ORIGINAL RESEARCH

# Gene Ontology Analysis of Calcifying Odontogenic Cyst, Dentinogenic Ghost Cell Tumour and Ghost Cell Odontogenic Carcinoma

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## Abstract

**Background:** Ghost cell-associated odontogenic lesions share characteristic histopathological features, yet their molecular pathogenesis remains unclear.

**Aim:** This study aimed to identify commonly expressed genes and associated biological pathways in calcifying odontogenic cysts (COC), dentinogenic ghost cell tumors (DGCT) and ghost cell odontogenic carcinomas (GCOC) using gene ontology analysis to gain insight into their shared molecular pathogenesis.

**Materials and Methods:** Genes associated with calcifying odontogenic cysts, dentinogenic ghost cell tumours, and ghost cell odontogenic carcinomas were identified through a literature search. Common genes were analyzed using the PANTHER Classification System, and their functional relevance was further assessed by molecular docking and MM-GBSA binding energy calculations.

**Results:** Three common genes (CTNNB1, P53, and COX2) were identified across all lesions. Gene ontology and pathway analyses implicated these genes in key regulatory, metabolic, and signalling processes, particularly the Cadherin, P53, and Wnt signalling pathways. Molecular docking identified Quercetin, Nutlin-3a, and Celecoxib as the strongest binders to CTNNB1, P53, and COX-2, respectively. CTNNB1, P53, and COX2 were identified as common molecular factors across ghost cell-associated odontogenic lesions, suggesting a shared pathogenic basis.

**Conclusion:** These findings provide a foundation for further studies and highlight potential targets for future diagnostic and therapeutic strategies.

## Introduction

Ghost cell-associated odontogenic lesions, including calcifying odontogenic cysts (COC), dentinogenic ghost cell tumour (DGCT), and ghost cell odontogenic carcinoma (GCOC), represent a spectrum of lesions characterized by the presence of eosinophilic anucleate ghost cells and varying degrees of calcification[1,2]. Although these entities share distinctive histopathological features, their biological behaviour ranges from benign cystic lesions to locally aggressive tumours and malignant neoplasms[3]. Despite extensive histopathological characterization, the molecular mechanisms underlying ghost cell formation and the biological relationships among these lesions remain poorly understood[4].

Several theories have been proposed regarding the origin and pathogenesis of ghost cell-associated lesions. COC is thought to arise from odontogenic epithelial remnants, such as the dental lamina or epithelial rests of Malassez, whereas DGCT is considered a neoplastic counterpart characterized by odontogenic epithelial proliferation and dentinoid formation[5,6]. GCOC is generally regarded as the malignant counterpart within this spectrum and may develop through malignant transformation of pre-existing COC or DGCT lesions[7]. Previous molecular studies have identified alterations in genes involved in cell proliferation, differentiation, apoptosis, and odontogenic development; however, these investigations have largely focused on individual lesions or isolated molecular markers, limiting a broader understanding of shared molecular mechanisms[8].

Despite increasing evidence of molecular alterations in ghost cell-associated odontogenic lesions, a comprehensive

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evaluation of genes commonly associated with COC, DGCT, and GCOC has not been undertaken. Consequently, the molecular pathways that may account for their overlapping histopathological features and potential biological continuum remain unclear. This lack of integrated molecular analysis represents a significant research gap in understanding the pathogenesis of these rare lesions.

Therefore, the present study aimed to identify genes commonly reported across COC, DGCT, and GCOC through a systematic literature-based approach and to investigate their biological significance using gene ontology and pathway enrichment analyses. By focusing on shared molecular signatures across the spectrum of ghost cell-associated odontogenic lesions, this study provides a novel perspective on their common pathogenic mechanisms and contributes to a better understanding of their molecular pathogenesis.

## Materials and Methods

### Study Design

This in silico bioinformatics study was conducted to identify genes implicated in the pathogenesis of calcifying odontogenic cyst (COC), dentinogenic ghost cell tumour (DGCT), and ghost cell odontogenic carcinoma (GCOC), and to investigate their shared molecular mechanisms through gene ontology and pathway enrichment analyses. The study was approved by the Institutional Human Ethics Committee (IHEC/SDC/OPATH-2005/23/037).

