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Review Article

Interaction between sclerostin and mast cells in the fibro-osseous lesions of the jaw – A systematic review.

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Abstract

Background
Fibro-osseous lesions (FOLs) of the jaw, including fibrous dysplasia and ossifying fibromas, are characterized by replacement of normal bone with fibrous tissue and abnormal mineralization. Sclerostin, a negative regulator of bone formation and mast cells involved in fibrosis and inflammation, has recently been implicated in FOL pathogenesis. Understanding their interaction may improve knowledge of lesion biology and identify potential diagnostic or therapeutic targets.

Materials and methods
This systematic review was conducted in accordance with the PRISMA guidelines. PubMed, Scopus, and Web of Science databases were searched up to July 2025. Original studies evaluating sclerostin and/or mast cell expression in jaw FOLs were included. Extracted data included study design, lesion subtype, methodology, and findings related to sclerostin–mast cell interactions.

Results
Five studies were included. Evidence consistently demonstrated abundant sclerostin-positive cells within the fibrous stroma of FOLs, and these cells tend to co-express tryptase, a mast cell marker. Surprisingly, dominant sclerostin-positive mast cells were present in fibrous dysplasia and ossifying fibroma tissues. β-catenin expression in fibrous tissue suggested potential interaction between Wnt signaling and mast cell-secreted sclerostin. Control bone tissue revealed sclerostin predominantly in osteocytes with minimal mast cell presence.

Conclusion
Current evidence supports a significant interaction between mast cells and sclerostin in jaw FOLs, potentially contributing to abnormal bone remodeling and fibrogenesis.

Future research
Further multicenter and mechanistic studies are required to clarify the biological role, prognostic value, and therapeutic potential of this interaction.

Keywords: *Fibro-osseous lesions, Sclerostin, Mast cells, Jaw, β-catenin, Bone remodeling, Immunohistochemistry*
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Introduction

Fibro-osseous lesions (FOLs) of the jaws represent a group of benign bone disorders characterized by replacement of normal lamellar bone by variably cellular fibrous connective tissue with interspersed irregular deposits of mineralized material.[1] Fibrous dysplasia (FD), ossifying fibroma (OF), and juvenile ossifying fibroma (JOF) are some of the most clinically relevant subtypes. Each has distinct epidemiological patterns, anatomical predilections, radiographic appearances, and patterns of biological activity.[2] For instance, fibrous dysplasia is typically painless swelling with ill-defined radiographic borders, primarily in adolescents and young adults, whereas ossifying fibromas are usually well-defined, expansile maxillary or mandibular lesions, and juvenile lesions can be aggressively growing. Their clinical significance is discernible from their potential for facial deformity, impaired function, and, although rare, recurrence following surgical intervention.[3,4] Despite progress in clinicopathologic staging, the precise molecular and cellular mechanisms of FOL development, growth, and recurrence remain poorly understood.

Within the microenvironment of the bone, there exists a fine balance between osteoclasts, which resorb bone, osteoblasts, which form bone, osteocytes, and a range of stromal and immune cells. Destabilization of this balance contributes to FOL pathogenesis.[5] Sclerostin, a secreted glycoprotein product of the SOST gene, is best recognized as a negative regulator of bone formation.[6] Sclerostin is secreted primarily by osteocytes and inhibits osteoblastic activity through disruption of the canonical Wnt/β-catenin pathway, leading to reduced bone deposition and increased bone resorption.[7] While sclerostin's physiologic expression is necessary for skeletal homeostasis, its aberrant or ectopic expression has increasingly been described in diverse bone tumors and tumor-like lesions, including FOLs, indicating a role for this protein in dysregulating bone turnover and matrix architecture.[7,8]

