Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Molar incisor hypomineralisation and pulp chamber dimensions in first permanent molars: a cross-sectional clinical and radiographic study

Beatriz Kelly Barros Lopes
School of Dentistry, University of São Paulo
Bianca Caroline Gomes
School of Dentistry, University of São Paulo
Antonio Assis Leandro Junior
School of Dentistry, University of São Paulo
Maria Júlia Delsin Gallo
School of Dentistry, University of São Paulo
Sofia Brito Bordini
School of Dentistry, University of São Paulo
Andiara de Rossi Daldegan
School of Dentistry, University of São Paulo
Francisco Wanderley Garcia de Paula-Silva
School of Dentistry, University of São Paulo
David John Manton
University Medical Center Groningen
Alexandra Mussolino de Queiroz
School of Dentistry, University of São Paulo
Fabrício Kitazono de Carvalho
fabricio_kc@forp.usp.br

School of Dentistry, University of São Paulo

Research Article

Keywords: molar incisor hypomineralisation, panoramic radiography, dental pulp cavity, dentine sensitivity, paediatric dentistry

Posted Date: November 12th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11483143/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Molar incisor hypomineralisation and pulp chamber dimensions in first permanent molars: a cross-sectional clinical and radiographic study

Corresponding author: Fabricio Kitazono de Carvalho

ABSTRACT

Molar incisor hypomineralisation (MIH) is a qualitative enamel defect that compromises tooth structure and is frequently associated with hypersensitivity, increased caries risk and early pulpal involvement. Evidence on the internal morphology of affected teeth remains limited. To analyse the association between MIH, across different degrees of severity, and pulp chamber dimensions in first permanent molars (FPMs). Cross-sectional clinical and radiographic study of 66 children and 237 FPMs, classified at tooth level as sound, mild MIH or severe MIH. The perimeter of the coronal pulp chamber was measured on pre-existing panoramic radiographs using ImageJ, by two examiners blinded to group allocation, and converted to millimetres. Data were analysed using linear mixed models with a random intercept for the participant, adjusted for age, sex and dental arch. Hypersensitivity and lesion extension were secondary variables restricted to MIH-affected molars. A total of 234 FPMs were analysed. Pulp chamber perimeter was 21.55 ± 3.70 mm in sound molars, 18.59 ± 7.29 mm in mild MIH and 16.95 ± 4.48 mm in severe MIH. In the adjusted model, molars with mild MIH showed a perimeter 3.96 mm smaller than sound molars (95% CI −6.08 to −1.84; $p < 0.001$) and molars with severe MIH 4.89 mm smaller (95% CI −6.84 to −2.95; $p < 0.001$), with no significant difference between mild and severe MIH ($p = 0.200$). The intraclass correlation coefficient for clustering of teeth within children was 0.545. Hypersensitivity was recorded in 35.3% of MIH-affected molars and in no sound molar, and increased with lesion extension (OR 2.50 per grade; 95% CI 1.56 to 4.00). MIH was associated with smaller pulp chamber dimensions in first permanent molars, irrespective of the degree of severity.

Keywords: molar incisor hypomineralisation; panoramic radiography; dental pulp cavity; dentine sensitivity; paediatric dentistry.

INTRODUCTION

Molar incisor hypomineralisation (MIH) is a qualitative defect of dental enamel that arises during the mineralisation phase, characterised by asymmetrical demarcated opacities ranging from white-cream to yellow-brown, affecting at least one first permanent molar (FPM) and potentially involving the incisors [1, 2], and other teeth [3]. The severity of MIH lesions may range from mild to severe and, in more advanced cases, post-eruptive enamel breakdown may occur, leading to dentine exposure, biofilm accumulation, increased risk of caries lesions in those at increased caries risk, hypersensitivity and early pulpal involvement [4, 5]. The proposed aetiology of MIH remains multifactorial and has not been fully established [6].

From a histological perspective, FPMs affected by MIH exhibit decreased enamel density of variable extent, with altered prismatic organisation and increased organic content. These structural characteristics decrease hardness and increase enamel porosity and permeability, facilitating the diffusion of oral fluids, acids and bacterial by-products through the enamel, and oral bacteria may reach and invade dentinal tubules [7]. In more severe presentations, loss of enamel structure exposes dentine directly to the oral environment, and the degree of severity has been related to caries experience in affected molars [8]. When subjected to persistent or repeated noxious stimuli, the dental pulp may respond protectively through the deposition of tertiary dentine, in an adaptive process that may progressively modify the internal morphology of the tooth and reduce the pulp chamber space. Reparative dentine has been described histologically beneath apparently intact but hypomineralised enamel in molars with MIH [7].

Recent evidence also indicates that MIH may affect the physiology of dentine and pulpal tissue. Using micro-computed tomography, Uyar et al. [9] observed that FPMs with MIH exhibited lower dentine mineral density across all root regions, as well as alterations in the pattern of accessory canals, particularly in the cervical and middle thirds. Although no differences in pulp volume were identified, the authors reported a reduced presence of pulp calcifications in hypomineralised teeth. These findings suggest that MIH-related alterations may extend beyond the enamel to involve the mineralisation and structural organisation of dentine and aspects of pulpal morphology.

Reduction of the pulp chamber space is not exclusive to MIH and has been described in other clinical contexts. In a systematic review, Vitali et al. [10] reported that orthodontic treatment may induce morphological changes in the pulp, including dentine deposition and transient inflammation, and volumetric analyses using cone-beam computed tomography have confirmed reductions in pulp space following orthodontic movement [11]. In periodontal disease, Terlemez et al. [12] observed that patients with periodontitis exhibited approximately a 20% reduction in pulp volume compared with sound teeth. This reduction may be associated with the inflammatory response induced by toxic products and microorganisms reaching the pulp through accessory and lateral canals and dentinal tubules. Similarly,

the progression of caries lesions and occlusal trauma may generate persistent stimuli capable of triggering tertiary dentine deposition. Dental bleaching may induce a pulpal inflammatory response and dentine deposition, with a consequent reduction in pulp chamber space [13, 14].