### Literature Search Strategy

A comprehensive literature search was performed in PubMed/MEDLINE, Scopus, Web of Science, Embase, and Google Scholar from database inception until the date of the search. Additional relevant studies were identified through manual screening of the reference lists of eligible articles.

The search strategy employed Boolean operators and included the following terms:

((((((calcifying odontogenic cyst) OR (calcifying cystic odontogenic tumour)) OR (Gorlin cyst)) OR (keratinising odontogenic cyst)) OR (((dentinogenic ghost cell tumour) OR (epithelial odontogenic ghost cell tumour)) OR (calcifying ghost cell odontogenic tumour))) OR ((((((ghost cell odontogenic carcinoma) OR (odontogenic ghost cell tumour)) OR (calcifying ghost cell odontogenic carcinoma)) OR (malignant epithelial

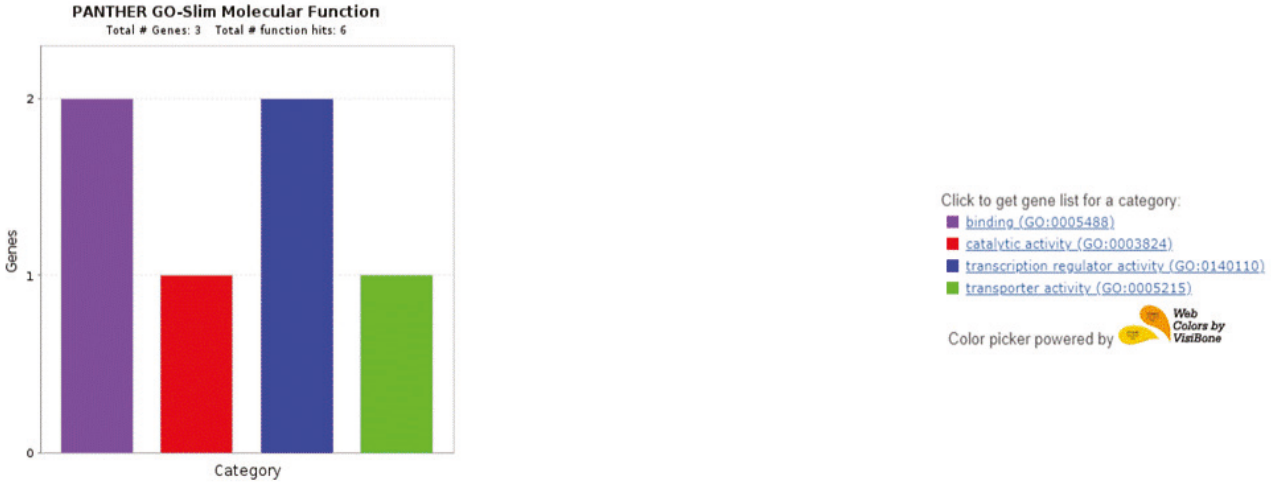


**Fig. 1:** Graph depicting molecular function


**Fig. 2:** Graph depicting biological process

odontogenic ghost cell tumour)) OR (aggressive epithelial ghost cell odontogenic tumour)) OR (malignant calcifying ghost cell odontogenic tumour))) AND (((genetic studies) OR (molecular biology)) OR (signalling molecule))

The search aimed to identify studies reporting molecular, genetic, genomic, transcriptomic, proteomic, or immunohistochemical findings associated with COC, DGCT, and GCOC.

**Eligibility Criteria**

**Inclusion Criteria**

Studies were included if they met the following criteria:

1. Investigated human cases of COC, DGCT, and/or GCOC.
2. Reported molecular, genetic, genomic, transcriptomic, proteomic, or immunohistochemical findings related to lesion pathogenesis.
3. Identified specific genes, proteins, signalling molecules, molecular markers, or pathways associated with these lesions.
4. Were published as original research articles, case reports, or case series in peer-reviewed journals.
5. Were available in the English language.