Mast cells, traditionally associated with allergy and immune monitoring, are increasingly known to possess complex roles in fibrosis, tissue repair, angiogenesis, and the regulation of bone remodeling.[9] These tissue-resident immune cells are usually found in higher numbers within the stroma of FOLs compared to normal bone and have the potential to alter the microenvironment by secreting a variety of heterotypic cytokines, growth factors, and proteases.[10] Specifically, mast cells have also been implicated in matrix remodeling and regulation of osteoclast and osteoblast function. Their occurrence within FOLs suggests an underestimated role in cellular crosstalk that is accountable for pathological tissue architecture and mineralization.[11]

Currently, robust immunohistochemical data have been reported demonstrating a direct correlation between mast cells and sclerostin expression within the fibrous stroma of jaw FOLs. These findings favor the consideration that mast cells may be a non-osteocytic, alternative source of sclerostin within the lesion's microenvironment that might further suppress bone formation and promote persistence or exacerbation of fibrous tissue. This systematic review critically evaluates the literature on sclerostin-mast cell interactions in jaw FOLs, highlighting their biological, diagnostic, and therapeutic importance in the context of contemporary maxillofacial pathology.

Materials and methods

This review was conducted in strict accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines[12] (Figure 1). As this review did not involve any new studies with human or animal subjects performed by the authors, ethical approval and patient consent were not required. All included studies were published peer-reviewed articles presumed to adhere to appropriate ethical standards.

Search strategy

A comprehensive literature search was conducted in PubMed, Scopus, and Web of Science databases, covering studies published up to July 2025. Search terms included: "fibro-osseous lesions," "fibrous dysplasia," "ossifying fibroma," "sclerostin," "SOST," "mast cells," "tryptase," "jaw," "maxilla," and "mandible."

Inclusion criteria

- Original studies in English
- Assessment of sclerostin and/or mast cell expression in FOLs of the jaw
- Use of immunohistochemistry, histology, or molecular methods

Exclusion criteria

- Case reports, conference abstracts, or editorials
- Studies focusing on non-jaw FOLs
- Studies not directly addressing both sclerostin and mast cell expression

Data extraction and quality assessment

Two independent reviewers screened all records and extracted data on study design, lesion type, sample size, methodology, and principal findings relating to sclerostin

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and mast cell expression and interaction. Differences were resolved by consensus.

Study risk of bias assessment

The methodological quality and risk of bias of the included studies were independently assessed by two reviewers using the Joanna Briggs Institute (JBI) critical appraisal tools appropriate for each study design. Narrative reviews were evaluated qualitatively for relevance and methodological transparency. Any disagreements between reviewers were resolved through discussion and consensus. Due to the limited number of eligible studies and the predominance of descriptive observational data, the risk of bias assessment was primarily used to support the interpretation of findings rather than quantitative exclusion.

Effect measures

As the included studies were heterogeneous in design, methodology, and outcome reporting, quantitative synthesis and pooled effect estimates were not feasible. Therefore, findings were summarized descriptively. Outcomes of interest included qualitative assessment of sclerostin expression, mast cell distribution, tryptase positivity, and β-catenin localization within fibro-osseous lesions.

Synthesis methods

Data extracted from individual studies were organized into standardized evidence tables, including study design, lesion subtype, methodology, sample size, and principal findings. Due to substantial methodological heterogeneity and the absence of uniform quantitative outcome measures, a narrative synthesis approach was adopted. Missing or incompletely reported numerical data were described qualitatively where appropriate. Tabular presentation was used to facilitate comparison of study characteristics and findings across the included literature.

Sensitivity analysis

Formal sensitivity analysis was not performed because no meta-analysis or quantitative pooling of data was undertaken. However, consistency of findings across studies was evaluated qualitatively during narrative synthesis.

Reporting bias assessment

Assessment of reporting bias was limited because of the small number of included studies and the absence of a quantitative synthesis. To minimize selection bias, multiple electronic databases were searched, and predefined inclusion and exclusion criteria were applied during study selection.

Certainty assessment

A formal certainty of evidence assessment using GRADE methodology was not feasible because the included studies consisted primarily of descriptive and observational evidence without comparable quantitative outcomes. The certainty of evidence was therefore interpreted qualitatively based on methodological consistency, reproducibility of findings, and overall study quality.