These observations indicate that the internal morphology of teeth is dynamic and may be modified by inflammatory, mechanical or iatrogenic stimuli. In the context of MIH, Sezer et al. [15] demonstrated that the condition does not affect the chronology of dental development or the dental age of children, but the hypothesis of a reduction in pulp chamber dimensions was not addressed, which constitutes a specific gap in the literature.

Panoramic radiographs, despite the limitations inherent to a two-dimensional image, are accessible, low in cost and associated with a low radiation dose, and allow comparative analysis between sound and affected teeth within the same individual [16]. In children who already have a panoramic radiograph obtained for diagnostic purposes, this approach permits the assessment of dimensional alterations of the pulp chamber without additional radiation exposure.

The literature on enamel characteristics and clinical manifestations of MIH is extensive, but evidence on the internal morphology of affected teeth remains limited, and the relationship between MIH and pulp chamber dimensions has not been directly investigated. The aim of the present study was to analyse the association between MIH, across different degrees of severity, and pulp chamber dimensions in FPMs. The specific objectives were to compare pulp chamber perimeter between sound FPMs, FPMs with mild MIH and FPMs with severe MIH; to examine whether these measurements were associated with age, sex and dental arch; and to describe the prevalence of hypersensitivity and the distribution of enamel lesion extension across severity levels.

The null hypothesis was that pulp chamber dimensions in FPMs would not differ according to the presence or severity of MIH.

MATERIALS AND METHODS

Study design and ethical approval

This was a cross-sectional observational clinical and radiographic study. The protocol was approved by the Research Ethics Committee. Parents or legal guardians provided written informed consent and children provided written assent. The study was conducted in accordance with the Declaration of Helsinki and reported according to the STROBE statement for cross-sectional studies [17].

Setting and participants

Participants were recruited by convenience sampling during routine appointments at the Enamel Clinic of FORP-USP between November 2021 and December 2023. Clinical examination and

group allocation were performed by a single dentist (BKBL) previously calibrated for the diagnosis of developmental enamel defects, with intra-examiner agreement above 0.8 (Cohen kappa).

Eligibility criteria

Children were eligible if they were receiving preventive or restorative care at FORP-USP, had a complete medical and dental history, and had a panoramic radiograph obtained previously for diagnostic purposes unrelated to the present study. In the control group, FPMs were required to be free of enamel defects, caries lesions, fractures and radiographically detectable periapical lesions, and to show no fistulae or symptoms suggestive of pulpitis or dentoalveolar abscess (Figures 1 and 2). In the MIH groups, FPMs were required to present demarcated opacities or post-eruptive enamel breakdown, with the pulp chamber outline fully identifiable on the radiograph (Figures 3 and 4). Teeth with restorations or caries lesions extending over the pulp chamber outline were not measured. Children with systemic conditions such as diabetes, congenital or acquired heart disease, leukaemia, epilepsy and chronic asthma were excluded, as were those using analgesics, anxiolytics or anti-inflammatory drugs, those undergoing orthodontic treatment, and those presenting gingival recession in the teeth assessed.

Sample size

The sample size was estimated from the data of Terlemez et al. [12] and from a pilot study of 21 FPMs, 11 with severe MIH and 10 sound. Pulp chamber dimension was used as the reference parameter, considering its standard deviation and the difference between the mean values observed in the pilot. A power of 80%, a significance level of 5% and an estimated attrition of 20% were adopted, resulting in a minimum of 52 teeth per group.

Groups and MIH classification

MIH was diagnosed according to Ghanim et al. [18] and severity was classified according to Lygidakis et al. [2]. Classification was performed at tooth level. Each FPM was allocated to one of three groups: control, comprising sound molars; mild MIH, comprising molars with demarcated opacities without loss of enamel structure; and severe MIH, comprising molars with post-eruptive enamel breakdown, atypical restorations or atypical caries lesions. Because MIH may affect molars of the same child with different severities, both mild and severe teeth could originate from the same participant. For descriptive purposes only, each child was additionally classified by the most severely affected molar.

Hypersensitivity and lesion extension

Hypersensitivity and lesion extension were secondary variables and were recorded only for MIH-affected FPMs. Hypersensitivity was assessed during the clinical examination using two instruments applied in sequence. A one-second air blast was delivered from a triple syringe at a distance of approximately one centimetre from the buccal surface, and the behavioural response was recorded using the Schiff Cold Air Sensitivity Scale (SCASS), ranging from 0, no response to the stimulus, to 3,

response, withdrawal and request to discontinue the stimulus. The child then rated the perceived intensity on a Visual Analogue Scale (VAS) from 0 to 10. A tooth was classified as hypersensitive when SCASS or VAS scored above zero. The extent of enamel involvement was recorded as less than one third, between one third and two thirds, or more than two thirds of the affected surface.

Radiographic images

Pre-existing panoramic radiographs obtained during routine diagnostic care were used. All images were acquired with the same panoramic unit and exported at a native resolution of 2903 × 1536 pixels, with a recorded density of 264 dpi. No resampling, cropping or contrast adjustment was applied before measurement.

Pulp chamber measurement

Measurements were performed in ImageJ (version 1.53, NIH, Bethesda, USA) by two examiners (BKBL and AALJ), both blinded to group allocation and to the clinical data of the participants. The coronal pulp chamber of each FPM was outlined with the freehand selection tool, and the perimeter of the outline was recorded (Figure 5). Each tooth was measured in three consecutive sessions and the mean of the three values was used in the analysis. Pixel values were converted to millimetres using the constant derived from the recorded image density, 1 pixel corresponding to 0.096212 mm. Measurements are reported, without correction for the intrinsic magnification of panoramic radiography, which applies equally to all groups (Figure 6).