**Exclusion Criteria**

Studies were excluded if they:

1. Were review articles, systematic reviews, meta-analyses, editorials, letters to the editor, conference abstracts, or book chapters without original molecular data.
2. Focused exclusively on clinical, radiographic, or histopathological findings without molecular characterization.
3. Involved animal studies or in vitro experiments not directly related to human odontogenic lesions.
4. Provided insufficient molecular information for gene extraction.
5. Represented duplicate publications or overlapping datasets.

**Study Selection and Data Extraction**

All retrieved records were screened based on titles and abstracts. Full-text articles were subsequently assessed for eligibility according to the predefined inclusion and exclusion criteria. Relevant studies were selected for data extraction.

The following information was extracted from each eligible study: author, year of publication, lesion type, molecular

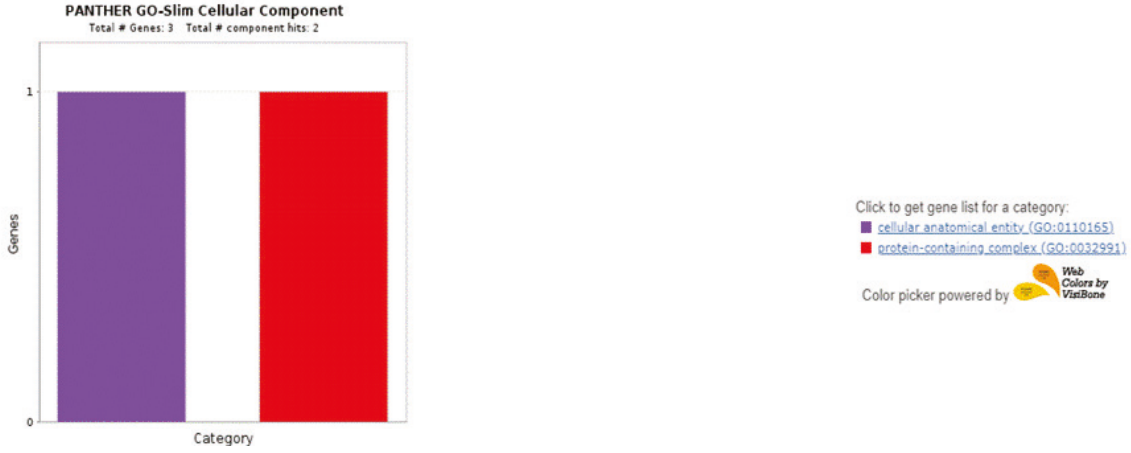


**Fig. 3:** Graph depicting cellular component


**Fig. 4:** Graph depicting protein class

markers investigated, genes identified, signalling pathways reported, and principal molecular findings. Gene symbols were standardized according to the HUGO Gene Nomenclature Committee (HGNC) nomenclature to ensure consistency and eliminate duplication arising from alternative gene names.

**Identification of Commonly Expressed Genes**

Genes reported in association with COC, DGCT, and GCOC were compiled into a unified dataset. Comparative analysis was performed to identify genes commonly expressed across two or more lesion types. These shared genes were considered potential contributors to the common molecular pathogenesis of ghost cell-associated odontogenic lesions and were selected for downstream bioinformatic analyses.

**Gene Ontology and Pathway Enrichment Analysis**

Functional characterization of the commonly expressed genes was performed using the PANTHER (Protein ANalysis THrough Evolutionary Relationships) Classification System. Gene ontology (GO) analysis was conducted to evaluate the molecular functions, biological processes, cellular components, protein classes, and signalling pathways associated with the identified genes.

The enrichment analysis enabled the classification of genes according to their biological roles and facilitated the identification of molecular pathways potentially involved in odontogenic differentiation, epithelial proliferation, calcification, apoptosis, and tumour progression.

**Molecular Docking Analysis**

To further investigate the functional relevance of the commonly expressed genes identified through bioinformatic analysis, molecular docking studies were performed. Protein structures corresponding to the selected genes were retrieved from the Protein Data Bank (PDB). Representative ligands with known interactions involving similar molecular targets were selected based on published literature and available structural data.