Results

Out of all records identified, five studies fulfilled the inclusion criteria for qualitative synthesis.

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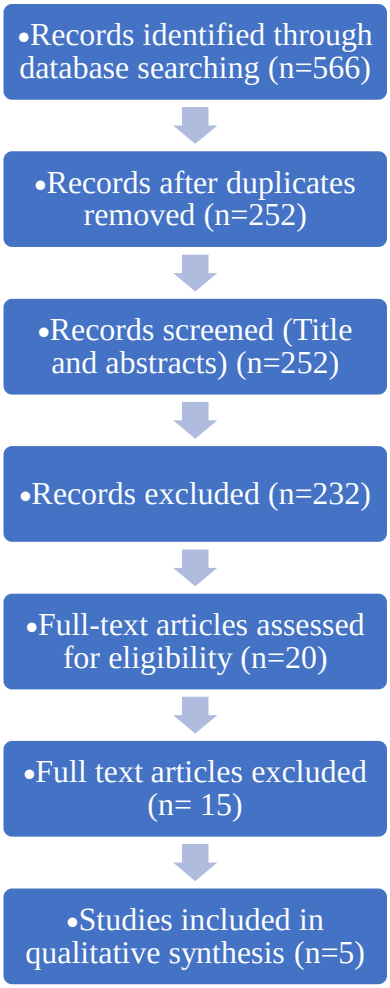

Figure 1: PRISMA flowchart

Study selection

The PRISMA flow diagram illustrating the study selection process is presented as Figure 1. A total of 566 records were identified through database searching. After duplicate removal and screening, five studies fulfilled the eligibility criteria and were included in the qualitative synthesis.

Risk of bias in included studies

Risk of bias assessment demonstrated that most included studies showed moderate methodological quality. The primary limitations identified were small sample sizes, retrospective study designs, lack of standardized outcome reporting, and absence of longitudinal clinical follow-up. The immunohistochemical studies by Schulz et al.[13] and Inagaki et al.[15] demonstrated comparatively lower risk of bias due to clearly defined methodologies and lesion characterization. Narrative reviews included in the synthesis were considered supportive background evidence and were not weighted equally with primary observational studies.

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Table 1. Summary table of included studies

Author (Year)	Study	Lesion Types	Methods	Sample Size	Key Findings
Schulz et al. (2024)[13]	Interaction Between Sclerostin and Mast Cells in Fibro-Osseous Lesions of the Jaws	FD, JTOF, PsOF, FOL	IHC (SOST, tryptase, β-catenin)	46 biopsies, 38 patients	Sclerostin-positive cells in fibrous tissue, 81% tryptase+ (mast cells), β-catenin cytoplasmic in fibrous stroma
Pick et al. (2022)[14]	Clinical, Radiological and Pathological Diagnosis of Fibro-Osseous Lesions of the Oral and Maxillofacial Region: A Retrospective Study.	FOLs (retrospective)	Clinical-pathological	Not specified	Fibrous tissue cellularity supports immune–bone crosstalk
Inagaki et al. (2016)[15]	Sclerostin expression in bone tumours and tumour-like lesions.	Bone tumors, tumor-like lesions	IHC, tissue arrays	Multiple	Increased sclerostin in FOLs vs. non-lesional bone
Dreyer et al. (2023)[16]	Novel insights into the effect of sclerostin on bone and other organs.	Jaw lesions (review)	Narrative	-	Immune cell involvement, including mast cells in the lesion stroma
Nelson et al. (2019)[17]	Benign Fibro-Osseous Lesions of the Head and Neck.	FOLs (review)	Narrative	-	Clinical and molecular overview of FOLs mentions sclerostin and mast cells.