Statistical analysis

The tooth was the unit of analysis. Because up to four FPMs were obtained from the same child, all models included a random intercept for the participant to account for the non-independence of teeth within individuals. Continuous variables are presented as means, standard deviations and 95% confidence intervals, and categorical variables as absolute and relative frequencies. The significance level was set at 5%. The statistician was blinded to group allocation throughout the analysis. Repeatability of the radiographic measurement was assessed from the three consecutive measurements obtained for each tooth, using a two-way random-effects intraclass correlation coefficient for single measurements, ICC(2,1), and the within-tooth coefficient of variation.

The primary analysis was a linear mixed model with pulp chamber perimeter as the dependent variable and group as the fixed factor of interest, adjusted for age, sex and dental arch, with a random intercept for the participant. Regression coefficients are reported with 95% confidence intervals, and the intraclass correlation coefficient is reported to describe the proportion of variance attributable to differences between children. Pairwise comparisons between the three groups were obtained by refitting the model with different reference categories.

Two sensitivity analyses were performed. In the first, MIH was entered as a dichotomous variable, contrasting all affected molars with sound molars. In the second, the model was refitted after excluding two teeth with extreme measurements. Agreement between the sensitivity analyses and the primary model was used to assess the stability of the estimates.

Hypersensitivity and lesion extension were analysed as secondary variables and restricted to MIH-affected molars. Proportions are reported with 95% confidence intervals calculated by the Wilson method. Associations between these variables and MIH severity were assessed using generalised estimating equations with a binomial distribution, a logit link and an exchangeable working correlation structure, with the participant as the clustering unit. The distribution of lesion extension across severity levels was compared using the chi-square test. These analyses were exploratory and no adjustment for multiplicity was applied. Analyses were conducted in jamovi 2.6 (The Jamovi Project, Sydney, Australia) and R 4.4 (R Foundation for Statistical Computing, Vienna, Austria.).

RESULTS

A total of 66 children and 237 FPMs were assessed. Three molars were excluded because the pulp chamber outline could not be measured, leaving 234 teeth for analysis: 87 sound molars, 51 with mild MIH and 96 with severe MIH (Figure 7). Teeth were distributed across 117 maxillary and 117 mandibular molars. The characteristics of the participants are shown in Table 1. Sex distribution and age did not differ between groups ($p = 0.455$ and $p = 0.328$, respectively). Children were classified by the most severely affected molar. MIH severity was classified at tooth level, and molars of different severities could originate from the same child.

The three consecutive measurements showed high repeatability, with ICC(2,1) = 0.986 and a median within-tooth coefficient of variation of 1.31% (95th percentile 6.27%) These results are presented in Table 1.

Pulp chamber perimeter was largest in sound molars and smallest in molars with severe MIH (Table 2). Mandibular molars showed larger perimeters than maxillary molars (20.36 ± 6.04 mm and 17.68 ± 4.22 mm, respectively).

In the linear mixed model, both MIH groups showed smaller pulp chamber perimeters than the control group (Table 3). Molars with mild MIH were on average 3.96 mm smaller than sound molars (95% CI −6.08 to −1.84; $p < 0.001$) and molars with severe MIH were 4.89 mm smaller (95% CI −6.84 to −2.95; $p < 0.001$). The difference between mild and severe MIH was not statistically significant (−0.94 mm; 95% CI −2.37 to 0.50; $p = 0.200$). Maxillary molars showed smaller perimeters than mandibular molars, and boys showed smaller perimeters than girls. Age was not associated with pulp chamber perimeter within the age range studied.

More than half of the total variance in pulp chamber perimeter was attributable to differences between children (ICC = 0.545).

Both sensitivity analyses reproduced the primary result. When MIH was entered as a dichotomous variable, affected FPMs showed a perimeter 4.59 mm smaller than sound molars (95% CI −6.49 to −2.69; $p < 0.001$). After excluding the two teeth with extreme measurements, the estimates were −4.06 mm for mild MIH (95% CI −6.10 to −2.02) and −4.73 mm for severe MIH (95% CI −6.57 to −2.89), both $p < 0.001$.

Hypersensitivity was recorded in 53 of the 150 MIH-affected FPMs (35.3%; 95% CI 28.1 to 43.3). No sound molar in the control group presented a positive response to the cold air stimulus. The prevalence was 23.1% in mild MIH and 41.8% in severe MIH (Table 4). After accounting for clustering of teeth within children, the difference between severity groups did not reach statistical significance (OR 2.40; 95% CI 0.95 to 6.06; $p = 0.065$).

Lesion extension differed markedly between severity groups (Table 5; $p < 0.001$). Among molars with mild MIH, 78.8% presented lesions involving less than one third of the surface, whereas among molars with severe MIH, 44.9% presented lesions involving more than two thirds. Lesion extension was associated with hypersensitivity in an exploratory model accounting for clustering, with the odds of hypersensitivity increasing by a factor of 2.50 for each additional grade of extension (95% CI 1.56 to 4.00; $p < 0.001$).

DISCUSSION

The null hypothesis was rejected as FPMs affected by MIH showed smaller pulp chamber dimensions than sound FPMs, and the difference persisted after adjustment for age, sex and dental arch and after accounting for the clustering of teeth within children. The magnitude of the difference was similar in mild and severe MIH-affected FPMs, and the comparison between the two severity levels was not statistically significant. Approximately half of the variance in pulp chamber perimeter was attributable to differences between children, which indicates that analyses treating teeth from the same child as independent observations would overstate the precision of the estimates.

The reduction observed may reflect an adaptive response of the dentine-pulp complex to chronic stimuli associated with the structural characteristics of hypomineralised enamel. Increased enamel porosity and permeability facilitate the diffusion of oral fluids, acids and bacterial by-products, and oral bacteria may invade dentinal tubules beneath enamel that is apparently intact, a finding also described histologically in molars with MIH, together with the presence of reparative dentine [7]. Deposition of secondary and tertiary dentine is a plausible explanation for a smaller pulp chamber space.

