

Original Research

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Evaluation of MMP-8 and MMP-9 Gene Variants and Their Association with Orthodontically Induced External Apical Root Resorption

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ABSTRACT:

Background: Orthodontically induced external apical root resorption (EARR) is a multifactorial adverse effect of orthodontic tooth movement, with evidence supporting contributions from treatment-related and host genetic factors. Objective: To examine the simulated association of MMP-8 and MMP-9 genotype categories with EARR while accounting for age, sex, and treatment duration.

Methods: A synthetic dataset of 400 participants was analyzed, comprising 160 EARR and 240 non-EARR records. Genotype distributions were compared using chi-square tests, crude odds ratios (ORs) with 95% confidence intervals (CIs) were estimated, and multivariable binary logistic regression was performed. Results: EARR records had longer simulated treatment duration than non-EARR records (24.52 ± 4.59 vs 21.67 ± 4.52 months; $p < 0.001$). MMP-8 genotype distribution differed between groups ($\chi^2 = 12.13$, $p = 0.002$), as did MMP-9 genotype distribution ($\chi^2 = 15.97$, $p < 0.001$). Relative to AA, MMP-8 GG showed a crude OR of 2.74 (95% CI 1.54–4.88), while relative to CC, MMP-9 TT showed an OR of 3.17 (95% CI 1.78–5.65). In multivariable analysis, MMP-8 GG (adjusted OR 2.30, 95% CI 1.24–4.28), MMP-9 TT (adjusted OR 2.85, 95% CI 1.54–5.27), male sex (adjusted OR 2.02, 95% CI 1.30–3.14), and treatment duration (adjusted OR 1.14 per month, 95% CI 1.08–1.20) were independently associated with simulated EARR. Conclusion: The simulated dataset demonstrates a genotype-associated pattern involving MMP-8 and MMP-9 alongside treatment duration. These results are hypothesis-

generating only and require validation in a prospectively collected, ethically approved clinical cohort.

INTRODUCTION

EARR is a recognized complication of orthodontic tooth movement and is characterized by loss of root structure, most often at the apical region. The clinical expression ranges from limited apical shortening to substantial resorption that may compromise long-term root support. The biological process is considered multifactorial and involves mechanical loading, periodontal ligament injury, inflammatory signaling, individual root morphology, treatment characteristics, and host susceptibility.[1–4,18–20]

Genetic contribution to EARR has received sustained attention because patients receiving broadly similar orthodontic treatment can exhibit markedly different amounts of root resorption. Familial and genetic studies have suggested substantial heritable variation, while candidate-gene investigations have implicated inflammatory and bone-remodeling pathways.[4–7,27–31] Contemporary reviews emphasize that genetic susceptibility should be interpreted within the context of treatment-related exposures rather than as a single-gene determinant.[8–10,32–35]

Matrix metalloproteinases (MMPs) are extracellular-matrix-remodeling enzymes involved in inflammatory tissue turnover and connective-tissue degradation. MMP-8 and MMP-9 are biologically plausible candidates in the context of orthodontic root resorption because periodontal and periapical tissue remodeling requires coordinated extracellular-matrix turnover.[36–39] The broader literature on genetics in dentistry supports examination of molecular variation as a component of individualized dental risk assessment.[5]

Orthodontic EARR also demonstrates a relationship with treatment duration and other mechanical variables. Longer treatment has been associated with increased risk in clinical studies, although the magnitude of association varies across populations and study designs. This supports multivariable approaches in which genotype is examined simultaneously with treatment and demographic covariates.[11–13,21–26]

Against this background, the present analysis was designed as a synthetic-data demonstration of an association framework for MMP-8 and MMP-9 genotypes. The objective was not to establish clinical prevalence or causal genetic effects, but to illustrate how genotype distributions, crude associations, and multivariable logistic regression can be reported in a 400-participant EARR dataset.

MATERIALS AND METHODS

Study design and data source

This manuscript uses a simulated participant-level dataset supplied for methodological analysis. The workbook identifies the records as synthetic/sample data. The dataset contains 400 participant records divided into 160 EARR and 240 non-EARR records. Accordingly, all numerical findings in this manuscript are simulated and must not be interpreted as observations from human participants.