Table 2. Risk of bias assessment of included studies

Study	Study Design	Main Limitations	Overall Risk of Bias
Schulz et al. (2024)[13]	Immunohistochemical observational study	Limited sample size, absence of functional validation	Moderate
Pick et al. (2022)[14]	Retrospective study	Lack of molecular correlation data	Moderate
Inagaki et al. (2016)[15]	Tissue expression study	Limited lesion-specific subgroup analysis	Moderate
Dreyer et al. (2023)[16]	Narrative review	Non-systematic methodology	High
Nelson et al. (2019)[17]	Narrative review	Descriptive evidence only	High

Results of individual studies

The principal findings of the included studies are summarized in Table 1. Quantitative effect estimates, such as risk ratios or mean differences, could not be calculated because the included studies primarily reported descriptive immunohistochemical and observational findings without standardized numerical outcome measures. Schulz et al.[13] demonstrated that approximately 81% of sclerostin-positive stromal cells co-expressed tryptase, indicating mast cell origin. Inagaki et al.[15] reported increased sclerostin expression in fibro-osseous lesions compared to non-lesional bone tissue. Pick et al.[14] identified increased stromal cellularity and altered extracellular matrix characteristics in fibro-osseous lesions. The review studies by Dreyer et al.[16] and Nelson et al.[17] supported the role of immune-

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mediated stromal interactions in fibro-osseous lesion biology.

Results of synthesis

Narrative synthesis demonstrated consistent evidence supporting increased sclerostin expression within the fibrous stroma of jaw fibro-osseous lesions, particularly in association with mast cells. Across the included studies, fibrous dysplasia and ossifying fibroma showed similar immunohistochemical patterns involving sclerostin positivity and stromal mast cell accumulation. The contributing studies were methodologically heterogeneous and predominantly descriptive, limiting quantitative comparability. Nevertheless, the findings showed reasonable consistency despite a moderate risk of bias.

Sensitivity analysis

Formal sensitivity analysis was not feasible because quantitative meta-analysis was not performed. Qualitative comparison of findings after exclusion of narrative reviews did not substantially alter the overall interpretation, as the principal conclusions were primarily derived from the observational immunohistochemical studies.

Reporting biases

Assessment of reporting bias was limited by the small number of eligible studies and the absence of registered protocols in several included studies. Although publication bias could not be formally evaluated, comprehensive database searching and predefined eligibility criteria were used to reduce selection bias.

Certainty of evidence

The certainty of evidence for the association between mast cells and sclerostin expression in fibro-osseous lesions was considered low to moderate. This grading was based on the observational nature of the included studies, limited sample sizes, methodological heterogeneity, and absence of mechanistic or longitudinal clinical data. However, the consistency of immunohistochemical findings across studies strengthened confidence in the observed association.

Discussion

The findings of this systematic review presented a unifying theme among the recent studies: mast cells are a significant and potentially previously underestimated source of sclerostin in the fibrous stroma of FOLs of the jaw. Schulz et al. (2024)[13] had direct immunohistochemical evidence for this association, confirming that more than 80% of sclerostin-positive cells in FOLs were also tryptase-positive, a classic mast cell marker. This unambiguous co-localization indicated that mast cells and not osteocytes in isolation were potentially contributing to the production of sclerostin in the lesional microenvironment. These findings were corroborated across a range of FOL subtypes, including fibrous dysplasia and juvenile ossifying fibromas, suggesting an overlap of pathogenic mechanisms in disparate histological entities.

Corroborating these findings, Inagaki et al. (2016)[15] also reported elevated levels of sclerostin expression in fibro-osseous tumor-like lesions compared to non-lesional bone. However, while Inagaki et al. obtained evidence that sclerostin is upregulated in FOLs, the study in their report failed to clearly indicate the cellular source. Schulz et al.[13] thus extended these prior observations by presenting definitive proof that mast cells are a major source of this increase, specifically in the fibrous stroma. This variation is clinically important because it highlights the complexity of the cellular interaction that regulates matrix turnover and dysplastic bone formation within the lesions.