Protein and ligand preparation followed a standard AutoDock Vina-based workflow. Protein structures were processed by removing crystallographic water molecules, adding polar hydrogen atoms, and assigning Gasteiger charges. Ligand structures were energy-minimized prior to docking. Binding sites were defined based on reported active, catalytic, or regulatory regions of the target proteins.

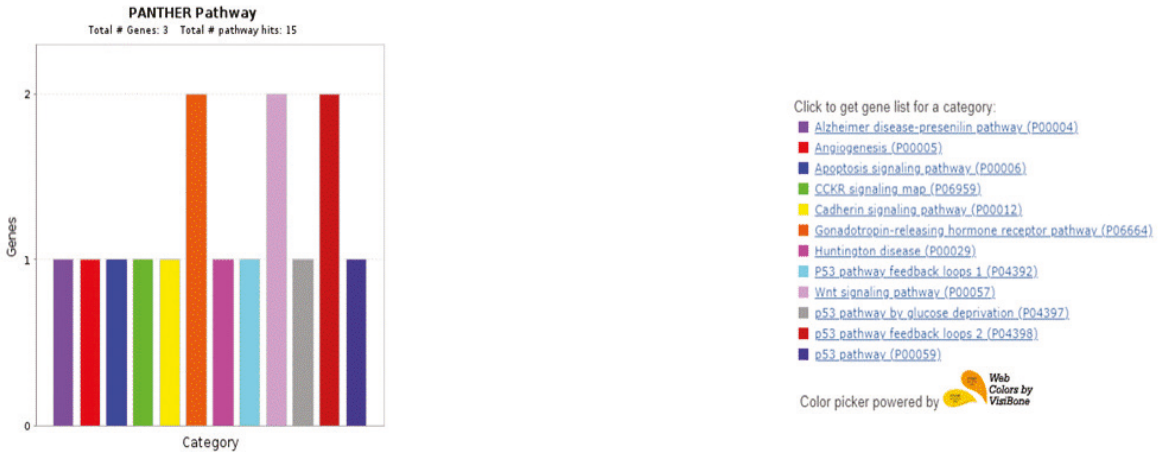


**Fig. 5:** Graph depicting pathway

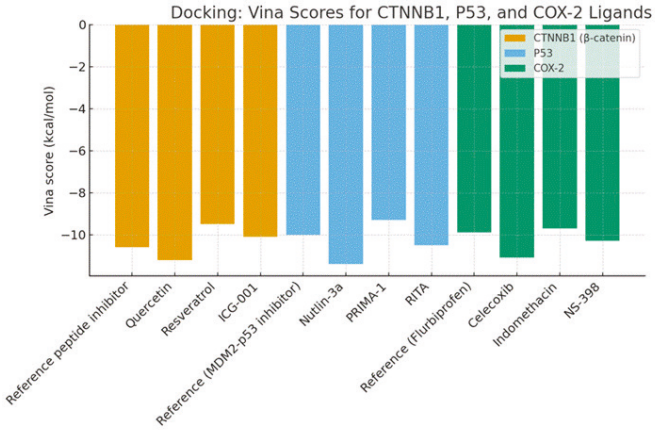


**Fig. 6.** AutoDock Vina binding scores for ligands against CTNNB1, P53, and COX-2.

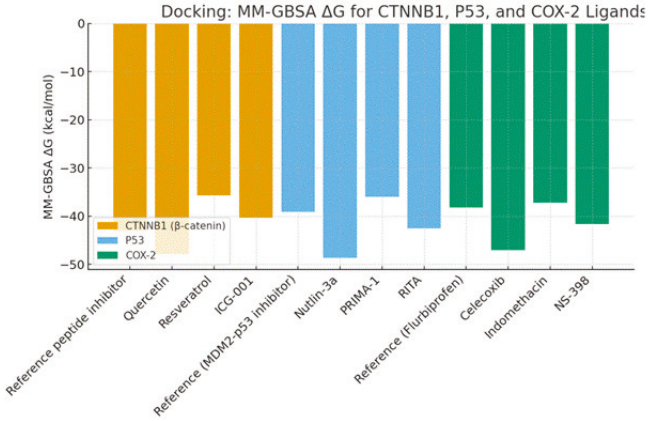


**Fig. 7.** MM-GBSA ΔG rescoring for CTNNB1, P53, and COX-2 ligand complexes.

Docking simulations were performed using AutoDock Vina, and binding affinities were expressed as binding free energies (ΔG, kcal/mol). Protein–ligand interactions were subsequently visualized and analyzed using molecular visualization software.

