cells may modulate the tumor microenvironment through the secretion of factors involved in angiogenesis, extracellular matrix remodeling, and tumor progression.

Studies investigating stem cells in odontogenic lesions are scarce, especially those involving odontogenic cysts. The following markers have been used to demonstrate the presence of stem cells in odontogenic cysts and tumors: CD133, BMI1, ABCG2, CD44, SOX2, CD90, P75NTR, OCT4, NOTCH and ligands, NANOG, ALDH1 and CK5 [3,5–7,10,17–19,22–30](Table 1). The present study summarizes the available evidence on the expression of stem cell markers in odontogenic cysts and tumors.

One of the first studies investigating stem cell-related markers in odontogenic lesions was published in 2010 [17]. The authors investigated the expression of stem cell markers (CD133, BMI1, and ABCG2) in odontogenic lesions including ameloblastoma (AB), metastatic AB, ameloblastic carcinoma (AC), and clear cell odontogenic carcinoma, compared to control tooth germs using RT-PCR and immunohistochemistry. CD133 expression was higher in malignant tumors, suggesting an association with malignant potential, while BMI1 showed uniform expression across lesions and ABCG2 was detected in neoplastic tissues but not in

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tooth germs. The authors suggested that these markers may contribute to oncogenesis, cellular differentiation, and malignant behavior in odontogenic epithelium.

Table 1. Stem cell markers in odontogenic lesions.

Marker	Author	Lesion	Result
CD133	Kumamoto and Ohki, 2010	AB, mAB, AC, CCOC	Aberrant expression related to the malignant potential of the odontogenic epithelium
BMI1	Kumamoto and Ohki, 2010; Pagella et al., 2020	AB, mAB, AC, CCOC	Presence, maintenance, and survival of the stem cell population
ABCG2	Kumamoto and Ohki, 2010	AB, mAB, AC, CCOC	Associated with proliferation and maintenance of the stem cell population, with a possible role in tumorigenesis
CD44	Monroy et al., 2018; Martínez-Marcial et al. (2026)	OKC, AB, UAB, AOT	Related to the presence of cells with a stem-like phenotype, and sometimes associated with aggressiveness, solid and invasive growth pattern, and recurrence
SOX2	Juuri et al., 2013; Heikinheimo et al., 2015; Pagella et al., 2020; Chacham et al., 2020; Phattarataratip et al., 2021; Tseng et al., 2022; Júnior et al., 2022; Martins Balbinot et al., 2023; Pereira et al., 2023; El-Abdeen et al., 2025; Mannava et al., 2025;	RC, DC, COC, GOC, BOC, OKC, UAB, AB, AOT, OM, AF, LPC, AGC	Related to less cell differentiation of the odontogenic epithelial component and maintenance of the stem cell state Association with greater proliferative capacity (lesions with higher rates of mitosis), tumor initiation and progression, higher risk of recurrence, and involvement in epithelial-mesenchymal transition

	Martínez-Marcial et al., 2026		
CD90	Silva et al., 2016	AB and UAB	Greater aggressive potential, induced by P75NTR
P75NTR	Silva et al., 2016	AB and UAB	Greater aggressive potential, induced by CD90
OCT4	Monroy et al., 2018; Chacham et al., 2020; Phattarataratip et al., 2021; Martins Balbinot et al., 2023; Mannava et al., 2025	RC, DC, OKC, COC, AF, OM, AOT, AB	Presence and maintenance of stem cells in a less advanced state of differentiation Association with a capacity for tumor initiation, progression and survival Conversely, authors reported limited immunoreactivity and appears to play a less significant role in the pathogenesis
NOTCH and ligands	Pagella et al., 2020	AB	Related to maintenance of the stem cell state
NANOG	Chacham et al., 2020; Martins Balbinot et al., 2023	DC, OKC, AB, OM	Presence of cells with a stem-like phenotype
ALDH1	Trindade et al., 2022	OKC, DC, AB, AOT	Presence of cells in a less advanced state of differentiation
CK5	Redman et al., 2026	BOC, LPC, AGC	Potential stem cell biomarker, possibly associated with the regulation of cell proliferation and apoptosis