The present design does not allow this mechanism to be demonstrated. A panoramic radiograph documents a dimensional change and cannot establish the histological process responsible for it, nor the temporal sequence between exposure and outcome.

The absence of a significant difference between mild and severe MIH deserves comment. The mild and severe categories summarise the clinical appearance of the molar at a single point in time, and both include teeth with widely differing amounts of affected surface. In the present sample, lesion extension was distributed very differently across the two categories and was associated with hypersensitivity in a graded manner, whereas the binary severity label was not. This suggests that a measure of how much of the surface is affected may capture the degree of structural compromise more closely than the mild-severe dichotomy. Because hypomineralised enamel is susceptible to post-eruptive breakdown, further loss of hypomineralised tissue may occur over time. Therefore, the extent of tissue breakdown recorded at a single examination represents the cumulative structural loss up to that point rather than a stable characteristic of the tooth, which limits the interpretation of this variable.

Among the clinical manifestations of MIH, dentine hypersensitivity is of particular relevance, as it may affect the quality of life of children and complicates dental management [19, 20]. In the present study, hypersensitivity was recorded in 35.3% of MIH-affected molars, and in none of the sound molars. The prevalence was 23.1% in mild MIH and 41.8% in severe MIH, although this difference did not reach statistical significance once clustering was taken into account. These values are close to those reported by Vicioni-Marques et al. [21], who described 40.2%, and by Raposo et al. [22], who described 34.7%, and lower than the 60% reported by Gomes et al. [23]. Variation between studies may reflect differences in the instruments used, in the stimulus applied and in the age of the children, since the assessment of pain intensity in paediatric populations depends on the scale adopted and on the developmental stage of the child [24].

Reduction of the pulp chamber space is not specific to MIH and has been reported in other clinical contexts. Vitali et al. [10] described dentine deposition, transient pulpal inflammation and reduction of pulp chamber volume associated with orthodontic forces, and volumetric analyses using cone-beam computed tomography have confirmed a reduction in pulp space after orthodontic movement [11]. Pulp volume can be quantified on both cone-beam computed tomography and panoramic images and varies systematically with chronological age [25]. In periodontal disease, Terlemez et al. [12] reported a reduction of approximately 20% in pulp volume in patients with periodontitis. These observations support the interpretation that the pulp chamber is a dynamic structure that responds to persistent mechanical, inflammatory or infectious stimuli, and they place the present findings in a broader context rather than establishing a mechanism specific to MIH.

The present results differ from those of Uyar et al. [9], who used micro-computed tomography and did not identify differences in pulp volume between molars with and without MIH, although they

reported lower dentine mineral density and alterations in accessory canals. Several methodological differences may account for the divergence. Uyar et al. analysed extracted teeth at microscopic resolution and quantified pulp volume in three dimensions, whereas the present study assessed a two-dimensional outline of the coronal pulp chamber on radiographs of children under clinical care. The two approaches measure different quantities and may reveal complementary aspects of the internal alterations associated with MIH.

The present findings also differ from those of Sezer et al. [15], who concluded that MIH does not influence dental age or the chronology of dental development. The two questions are distinct. Dental age reflects the timing of odontogenesis, whereas pulp chamber dimensions reflect the internal morphology of a tooth that has already erupted and may be modified by later stimuli. The comparability of dental age between children with and without MIH reported by those authors is useful for the present study, since it makes maturational differences less likely to explain the reduction observed here.

The choice of panoramic radiography deserves consideration. Panoramic images have limitations, including structural superimposition, non-uniform magnification and lower resolution than cone-beam computed tomography. They were used because they are widely available, low in cost and associated with a low radiation dose, which is particularly relevant in paediatric populations [26-28]. No radiograph was obtained for the purpose of this study. Cone-beam computed tomography would provide greater accuracy for volumetric analysis, but the authors considered its use was not justified in children solely for the measurement of pulp chamber dimensions.

Measurement of the pulp chamber has been used as a parameter in different areas of dentistry, including the assessment of pulp canal calcification and the description of pulp chamber anatomy [29, 30]. In the context of MIH, the observation that smaller pulp chambers were present even in molars with mild involvement indicates that internal alterations may be present in teeth without post-eruptive breakdown or cavitated lesions. This observation supports individualised treatment planning and conservative, pulp-preserving approaches in affected molars, and periodic radiographic monitoring when radiographs are already indicated for other reasons.

The cross-sectional design does not allow temporal or causal inferences. Exposure and outcome were recorded at the same moment, so it is not possible to establish whether the dimensional differences preceded, accompanied or followed the clinical manifestations of MIH. Pulp chamber dimensions were assessed on two-dimensional images subject to the intrinsic magnification of panoramic radiography, which is not uniform across regions of the image; measurements are therefore reported at the image plane and are valid for comparison between groups within this sample rather than as absolute anatomical values. Although examiners were blinded to group allocation, extensive post-eruptive breakdown may occasionally be perceptible on a panoramic radiograph, so blinding cannot be considered complete for the most severely affected molars. Radiographs were pre-existing and were not necessarily obtained on

the same date as the clinical examination, which introduces a variable interval between the assessment of hypersensitivity and the radiographic measurement. Lesion extension and hypersensitivity were recorded at a single examination in a condition whose enamel loss is progressive, and both should be read as a snapshot rather than a stable attribute of the tooth. Recruitment was by convenience sampling at a referral clinic for enamel defects, and the proportion of severe cases is therefore higher than would be expected in the general population. Finally, the secondary analyses were exploratory, were not adjusted for multiplicity and should be interpreted as hypothesis-generating.