**Binding Free Energy and Dissociation Constant Estimation**

Post-docking binding free energies were further evaluated using the Molecular Mechanics Generalized Born Surface Area (MM-GBSA) method to obtain more accurate estimates of protein–ligand binding stability. Corresponding dissociation constants (Kd) were calculated using established thermodynamic relationships between binding free energy and equilibrium dissociation constants. These analyses were performed to assess the relative binding strengths and potential biological relevance of the identified molecular interactions.

**Data Interpretation**

The results of the gene ontology, pathway enrichment, and molecular docking analyses were interpreted in the context of odontogenic development, epithelial differentiation, calcification, cell proliferation, apoptosis, and tumour progression. Shared molecular signatures identified among COC, DGCT, and GCOC were evaluated to elucidate potential common pathogenic mechanisms underlying ghost cell-associated odontogenic lesions.

## Results

The analysis of commonly expressed genes across calcifying odontogenic cyst (COC), dentinogenic ghost cell tumour (DGCT), and ghost cell odontogenic carcinoma (GCOC) revealed three key genes: CTNNB1, P53, and COX2. These genes exhibited diverse molecular functions, including binding (CTNNB1 and P53), catalytic activity (COX2), transcription regulatory activity (CTNNB1 and P53), and transporter activity (COX2) (Figure 1). In terms of biological processes, the identified genes were involved in biological regulation (CTNNB1 and P53), cellular processes (CTNNB1, P53, and COX2), localization (COX2), and metabolic processes (CTNNB1, P53, and COX2) (Figure 2). Regarding cellular components, CTNNB1 was associated with cellular anatomical entity and protein-containing complex (Figure 3). Additionally, P53 was categorised as a gene-specific transcriptional regulator, while COX2 was classified as a metabolic interconversion enzyme (Figure 4). Furthermore, pathway analysis revealed the involvement of these genes in several crucial pathways, including the Cadherin signalling pathway (CTNNB1), P53 pathway feedback (P53), and Wnt signalling pathway (CTNNB1 and P53) (Figure 5-8).