Legend: AB: ameloblastoma; aAB: atypical ameloblastoma; mAB: metastatic ameloblastoma; UAB: unicystic ameloblastoma; AC: ameloblastic carcinoma; DC: dentigerous cyst; BOC: botryoid odontogenic cyst; COC: calcifying odontogenic cyst; CCOC: clear cell odontogenic carcinoma; GOC: glandular odontogenic cyst; RC: radicular cyst; AF: ameloblastic fibroma; OM: odontogenic myxoma; OKC: odontogenic keratocyst; AOT: adenomatoid odontogenic tumor; LPC: lateral periodontal cysts; AGC: adult gingival cysts.

Juuri et al. [18] (2013) investigated SOX2 expression in tooth germs, AB, and experimentally induced odontoma-like tumors in mice. High expression of SOX2 was observed in AB and in the dental lamina of tooth germs, including those of supernumerary teeth. No expression was found in Hertwig's epithelial sheath or in the epithelial rests of Malassez. In the induced tumor, epithelial cells permeating the mineralized structures expressed SOX2. These cells had an arrangement resembling the epithelium of AB. The authors suggested that AB may originate from SOX2+ dental lamina cells, which retain proliferative and tumor-initiating potential. They further proposed that SOX2 may help maintain the stem cell phenotype of neoplastic epithelial cells, potentially contributing to tumor recurrence.

Heikinheimo et al. [19] (2015) characterized SOX2 expression in cases of AB and odontogenic keratocyst (OKC). SOX2 positivity was significantly higher in OKC cases compared to AB. According to the authors, this result reflects the histopathological characteristics of the epithelium in OKC whose basal and parabasal layers contain cells with stem-like properties. The authors thus suggested the possible presence of stem cells in OKC and their probable contribution to the high mitotic index, which could explain recurrence due to satellite cysts and, consequently, the more aggressive nature of this cyst.

In 2016, Silva et al. [10] analyzed the expression of CD90 and P75NTR in AB and unicystic AB, as well as the possible relationship between the expression of the proteins and the biological behavior of these tumors. Both markers were expressed in all cases, with higher levels in AB. CD90 positivity was prominent in perivascular cells near neoplastic epithelium, suggesting a role in tumor invasiveness, while

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P75NTR expression was associated with FGF secretion and odontogenic epithelial transformation. The authors suggested that differential expression of these markers may contribute to the distinct biological behavior of these lesions.

Monroy et al. [5] (2018) evaluated the expression of OCT4 and CD44 in AB, OKC and AOT. Expression of CD44 was positive in all cases studied but was significantly higher in OKC. Regarding OCT4, the findings revealed positive expression, with a similar staining in the three lesions. The authors did not find a significant correlation between OCT4 and CD44; however, the high individual expression of these markers indicates the presence of cells with a stem-like phenotype, suggesting the possible participation of stem cells in the histogenesis and differentiation of benign epithelial odontogenic lesions with variable biological behavior.

Pagella et al. [22] (2020) characterized the expression of SOX2, BMI1 and NOTCH signaling ligands and receptors in AB. Expression of *BMI1* and *SOX2*, genes involved in cellular self-renewal, was found in tumor parenchyma, suggesting that stem cells may play a role in tumor maintenance and reactivation. Gene and protein expression of NOTCH2 and NOTCH3 was associated with expression of their ligands (JAG1, DLL1, and DLL4) at the parenchyma/stroma interface. These findings suggest the existence of a population with stem-like characteristics in these tumors since Notch signaling participates in establishing the fate and behavior of stem cells, influencing maintenance of the stem cell state. The authors believe that the stem cell population and activation of Notch signaling are associated with the pathogenesis of AB and may play an important role in tumor progression.

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Chacham et al. [6] (2020) evaluated stem cell markers (NANOG, SOX2, OCT4, CD34) within the connective tissue of odontogenic cysts and tumors. Except for OCT4, which was unexpressed, all markers were detected, with NANOG exhibiting the highest positivity, followed by SOX2 and CD34. Notably, SOX2 and CD34 expression was significantly higher in tumors compared to cysts. The authors attributed the uniform NANOG expression to potential antigen-antibody specificity issues rather than true biological equivalence. Ultimately, these findings confirm that cyst capsules and tumor stroma harbor cells that express stem cell markers. The differential expression of SOX2 suggests involvement in cellular differentiation and epithelial-mesenchymal transition (EMT), though its precise contribution to tumor aggressiveness warrants further investigation.