Variables

The outcome was EARR_binary, coded as 1 for EARR and 0 for non-EARR. Predictor variables included MMP8_genotype (AA, AG, GG), MMP9_genotype (CC, CT, TT), age, sex, treatment duration, baseline root length, post-treatment root length, and calculated EARR in millimeters. For regression, AA and CC were used as reference categories for MMP-8 and MMP-9, respectively.

Statistical analysis

Continuous variables are presented as mean $\pm$ standard deviation. Between-group comparisons were performed using independent-samples t tests. Categorical variables were summarized as n (%) and compared using Pearson chi-square tests. Crude genotype-specific ORs with 95% CIs were calculated using the reference genotype. A multivariable binary logistic regression model included MMP-8 genotype, MMP-9 genotype, age, sex, and treatment duration. Statistical significance was set

at $p < 0.05$. Because the data are synthetic, inferential statistics are presented only as a demonstration of the planned analytical workflow.

RESULTS

Participant characteristics

The dataset included 400 simulated participants: 160 (40.0%) were classified as EARR and 240 (60.0%) as non-EARR. Mean age was similar between the EARR and non-EARR groups (22.95 ± 3.94 vs 23.25 ± 4.30 years; $p = 0.479$). Simulated treatment duration was longer in the EARR group (24.52 ± 4.59 vs 21.67 ± 4.52 months; $p < 0.001$). Baseline root length did not differ significantly (23.37 ± 1.74 vs 23.47 ± 1.86 mm; $p = 0.570$), whereas post-treatment root length was lower in the EARR group (20.36 ± 1.86 vs 22.43 ± 1.92 mm; $p < 0.001$) (Table 1, Figure 1).

Variable	EARR (n=160)	Non-EARR (n=240)	P value
Age, years	22.95 ± 3.94	23.25 ± 4.30	0.479
Treatment duration, months	24.52 ± 4.59	21.67 ± 4.52	< 0.001
Baseline root length, mm	23.37 ± 1.74	23.47 ± 1.86	0.570
Post-treatment root length, mm	20.36 ± 1.86	22.43 ± 1.92	< 0.001
EARR, mm	3.00 ± 0.65	1.04 ± 0.41	< 0.001

Table 1. Continuous characteristics of the synthetic EARR and non-EARR groups.