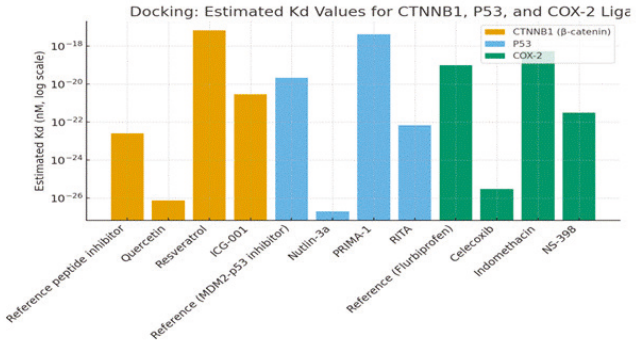


**Fig. 8.** Log-scaled estimated dissociation constants (Kd, nM) derived from MM-GBSA ΔG values.

## Discussion

The range of odontogenic lesions linked to ghost cells has long been a mysterious phenomenon, especially in light of the connections in their development[9,10]. In this study, we employed gene ontology analysis to identify commonly expressed genes implicated in the pathogenesis across the COC, DGCT, and GCOC spectrum. The analysis revealed CTNNB1, P53, and COX2 as key commonly expressed genes, consistent with prior literature. The beta-catenin protein is encoded by the CTNNB1 gene, which is essential to the pathophysiology of COC. COC formation is associated with abnormal activation of the Wnt/β-catenin signaling pathway, which is frequently connected to CTNNB1 mutations. This leads to deregulation of cell differentiation and proliferation, which aids in the development of cysts[11][12]. The Wnt/β-catenin signaling pathway is abnormally activated by CTNNB1 mutations, which are commonly found in DGCT. DGCT formation and progression are driven by cell proliferation, differentiation, and death, all of which are disrupted by this imbalance[13,14]. Similarly, GCOC is frequently associated with CTNNB1 mutations, which cause dysregulated activation of the Wnt/β-catenin signaling pathway. The aggressive nature and tumor growth of GCOC are fueled by this dysregulation, which impacts cell proliferation, differentiation, and apoptosis[14][15]. Numerous studies have reported that DGCT and GCOC arise from COC, with GCOC further originating from DGCT[14][16][17][18][19]. From these observations, it can be concluded that CTNNB1 significantly influences cell proliferation and differentiation in COC, DGCT, and GCOC.

P53 is frequently expressed in GCOC, DGCT, and COC. The genesis and progression of COC may be influenced by dysregulation of P53 expression and function. Changes in P53 can interfere with apoptosis, DNA repair, and cell cycle regulation, which may promote carcinogenesis in COC[20][21]. The development and pathogenesis of DGCT are influenced by the P53 gene, which is involved in its etiology. P53 mutations are common in DGCT, which compromises its tumor-suppressive activity and permits uncontrolled cell survival and proliferation. The aggressive traits of DGCT are probably caused by this P53 dysregulation.[18,22,23]. The role of P53 gene is essential to the pathophysiology of GCOC. Tumor initiation and progression are facilitated by frequent deregulation of P53 expression and function in GCOC. Changes in P53 interfere with apoptosis, DNA repair, and cell cycle regulation, allowing GCOC to act aggressively and spread[24,25].

COX2 represents another gene commonly expressed in COC, DGCT, and GCOC. Although COX-2 expression has been observed in diverse odontogenic cysts, existing literature points to only minimal involvement of the COX2 gene in COC pathogenesis[26]. Cyclooxygenase-2, or COX2, is linked to both inflammatory and carcinogenic processes. In the cystic microenvironment, dysregulation of COX2 expression may promote angiogenesis, cell proliferation, and inflammation,

which could lead to the development of COC[27]. Due to a lack of focused studies, the function of COX2 in dentinogenic ghost cell tumors (DGCT) is still poorly understood. COX2, an enzyme essential to prostaglandin synthesis, promotes angiogenesis, inflammation, and cell division. Through its pro-inflammatory and pro-tumorigenic actions, COX2 overexpression correlates with tumor formation and progression in a variety of different tumors. Although the existence of COX2 in DGCT has been shown, there are no particular investigations on its role in DGCT.

In the literature, COX2's role in GCOC is likewise understudied. While COX2 is involved in inflammation and carcinogenesis in a variety of malignancies, its precise function in the pathophysiology of GCOC has not been thoroughly investigated. Further studies on COX2 expression and activity in GCOC hold potential for understanding tumor biology and discovering therapeutic targets within COX2-mediated pathways, given its ability to promote inflammatory responses and cell proliferation.

Molecular docking analysis indicates that Quercetin, Nutlin-3a, and Celecoxib could demonstrate robust predicted binding to β-catenin, p53, and COX-2, respectively. These interactions might regulate the Wnt/β-catenin pathway, P53 feedback regulation, and prostaglandin-mediated inflammation—key processes underlying the progression from COC to DGCT and GCOC.