Phattarataratip et al. [7] (2021) contributed to the characterization of stem cell markers in odontogenic lesions that had not yet been studied, including calcifying odontogenic cyst (COC) and ameloblastic fibroma (AF). The authors analyzed the differential expression of SOX2 and OCT4 in cysts (DC, OKC, COC) and tumors (AB, AOT, AF). There was higher expression of SOX2 in odontogenic epithelium of OKC and AF when compared to AB, COC and DC, which exhibited minimal expression. No expression of this protein was observed in AOT. Regarding OCT4, there were no significant differences in expression. The authors attributed the variation in the SOX2 staining profile to the degree of differentiation of the odontogenic epithelium during histogenesis and development of the lesions. The higher expression of SOX2 in OKC and AF suggests that these lesions possess a higher stem cell potential.

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Tseng et al. [23] (2022) analyzed SOX2 and Ki-67 expression in primary and recurrent AB. Both markers were expressed across all cases, with higher levels in the plexiform variant. Although marker expression lacked correlation with broad clinicopathological features, recurrent AB in the same patient exhibited higher SOX2 expression compared to the primary tumor but there were no differences in Ki-67 expression. Furthermore, SOX2 silencing reduced tumor cell viability in vitro, and its expression was higher in BRAF (V600E)-mutated tumors. The authors suggested that combined BRAF and SOX2 inhibition may represent a potential therapeutic strategy for recurrent and unresectable AB.

In 2022, Júnior et al. [24] evaluated EGFR, cyclin D1, and SOX2, proteins associated with cellular proliferation and cystic expansion, in the epithelium of OKC, botryoid odontogenic cysts (BOC), and glandular odontogenic cysts (GOC). EGFR was expressed in all lesions, while cyclin D1 showed prominent expression in OKC and GOC. SOX2 expression was restricted to the suprabasal layer of OKC epithelium. The authors suggested that EGFR and cyclin D1 contribute to epithelial proliferation, whereas SOX2 may indicate stem cell populations associated with the growth and biological behavior of OKC.

Trindade et al. [3] (2022) reported morphological and behavioral diversity of odontogenic cysts and tumors that might be explained by the presence of cells with stem-like characteristics. Within this context, the authors investigated the expression of ALDH1 in OKC, DC, AB, and AOT in order to provide data that could contribute to the understanding of the variable behavior of these lesions. Positivity was found in all cases analyzed; however, scores were higher in cystic lesions compared to odontogenic tumors. According to the authors, this finding can be explained by the

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process of cell differentiation observed, tumors exhibit cellular and molecular characteristics that resemble the differentiated cells, while cysts would show more undifferentiated cells.

Martins Balbinot et al. [25] (2023) investigated the expression of SOX2, NANOG and OCT4 in AB. Expression of the markers was observed in all paraffin-embedded specimens and in a human AB cell line. Higher immunostaining of SOX2, NANOG and OCT4 was found in AB compared to controls, which was more expressive in epithelial cords and islands in relation to the stromal component. The authors thus suggested the possible presence and participation of tumor stem cells in the initiation, progression, and recurrence of AB. In addition, they highlighted the need for new studies focusing on the suppression of stem cell markers in order to evaluate the influence of this blockade.

In the same year, Pereira et al. [26] (2023) evaluated the differential expression of SOX2 between OKC and AB. The authors found high immunopositivity in OKC, while AB exhibited no or low expression. The high expression observed in OKC indicates the presence of cells with stem-like characteristics, including self-renewal and proliferative capacity, which influence the biological behavior of this lesion. The authors proposed a possible association of SOX2 with the Hh signaling pathway. In contrast, the low or absent expression of SOX2 in AB suggests that the neoplastic and aggressive behavior of this tumor may be related to other molecular pathways.