The generalisability of these findings is limited to children of similar age assessed in a referral setting. Longitudinal studies, and histological investigations of teeth with different degrees and extensions of enamel involvement, are required to establish whether the dimensional differences observed correspond to dentine deposition and how they evolve over time.

CONCLUSIONS

Within the limitations of this cross-sectional study, it can be concluded that:

- First permanent molars affected by MIH showed smaller pulp chamber dimensions than sound first permanent molars, after adjustment for age, sex and dental arch and after accounting for the clustering of teeth within children.
- Pulp chamber dimensions did not differ significantly between molars with mild and severe MIH.

ACKNOWLEDGEMENTS

This study was funded by a research grant from the National Council for Scientific and Technological Development (CNPq), process 405914/2021-0.

REFERENCES

1. Weerheijm KL, Duggal M, Mejàre I, Papagiannoulis L, Koch G, Martens LC, et al. Judgement criteria for molar incisor hypomineralisation (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. Eur J Paediatr Dent. 2003;4(3):110-3.
2. Lygidakis NA, Garot E, Somani C, Taylor GD, Rouas P, Wong FSL. Best clinical practice guidance for clinicians dealing with children presenting with molar-incisor hypomineralisation (MIH): an updated European Academy of Paediatric Dentistry policy document. Eur Arch Paediatr Dent. 2022;23(1):3-21.
3. Kevrekidou A, Kosma I, Kotsanos I, Arapostathis KN, Kotsanos N. Enamel opacities in all teeth with the molar-incisor hypomineralization index in adolescents. Int J Paediatr Dent. 2021;31(2):270-7.
4. Neves AB, Americano GCA, Soares DV, Soviero VM. Breakdown of demarcated opacities related to molar-incisor hypomineralization: a longitudinal study. Clin Oral Investig. 2019;23(2):611-5.
5. Mittal N. Phenotypes of enamel hypomineralization and molar incisor hypomineralization in permanent dentition: identification, quantification and proposal for classification. J Clin Pediatr Dent. 2016;40(5):367-76.
6. Garot E, Rouas P, Somani C, Taylor GD, Wong F, Lygidakis NA. An update of the aetiological factors involved in molar incisor hypomineralisation (MIH): a systematic review and meta-analysis. Eur Arch Paediatr Dent. 2022;23(1):23-38.
7. Fagrell TG, Lingström P, Olsson S, Steiniger F, Norén JG. Bacterial invasion of dentinal tubules beneath apparently intact but hypomineralized enamel in molar teeth with molar incisor hypomineralization. Int J Paediatr Dent. 2008;18(5):333-40.
8. Negre-Barber A, Montiel-Company JM, Catalá-Pizarro M, Almerich-Silla JM. Degree of severity of molar incisor hypomineralization and its relation to dental caries. Clin Oral Investig. 2018;22(3):1248-55.
9. Uyar P, Yilmaz BD, Yildirim S, Ozkan B. Evaluation of hard tissue characteristics and calcifications in pulp tissue of hypomineralized permanent molars using micro-computed tomography. Clin Oral Investig. 2025;29:1-10.
10. Vitali F, Costa VP, Cirelli JA, Holzhausen M, Cirelli CC. Orthodontic force and dental pulp changes: a systematic review. J Endod. 2022;48(9):1217-28.
11. Venkatesh S, Ajmera S, Ganeshkar SV. Volumetric pulp changes after orthodontic treatment determined by cone-beam computed tomography. J Endod. 2014;40(11):1758-63.
12. Terlemez A, Alan R, Gezgin O. Evaluation of the periodontal disease effect on pulp volume. J Endod. 2018;44(1):111-4.
13. Souza JB, Gerlach RF, Line SRP, et al. Effects of dental bleaching on pulp chamber volume: a micro-CT study in an animal model. Arch Oral Biol. 2020;109:104558.
14. Terayama AM, Benetti F, de Araújo Lopes JM, Barbosa JG, Silva IJP, Sivieri-Araújo G, et al. Influence of low-level laser therapy on inflammation, collagen fiber maturation, and tertiary dentin deposition in the pulp of bleached teeth. Clin Oral Investig. 2020;24(11):3911-21.
15. Sezer B, Çarıkçıoğlu B, Kargül B. Dental age and tooth development in children with molar-incisor hypomineralization: a case-control study. Arch Oral Biol. 2022;134:105325.
16. American Dental Association Council on Scientific Affairs. The use of dental radiographs: update and recommendations. J Am Dent Assoc. 2006;137(9):1304-12.
17. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453-7.