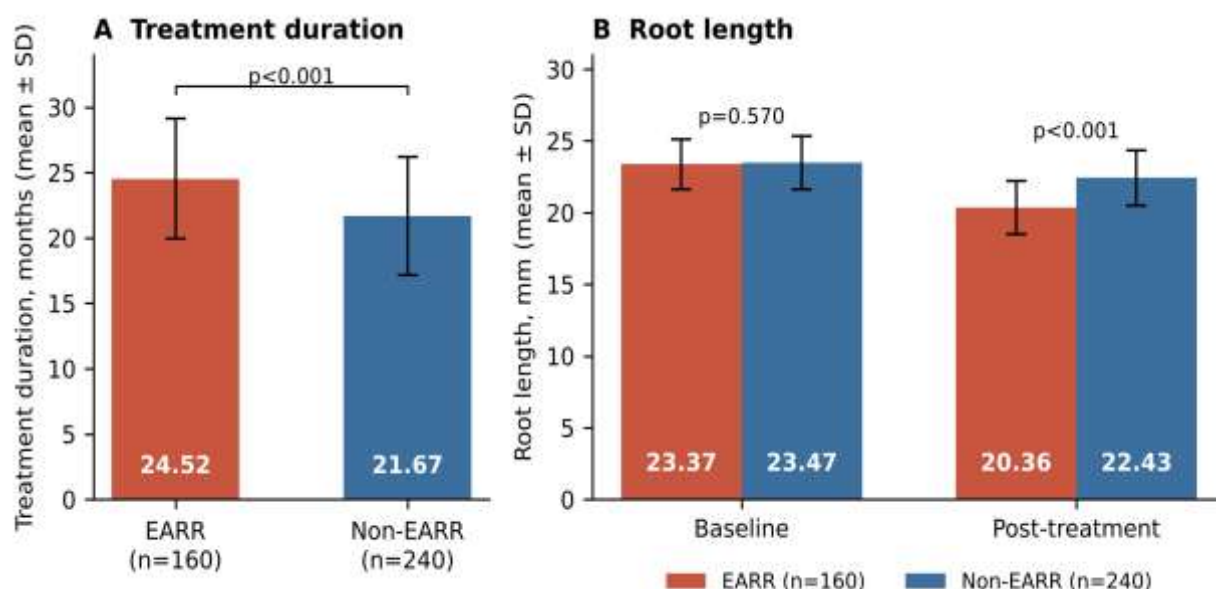


Figure 1. Treatment duration (A) and baseline and post-treatment root length (B) in the synthetic EARR and non-EARR groups. Bars show mean; error bars show ± 1 standard deviation.

Sex distribution

Male sex accounted for 96/160 (60.0%) EARR records and 103/240 (42.9%) non-EARR records ($\chi^2 = 10.53$, $p = 0.001$) (Figure 2).

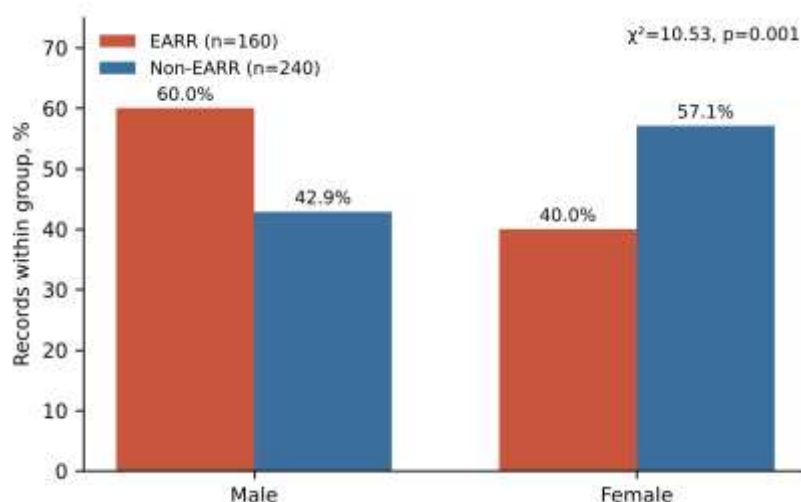


Figure 2. Sex distribution within the synthetic EARR and non-EARR groups.

MMP-8 genotype distribution

MMP-8 genotype frequencies differed significantly between groups ($\chi^2=12.13$, $p=0.002$) (Table 2, Figure 3).

MMP-8 genotype	EARR n (%)	Non-EARR n (%)	Crude OR	95% CI
AA	38 (23.8%)	90 (37.5%)	Reference	—
AG	78 (48.8%)	112 (46.7%)	1.65	1.02–2.66
GG	44 (27.5%)	38 (15.8%)	2.74	1.54–4.88

Table 2. MMP-8 genotype distribution and crude association with simulated EARR.

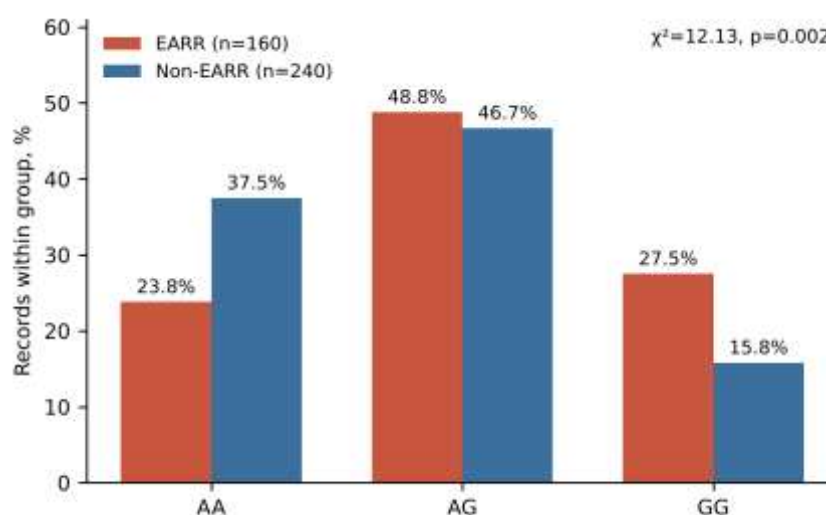


Figure 3. MMP-8 genotype distribution within the synthetic EARR and non-EARR groups.