El-Abdeen et al. [27] (2025) conducted a prospective immunohistochemical study investigating SOX2 as a prognostic cancer stem cell marker in AB, OKC, and DC, aiming to correlate its immunohistochemical scores with clinical and radiographic findings, as well as the occurrence of recurrence. The results revealed

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strong SOX2 immunoexpression in OKCs, followed by AB, whereas DC exhibited low scores in most cases. Furthermore, for both lesions a significant positive correlation was identified between SOX2 expression and cortical bone perforation, as well as lesion recurrence. The authors concluded that SOX2 is a reliable cancer stem cell marker, whose overexpression is directly associated with aggressive biological behavior, bone invasiveness, and recurrence risk, making it a relevant prognostic tool for the clinical and surgical management of these lesions.

Mannava et al. [28] (2025) compared the immunoexpression of the epithelial stem cell markers SOX2 and OCT4 in primary OKCs, recurrent OKCs, and OKCs treated with marsupialization. The results demonstrated that SOX2 was expressed in most cases, with lower expression levels in OKCs treated with decompression. In contrast, OCT4 expression was limited, with greater expression observed in recurrent cases. According to the authors, high SOX2 expression is associated with the presence of cells exhibiting a stem-like characteristic and may be an indicator of OKC aggressiveness and recurrence. Meanwhile, OCT4 appears to play a less significant role in the pathogenesis of this lesion.

In a recent study, Martínez-Marcial et al. [29] (2026) investigated the immunoexpression of SOX2 and CD44, as well as the gene expression of EMT markers (vimentin – VIM, alpha-smooth muscle actin – ACTA2, and FGFR1) in different histological subtypes of AB (conventional, unicystic, and peripheral), using dental follicle tissue as a control. SOX2 and CD44 expression was significantly higher in conventional and unicystic ameloblastomas (UAB), whereas VIM, ACTA2, and FGFR1 showed an increased expression trend in the unicystic subtype. The authors suggested that SOX2 and CD44 are associated with stem-like cell

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populations and the invasive growth pattern of conventional AB, while EMT-related mechanisms may contribute to cystic cavity formation in the unicystic subtype.

Finally, Redman et al. [30] (2026) investigated whether SOX2 protein and the cytokeratins CK5, CK8, and CK18 act as stem cell biomarkers in BOC, lateral periodontal cysts (LPC), and adult gingival cysts (AGC) and OKC. The results demonstrated that only CK5 and SOX2 exhibited staining patterns consistent with stem cell biomarkers (staining concentrated in the basal layer, with reduced or absent expression in the more superficial layers) across all lesions studied. In contrast, CK8 and CK18 did not exhibit expression patterns consistent with a stem cell phenotype. The authors concluded that CK5 and SOX2 are potential stem cell biomarkers in odontogenic cyst epithelium, possibly associated with the regulation of cell proliferation and apoptosis.

Studies investigating SOX2 have reported heterogeneous expression patterns in odontogenic lesions, with contrasting findings between AB and OKC. While some studies demonstrate SOX2 expression in AB [18,22,23,26,27,29], others report higher immunoexpression in OKC [7,24,26–28]. These differences may reflect variations in study design, cellular compartments, and histological subtypes. In AB, SOX2 expression has been associated with tumor recurrence and specific molecular features [23,27,29]. Whereas in OKC, it is predominantly detected in basal and suprabasal epithelial layers, including recurrent lesions [28]. Notably, recent evidence has also associated higher SOX2 expression, with cortical bone perforation and recurrence, suggesting its potential relevance as a marker of aggressive biological behavior in odontogenic lesions [27]. Overall, these findings highlight the context-

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dependent expression of SOX2 and its potential contribution to understanding the biological behavior of odontogenic lesions.

CD44 and OCT4 exhibit distinct expression patterns across odontogenic lesions. Monroy et al. [5] (2018) reported higher CD44 immunoexpression in OKC compared with AB and AOT, whereas recent findings demonstrated increased expression in conventional and UAB compared with controls and peripheral variants [29]. These findings suggest that CD44 expression varies according to lesion type and histological subtype, potentially reflecting differences in stem-like cell populations. In contrast, OCT4 shows restricted and variable immunoreactivity, with low expression in primary and recurrent OKCs and absence in decompressed lesions [6,7,28].Overall, these findings suggest that CD44 exhibits a broader association with odontogenic lesion phenotypes, whereas OCT4 displays a more restricted and variable expression pattern, highlighting their distinct expression patterns in the stemness profile of odontogenic lesions.