18. Ghanim A, Elfrink M, Weerheijm K, Mariño R, Manton D. A practical method for use in epidemiological studies on enamel hypomineralisation. Eur Arch Paediatr Dent. 2015;16(3):235-46.
19. de Oliveira MLR, Ferezin AN, Gomes BC, Mattos VS, Mazzi-Chaves JF, Sousa-Neto MD, et al. Optical coherence tomography and grayscale digital analysis as non-invasive techniques to assess the severity of molar-incisor hypomineralization: a comparative study with computed microtomography. Microsc Res Tech. 2024;87(8):1810-21.
20. Americano GC, Jacobsen PE, Soviero VM, Haubek D. A systematic review on the association between molar incisor hypomineralization and dental caries. Int J Paediatr Dent. 2017;27(1):11-21.
21. Vicioni-Marques F, Carvalho MR, Raposo F, de Paula-Silva FWG, de Queiroz AM, Leal SC, et al. Association between dentine hypersensitivity and anxiety in children with molar-incisor hypomineralisation (MIH). Eur Arch Paediatr Dent. 2023;24(3):313-19.
22. Raposo F, de Carvalho Rodrigues AC, Lia ÉN, Leal SC. Prevalence of hypersensitivity in teeth affected by molar-incisor hypomineralization (MIH). Caries Res. 2019;53(4):424-30.
23. Gomes BC, Cardoso-Silva L, Lopes BKB, Melo RPF, Catananti IS, Queiroz AM, et al. Influence of the severity of molar incisor hypomineralization on dental hypersensitivity, anxiety/fear, and aesthetic self-perception: a cross-sectional study. J Appl Oral Sci. 2025;33:1-12.
24. Hicks CL, von Baeyer CL, Spafford PA, van Korlaar I, Goodenough B. The Faces Pain Scale-Revised: toward a common metric in pediatric pain measurement. Pain. 2001;93(2):173-83.
25. Polat Y, Çelenk S. A comparison of age estimation based on pulp volume from cone-beam computed tomography (CBCT) images and panoramic radiograph data with chronological age. J Clin Pediatr Dent. 2024;48(2):149-62.
26. Pagnoncelli SD, Oliveira FAM. The use of panoramic radiography as an initial radiographic diagnostic option in pediatric dentistry. J Bras Odontopediatr Odontol Bebê. 1999;2(7):186-99.
27. White SC, Atchison KA, Hewlett ER, Flack VF. Efficacy of FDA guidelines for prescribing radiographs to detect dental and intraosseous conditions. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1995;80(1):108-14.
28. American Academy of Pediatric Dentistry. Prescribing dental radiographs for infants, children, adolescents, and individuals with special health care needs. The Reference Manual of Pediatric Dentistry. Chicago: AAPD; 2024.
29. Alamoudi RA, Alzayer FM, Alotaibi RA, Alghamdi F, Zahran S. Evaluation of the correlation between systemic conditions and pulp canal calcification: a case-control study. Cureus. 2023;15(9):e45484.
30. Ahmed HMA, Keleş A, Wolf TG, Rossi-Fedele G, Dummer PMH. A proposal to develop a new classification for the anatomy of the pulp chamber. Eur Endod J. 2024;9(1):1-7.

TABLES

Table 1. Characteristics of the participants according to MIH severity.

Group	Children (n)	Female / Male	Age, months (mean ± SD)
Control	25	13 / 12	110.2 ± 19.2
Mild MIH	6	2 / 4	123.7 ± 25.7
Severe MIH	35	21 / 14	115.3 ± 21.0
Total	66	36 / 30	114.1 ± 20.8

The three consecutive measurements showed high repeatability, with ICC(2,1) = 0.986 and a median within-tooth coefficient of variation of 1.31% (95th percentile 6.27%).

Table 2. Pulp chamber perimeter of first permanent molars according to group.

Group	Teeth (n)	Mean ± SD (mm)	95% CI (mm)	Range (mm)
Control	87	21.55 ± 3.70	20.76 – 22.34	14.72 – 31.20
Mild MIH	51	18.59 ± 7.29	16.54 – 20.64	7.73 – 42.63
Severe MIH	96	16.95 ± 4.48	16.04 – 17.86	10.20 – 30.92

Table 3. Linear mixed model for MIH-affected FPM pulp chamber perimeter (234 teeth from 66 children).

Term	β (mm)	95% CI	p value
Intercept	24.04	22.31 to 25.77	< 0.001
Mild MIH vs control	−3.96	−6.08 to −1.84	< 0.001
Severe MIH vs control	−4.89	−6.84 to −2.95	< 0.001
Severe vs mild MIH	−0.94	−2.37 to 0.50	0.200
Maxillary vs mandibular	−2.67	−3.50 to −1.84	< 0.001
Male vs female	−2.12	−3.88 to −0.36	0.018
Age (per month)	0.03	−0.02 to 0.07	0.217

Random intercept for participant. Between-child variance 11.80; residual variance 9.86; intraclass correlation coefficient 0.545.

Table 4. Prevalence of hypersensitivity in MIH-affected molars according to severity and lesion extension.

Category	Teeth (n)	Hypersensitive n (%)	95% CI (%)
Mild MIH	52	12 (23.1)	13.7 – 36.1
Severe MIH	98	41 (41.8)	32.6 – 51.7
Extension I (< 1/3)	62	10 (16.1)	9.0 – 27.1
Extension II (1/3–2/3)	41	17 (41.5)	27.8 – 56.6
Extension III (> 2/3)	47	26 (55.3)	41.2 – 68.6
All MIH molars	150	53 (35.3)	28.1 – 43.3

Hypersensitivity was defined as a score above zero on the SCASS or on the VAS.

Table 5. Distribution of lesion extension according to MIH severity.

Group	Extension I n (%)	Extension II n (%)	Extension III n (%)
Mild MIH (n = 52)	41 (78.8)	8 (15.4)	3 (5.8)
Severe MIH (n = 98)	21 (21.4)	33 (33.7)	44 (44.9)

Extension I, less than one third of the surface; II, between one third and two thirds; III, more than two thirds.

FIGURE LEGENDS

Figure 1. (A, B, C): Frontal photograph and upper and lower arches without MIH.

Figure 2. Representation of a panoramic radiograph of a patient without a diagnosis of MIH.

Figure 3. (A, B, C): Photographs of maxillary and mandibular central incisors and permanent molars diagnosed with MIH.

Figure 4. Representation of a panoramic radiograph of a patient diagnosed with MIH (teeth 16 and 26 diagnosed with severe MIH).