Furthermore, β-catenin expression, as documented by Schulz et al. (2024)[13], was seen to be diffusely distributed within the cytoplasm of fibrous tissue cells. This is interesting since it alludes to a dysregulation of the Wnt/β-catenin signaling pathway, which is inhibited by sclerostin. The abnormal activation of Wnt/β-catenin in the context of mast cell-derived sclerostin would thus create a pathological microenvironment of sustained fibrous proliferation and inadequate mineralization, further corroborating the mechanistic connection between these molecules in FOL pathogenesis.

The pathological and clinical findings of Pick et al. (2022)[14] supplemented these molecular observations by detailing aberrant stromal cellularity and extracellular matrix in FOLs, which could be the downstream effect of the sclerostin–mast cell axis. The narrative overviews by Nelson et al. (2019)[17] and Dreyer et al. (2023)[16] provided additional context, confirming the role of immune cells, most notably mast cells, in the stroma of jaw lesions and highlighting the growing importance of molecular markers like sclerostin in their diagnosis and classification.

Together, these studies validate a model in which mast cells not only play a role in the fibrotic environment of FOLs by releasing mediators but also have a profound impact on bone remodeling by acting as an alternative source of sclerostin. With this dual function, mast cells

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find themselves centrally located in both the immune response and tissue homeostasis in the etiology of FOLs.

Clinical and diagnostic implications

From a clinical perspective, immunohistochemical identification of sclerostin and mast cell markers can aid in the diagnosis of FOLs and may potentially inform prognostication, especially in ambiguous cases. Moreover, the unique mast cell-sclerostin interaction may provide a rationale for the development of targeted therapies in recalcitrant or aggressive lesions.

Limitations

Despite these insights, significant limitations exist:
The available evidence is derived primarily from small, retrospective studies.
There is a lack of functional and mechanistic data clarifying the biological impact of the sclerostin-mast cell axis.
Clinical correlations such as prognosis, recurrence risk, and therapeutic response have not been thoroughly investigated.

Future directions

Further research should prioritize:
Functional in vitro and in vivo studies to delineate the mechanistic contributions of mast cell-derived sclerostin in FOLs.
Prospective, multicenter studies to confirm the clinical significance of sclerostin-mast cell interactions.
Exploration of targeted molecular therapies aimed at modulating this interaction in FOL management.

Conclusion

This systematic review indicated a strong correlation between sclerostin and mast cells in the fibrous stroma of jaw fibro-osseous lesions. Mast cells were shown to be a major source of sclerostin in the lesions, which contributed to abnormal bone remodeling and sustained fibrous tissue through inhibition of the Wnt pathway. Understanding the mechanistic and clinical significance of this interaction could guide diagnostic approaches and therapeutic interventions in FOLs.

Registration and protocol

This systematic review was conducted in accordance with the PRISMA 2020 guidelines. The review was not prospectively registered in any international database such as PROSPERO. A formal review protocol was not prepared before the commencement of the study.

Support

The authors received no specific financial support, grant funding, or sponsorship for the conduct of this review. No non-financial institutional support influenced the study design, data collection, interpretation, manuscript preparation, or publication decisions.

Competing interests

The authors declare that they have no competing interests related to this study.

Availability of data, code, and other materials

All data generated or analyzed during this review are included within the published manuscript and its tables. No statistical code or specialized analytic software scripts were generated because quantitative meta-analysis was not performed. Additional materials related to data extraction may be made available from the corresponding author upon reasonable request.

Author contributions

Dr. Karthik Shunmugavelu conceptualized the study, designed the review methodology, supervised data interpretation, and prepared the manuscript draft. Dr. Evangeline Cynthia Dhinakaran contributed to literature screening, data extraction, risk of bias assessment, and manuscript revision. Dr. Rufus Ranjitsingh Edwin contributed to data interpretation, critical review of the manuscript, and final approval of the submitted version. All authors read and approved the final manuscript.

Author biography

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