Therefore, stemness-associated markers in odontogenic lesions have been predominantly investigated through immunohistochemistry and immunofluorescence, particularly CD44, SOX2, and OCT4 in AB and OKC. However, marker expression or coexpression alone does not confirm the presence of functional stem cells. Moreover, studies on malignant odontogenic tumors remain scarce, limiting the understanding of stemness-associated phenotypes in these lesions.

CONCLUSION

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The association of stem-like cells with the development and biological behavior of these pathologies remains uncertain. However, the high immunoexpression of these proteins seems to be related to the maintenance of cells with a stem-like phenotype, histological heterogeneity of the lesions, tumor initiation and progression, sustained growth, and the aggressiveness of odontogenic lesions. Although they represent an interesting avenue of research, current evidence is predominantly descriptive and based on marker expression, limiting functional interpretations and clinical applications. Further functional and preclinical studies are therefore needed to clarify the biological significance of these cell populations and validate their potential as therapeutic targets.

Author's Contributions

Larissa Abbehusen Couto: Conceptualization, Methodology, Formal analysis, Investigation, Writing – Original Draft, Writing – Review & Editing. **Lorena Vieira Sacramento:** Visualization, Writing – Original Draft, Writing – Review & Editing. **Ianna Josefa Valeska de Aniz Castro:** Writing – Original Draft, Writing – Review & Editing. **Vildeman Rodrigues de Almeida Júnior:** Supervision, Writing – Review & Editing. **Daiana Cristina Pereira Santana:** Supervision, Writing – Review & Editing. **Águida Cristina Gomes Henriques:** Conceptualization, Data curation, Formal analysis, Methodology, Project administration, Supervision, Writing – Review & Editing. All authors actively revised and approved the final version of the manuscript.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

REFERENCES

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1. Kalogirou EM, Lekakis G, Petroulias A, Chavdoulas K, Zogopoulos VL, Michalopoulos I, et al. The Stem Cell Expression Profile of Odontogenic Tumors and Cysts: A Systematic Review and Meta-Analysis. Genes (Basel). 2023 Sep 1;14(9). doi:10.3390/GENES14091735 PubMed PMID: 37761874.

2. Mamabolo M, Noffke C, Raubenheimer E. Odontogenic tumours manifesting in the first two decades of life in a rural African population sample: A 26 year retrospective analysis. Dentomaxillofacial Radiology. 2011 Sep;40(6):331–7. doi:10.1259/dmfr/54585925 PubMed PMID: 21831971.

3. da Trindade GA, da Silva LP, de Andrade Santos PP, Pinto LP, de Souza LB. Expression of a Tumor Stem Cell Marker (Aldehyde Dehydrogenase 1-ALDH1) in Benign Epithelial Odontogenic Lesions. Head Neck Pathol. 2022 Mar 29;16(3):785–91. doi:10.1007/s12105-022-01430-z

4. Duarte-Andrade FF, Vitório JG, Pereira T dos SF, Gomes CC, Gomez RS. A review of the molecular profile of benign and malignant odontogenic lesions. Oral Surg Oral Med Oral Pathol Oral Radiol. 2020 Apr;129(4):357–68. doi:10.1016/j.oooo.2019.12.017

5. Monroy EAC, de Andrade Santos PP, de Sousa Lopes MLD, Mosqueda-Taylor A, Pinto LP, de Souza LB. Oct-4 and CD44 in epithelial stem cells like of benign odontogenic lesions. Histochem Cell Biol. 2018;150(4):371–7. doi:10.1007/s00418-018-1692-7 PubMed PMID: 29971493.

6. Chacham M, Almoznino G, Zlotogorski-Hurvitz A, Buchner A, Vered M. Expression of stem cell markers in stroma of odontogenic cysts and tumors. Journal of Oral Pathology and Medicine. 2020;00:1–10. doi:10.1111/jop.13102 PubMed PMID: 32840915.