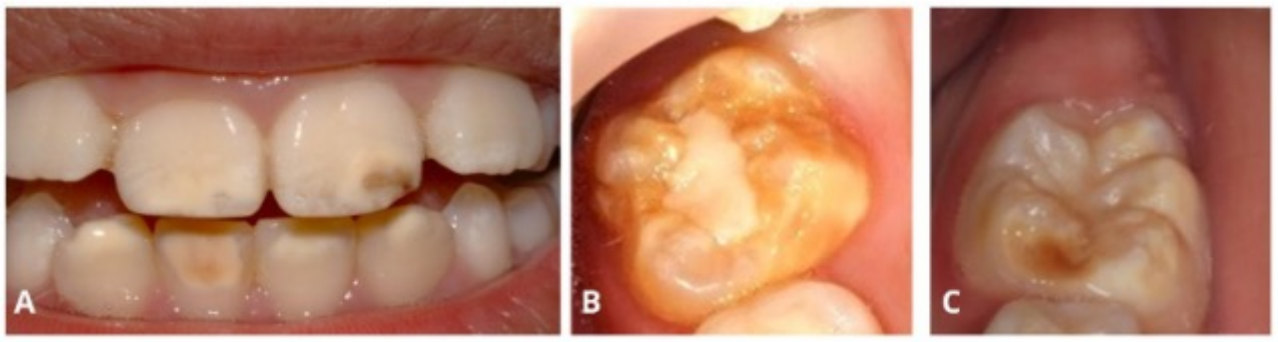
Figure 5. Representative image showing the yellow delineation of molar images obtained using ImageJ software.

Figure 6. Representative image showing the three-time-point analysis of the images obtained using ImageJ software, highlighted in yellow.

Figure 7. Flow of participants and teeth through the study, according to the STROBE recommendations (von Elm et al., 2007).