Based on the existing data, it is possible to hypothesize the mechanistic evolution of ghost cell lesions, which range from benign calcifying odontogenic cysts to malignant ghost cell odontogenic carcinomas (GCOCs), which are caused by a coordinated oncogenic cascade involving CTNNB1, COX2, and TP53. The canonical β-catenin destruction complex is disrupted by somatic driver mutations in the CTNNB1 gene. This leads to abnormal cytoplasmic accumulation and nuclear translocation of β-catenin, which activates the constitutive Wnt signaling pathway[15][12,14]. This sustained Wnt signaling directly upregulates COX2 (cyclooxygenase-2) expression, generating an inflammatory tumor microenvironment and pro-survival prostaglandin ($PGE_2$) feedback loops that accelerate epithelial cell division. While initial pathogenesis and ghost cell formation are dictated by these CTNNB1-driven alterations, the subsequent or concurrent loss of TP53 (p53) tumor suppressor function removes critical cell cycle checkpoints and apoptotic mechanisms that would otherwise halt cells harboring CTNNB1 mutations. Together, the synergistic effects of CTNNB1-mediated proliferation, COX2-driven inflammation, and TP53 inactivation accelerate the aggressive local tissue invasion, rapid recurrence, and malignant transformation characteristic of advanced ghost cell neoplasms[8,12].

**Limitations of the Study**

Few considerations should be taken into account when interpreting the findings of the present study. First, the analysis was based on data available in the published literature and therefore reflects the scope and depth of existing molecular investigations. Given the relative rarity of dentinogenic ghost cell tumour (DGCT) and ghost cell odontogenic carcinoma (GCOC), the available evidence remains limited, highlighting opportunities for further molecular characterization and validation of the findings presented herein.

Second, the included studies encompassed a variety of investigative approaches, including immunohistochemistry, molecular genetic techniques, transcriptomic analyses, and case-based reports. While this diversity provides a broad perspective on the molecular landscape of ghost cell-associated lesions, differences in study design and methodology may have influenced the range of reported molecular markers and gene expression profiles. In addition, many studies focused on selected candidate genes, underscoring the value of future comprehensive genomic and transcriptomic investigations.

Third, the identification of commonly expressed genes was derived from recurring observations reported across studies rather than from standardized quantitative expression datasets. Accordingly, the results should be viewed as highlighting potentially important molecular patterns and biological processes that warrant further investigation. Similarly, gene ontology and pathway enrichment analyses offer valuable insights into potential functional associations and molecular networks but should be interpreted within the context of exploratory bioinformatic analyses.

In addition, the molecular docking analyses were performed using computational models and publicly available protein structures. These approaches provide useful predictions regarding protein–ligand interactions and can assist in prioritizing candidate targets for future study. Nevertheless, experimental validation remains an important next step to confirm the biological relevance and therapeutic implications of the predicted interactions.

Finally, the study focused primarily on molecular findings and did not incorporate patient-level clinical, radiographic, histopathological, or outcome data. Consequently, the relationship between the identified molecular alterations and clinical characteristics, such as lesion behaviour, recurrence, malignant transformation, and treatment response, represents an important area for future investigation.

**Future Directions**

Future studies should focus on generating larger, multicentre molecular datasets for calcifying odontogenic cysts (COC), dentinogenic ghost cell tumours (DGCT), and ghost cell odontogenic carcinomas (GCOC). Advanced genomic, transcriptomic, and proteomic approaches, combined with functional validation studies, are needed to clarify the molecular mechanisms underlying ghost cell formation, lesion progression, and malignant transformation.

Integrating molecular findings with clinical and histopathological data may facilitate the development of diagnostic biomarkers, prognostic indicators, and targeted therapies. Experimental validation of candidate molecular targets identified through bioinformatic and docking analyses will further support translational and therapeutic advances in ghost cell-associated odontogenic lesions.

## Conclusion

To conclude, this study delivers key insights into the genetic foundations of COC, DGCT, and GCOC. Gene ontology analysis

revealed CTNNB1, P53, and COX2 as commonly expressed genes spanning these lesions, illuminating their prospective contributions to pathogenesis. Although these results establish a basis for additional research, limitations such as dependence on in silico methods and literature synthesis necessitate prudent interpretation. Yet, these constraints highlight avenues for subsequent exploration. Integrating experimental validation and mitigating potential biases in future work could bolster the reliability of these observations. In the long term, greater comprehension of the molecular mechanisms driving these odontogenic lesions may foster progress in diagnostic and therapeutic approaches, ultimately benefiting patient care. Accordingly, this research acts as a foundational step in deciphering the intricacies of COC, DGCT, and GCOC, with implications for advancing odontogenic carcinoma studies.

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