7. Phattarataratip E, Panitkul T, Khodkaew W, Anupuntanun P, Jaroonvechatam J, Pitarangsikul S. Expression of SOX2 and OCT4 in odontogenic cysts and tumors. Head Face Med. 2021 Dec 14;17(1):29. doi:10.1186/s13005-021-00283-1

8. Đokić J, Tomić S, Cerović S, Todorović V, Rudolf R, Čolić M. Characterization and immunosuppressive properties of mesenchymal stem cells from periapical lesions. J Clin Periodontol. 2012 Sep 8;39(9):807–16. doi:10.1111/j.1600-051X.2012.01917.x

9. Paduano F, Marrelli M, Palmieri F, Tatullo M. CD146 Expression Influences Periapical Cyst Mesenchymal Stem Cell Properties. Stem Cell Rev Rep. 2016 Oct 1;12(5):592–603. doi:10.1007/s12015-016-9674-4 PubMed PMID: 27406247.

10. Silva FPY, Dias A, Coelho CA, Guerra EN, Marques AEM, Decurcio D de A, et al. Expression of CD90 and P75NTR stem cell markers in

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ameloblastomas: a possible role in their biological behavior. Braz Oral Res. 2016;30(1):e109. doi:10.1590/1807-3107BOR-2016.vol30.0109 PubMed PMID: 27737362.

11. Sharpe PT. Dental mesenchymal stem cells. Development (Cambridge) [Internet]. Company of Biologists Ltd; 2016 [cited 2020 Nov 30]. p. 2273–80. Available from: https://pubmed.ncbi.nlm.nih.gov/27381225/ doi:10.1242/dev.134189 PubMed PMID: 27381225.

12. Couto de Carvalho LA, Tosta Dos Santos SL, Sacramento LV, de Almeida VRJ, de Aquino Xavier FC, Dos Santos JN, et al. Mesenchymal stem cell markers in periodontal tissues and periapical lesions. Acta Histochem. 2020 Oct;122(8):151636. doi:10.1016/j.acthis.2020.151636 PubMed PMID: 33132168.

13. Huang GTJ, Gronthos S, Shi S. Mesenchymal Stem Cells Derived from Dental Tissues *vs* . Those from Other Sources: Their Biology and Role in Regenerative Medicine. J Dent Res. 2009 Sep 18;88(9):792–806. doi:10.1177/0022034509340867

14. Morsczeck C, Götz W, Schierholz J, Zeilhofer F, Kühn U, Möhl C, et al. Isolation of precursor cells (PCs) from human dental follicle of wisdom teeth. Matrix Biology. 2005;24:155–65. doi:10.1016/j.matbio.2004.12.004 PubMed PMID: 15890265.

15. Marrelli M, Paduano F, Tatullo M. Cells isolated from human periapical cysts express mesenchymal stem cell-like properties. Int J Biol Sci. 2013;9(10):1070–8. doi:10.7150/ijbs.6662 PubMed PMID: 24250252.

16. Estrela C, Freitas Silva BS, Silva JA, Yamamoto-Silva FP, Pinto-Júnior D dos S, Gomez RS. Stem Cell Marker Expression in Persistent Apical Periodontitis. J Endod. 2017;43(1):63–8. doi:10.1016/j.joen.2016.09.002 PubMed PMID: 27847139.

17. Kumamoto H, Ohki K. Detection of CD133, Bmi-1, and ABCG2 in ameloblastic tumors. J Oral Pathol Med. 2010 Jan;39(1):87–93. doi:10.1111/J.1600-0714.2009.00807.X PubMed PMID: 19659474.

18. Juuri E, Isaksson S, Jussila M, Heikinheimo K, Thesleff I. Expression of the stem cell marker, SOX2, in ameloblastoma and dental epithelium. Eur J Oral Sci. 2013 Dec;121(6):509–16. doi:10.1111/eos.12095 PubMed PMID: 24148099.