A
B
C


R
L


A
B
C

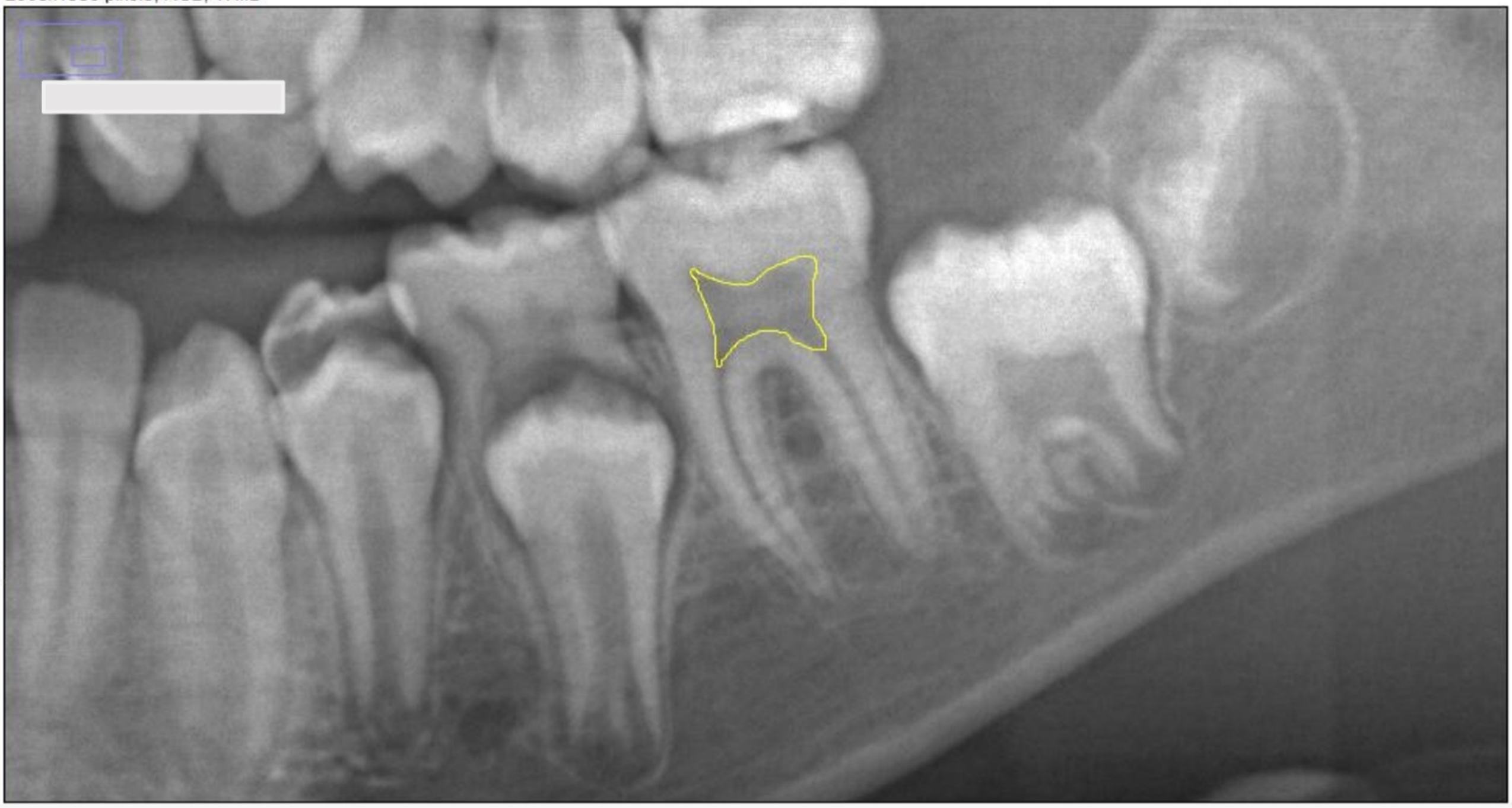


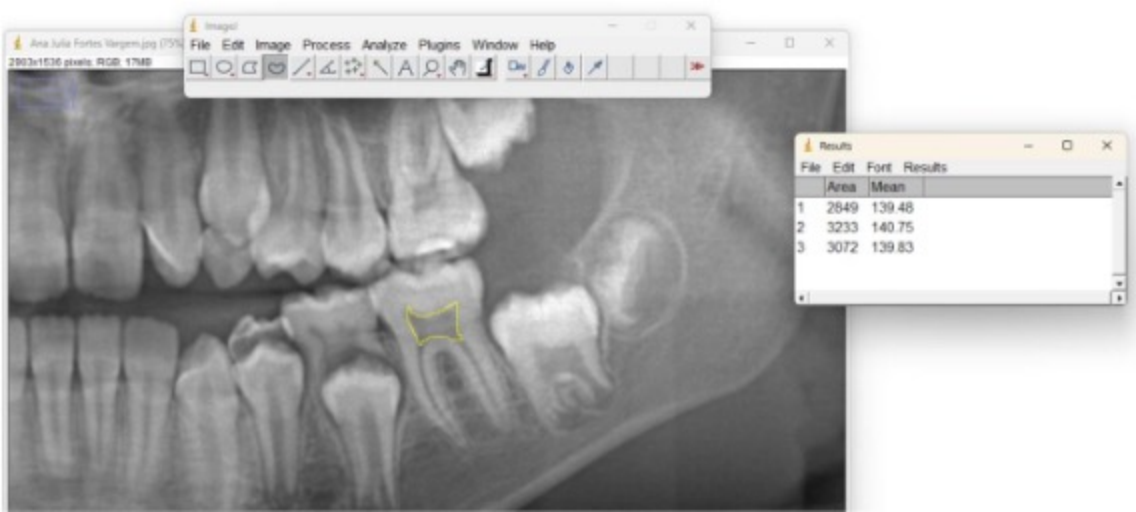
ImageJ

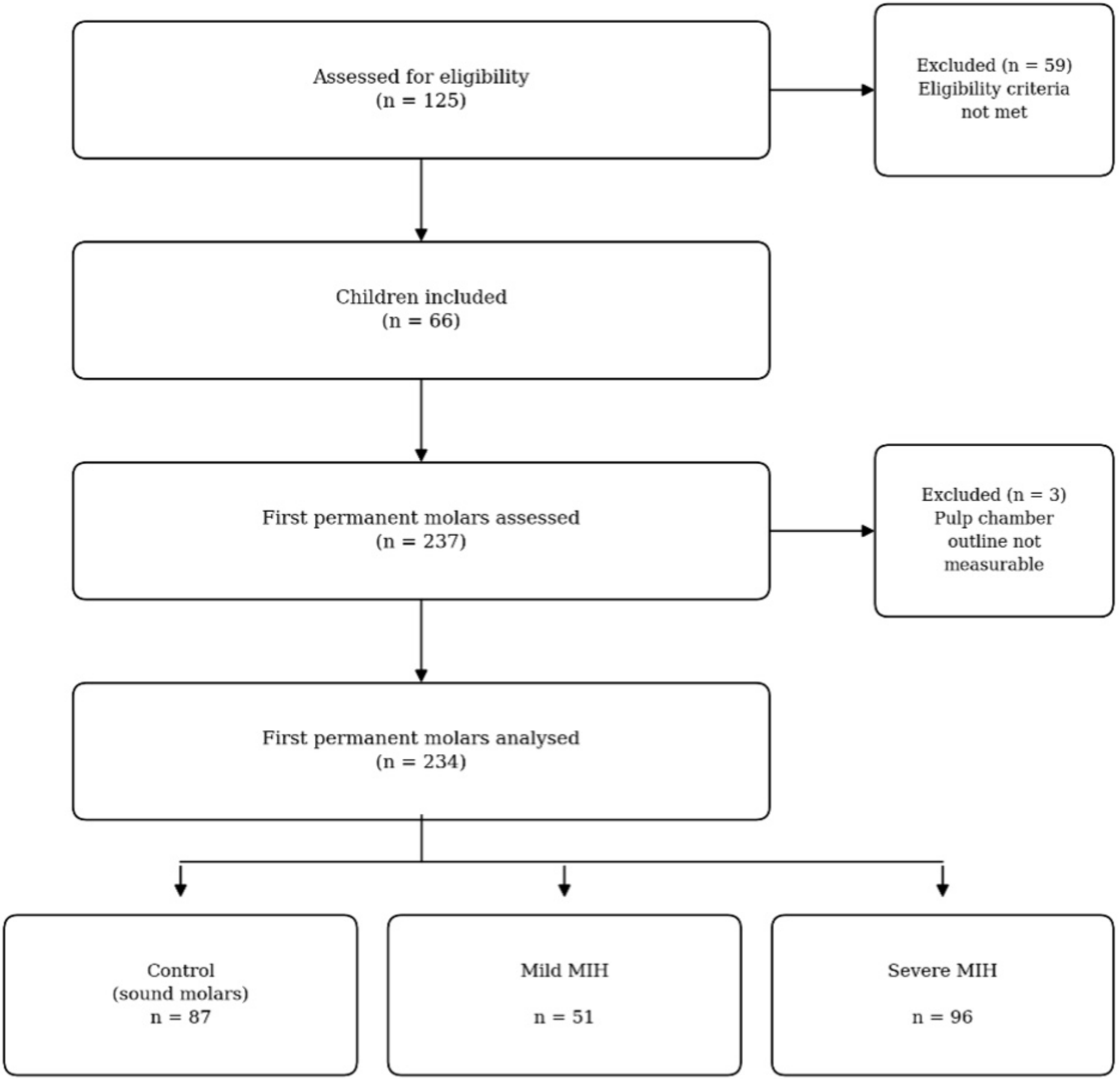
File Edit Image Process Analyze Plugins Window Help

Ana Julia Fortes Vargem.jpg

2903x1536 pixels; RGB; 17MB




ImageJ
File Edit Image Process Analyze Plugins Window Help
Results
File Edit Font Results
Area Mean
1 2849 139.48
2 3233 140.75
3 3072 139.83


Assessed for eligibility
(n = 125)
Excluded (n = 59)
Eligibility criteria
not met
Children included
(n = 66)
First permanent molars assessed
(n = 237)
Excluded (n = 3)
Pulp chamber
outline not
measurable
First permanent molars analysed
(n = 234)
Control
(sound molars)
n = 87
Mild MIH
n = 51
Severe MIH
n = 96