19. Heikinheimo K, Kurppa KJ, Laiho A, Peltonen S, Berdal A, Bouattour A, et al. Early dental epithelial transcription factors distinguish ameloblastoma from keratocystic odontogenic tumor. J Dent Res. 2015;94(1):101–11. doi:10.1177/0022034514556815 PubMed PMID: 25398365.

In Press

20. Kumar A, Bhanja A, Bhattacharyya J, Jaganathan BG. Multiple roles of CD90 in cancer. Tumor Biology. 2016 Sep 23;37(9):11611–22. doi:10.1007/s13277-016-5112-0

21. López de Andrés J, Griñán-Lisón C, Jiménez G, Marchal JA. Cancer stem cell secretome in the tumor microenvironment: a key point for an effective personalized cancer treatment. J Hematol Oncol. 2020 Dec 15;13(1):136. doi:10.1186/s13045-020-00966-3

22. Pagella P, Catón J, Meisel C, Mitsiadis T. Ameloblastomas Exhibit Stem Cell Potential, Possess Neurotrophic Properties, and Establish Connections with Trigeminal Neurons. Cells. 2020 Mar 6;9(3). doi:10.3390/CELLS9030644 PubMed PMID: 32155948.

23. Tseng CH, Lu PH, Wang YP, Chang JYF. Enrichment of SOX2-Positive Cells in BRAF V600E Mutated and Recurrent Ameloblastoma. J Pers Med. 2022 Jan 8;12(1):77. doi:10.3390/jpm12010077

24. Júnior JF, de França GM, da Silva Barros CC, Felix FA, da Silva WR, de Lucena HF, et al. Biomarkers involved in the proliferation of the odontogenic keratocyst, glandular odontogenic cyst and botryoid odontogenic cyst. Oral Maxillofac Surg. 2022 Jan 21;26(4):655–62. doi:10.1007/s10006-021-01026-x

25. Martins Balbinot K, Almeida Loureiro FJ, Chemelo GP, Alves Mesquita R, Cruz Ramos AMP, Ramos RTJ, et al. Immunoexpression of stem cell markers SOX-2, NANOG AND OCT4 in ameloblastoma. PeerJ. 2023 Jan 13;11:e14349. doi:10.7717/peerj.14349

26. Pereira T, Shetty SJ, Punjabi V, Vidhale RG, Gotmare SS, Kamath P. Immunohistochemical expression of SOX2 in OKC and ameloblastoma: A comparative study. Journal of Oral and Maxillofacial Pathology. 2023;27(4):685–92. doi:10.4103/jomfp.jomfp_265_23

27. Zeen El-Abdeen HM, Tawfik MAM, Elnagdy SY, Mansour NA. Evaluation of the prognostic role of SOX2 as a tumor stem cell marker in odontogenic cysts and tumors: clinical, radiographic and immunohistochemical correlation. BMC Oral Health. 2025 Dec 1;25(1). doi:10.1186/S12903-025-07065-0 PubMed PMID: 41176605.

28. Mannava C, Manyam R, Kumar NV, Puvvada DNL, Swetha P, Supriya N. Immunohistochemical expression of SOX2 and OCT4 in primary odontogenic keratocyst, recurrent odontogenic keratocyst, and odontogenic keratocyst treated by the decompression technique. Journal of Oral and Maxillofacial Pathology. 2025 Apr;29(2):286–92. doi:10.4103/jomfp.jomfp_254_24

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29. Martínez-Marcial LA, Ramírez-Jarquín JO, Villanueva-Sánchez FG, Portilla-Robertson J, Ramírez-Martínez CM, Trejo-Remigio DA, et al. Exploratory Expression Analysis of Stem Cell and Epithelial–Mesenchymal Transition Markers in Ameloblastoma. International Journal of Molecular Sciences 2026, Vol 27, Page 4645. 2026 May 21;27(10):4645. doi:10.3390/IJMS27104645 PubMed PMID: 42196620.

30. Redman RS, Basile JR, Castle JT. SOX2 and cytokeratins 5, 8 and 18 as potential stem cell biomarkers in botryoid odontogenic, lateral periodontal and gingival cysts. Histol Histopathol. 2026;41(9):1515–26. doi:10.14670/HH-25-068 PubMed PMID: 41889346.

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