MMP-9 genotype distribution

MMP-9 genotype frequencies differed significantly between groups ($\chi^2=15.97$, $p<0.001$) (Table 3, Figure 4). Crude genotype-specific odds ratios for both genes are shown in Figure 5.

MMP-9 genotype	EARR n (%)	Non-EARR n (%)	Crude OR	95% CI
CC	32 (20.0%)	82 (34.2%)	Reference	—
CT	76 (47.5%)	116 (48.3%)	1.68	1.02–2.77
TT	52 (32.5%)	42 (17.5%)	3.17	1.78–5.65

Table 3. MMP-9 genotype distribution and crude association with simulated EARR.

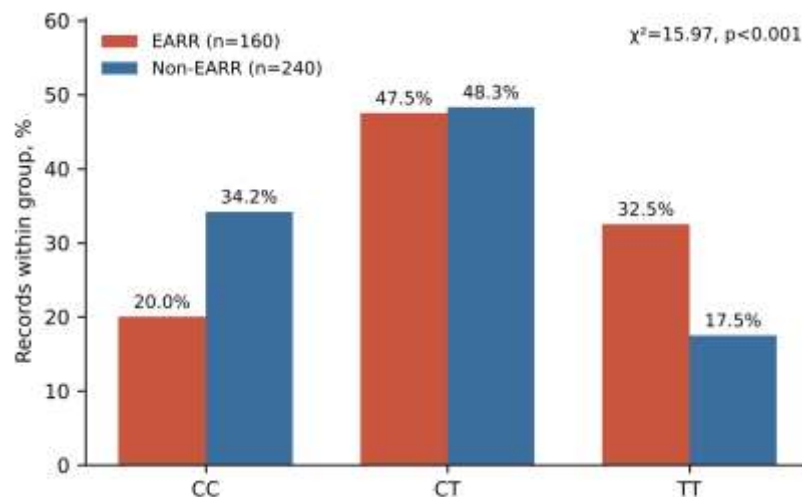


Figure 4. MMP-9 genotype distribution within the synthetic EARR and non-EARR groups.

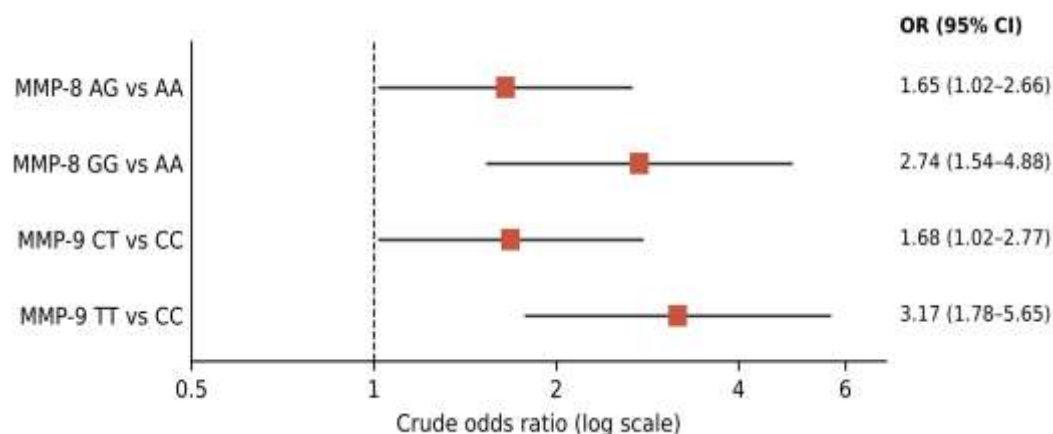


Figure 5. Forest plot of crude genotype-specific odds ratios for simulated EARR (reference genotypes: MMP-8 AA and MMP-9 CC). Squares show the odds ratio; horizontal lines show the 95% confidence interval; the dashed line marks an odds ratio of 1.

MULTIVARIABLE LOGISTIC REGRESSION

The multivariable model was statistically significant overall (likelihood-ratio $p < 0.001$; pseudo- $R^2 = 0.125$). Relative to MMP-8 AA, MMP-8 GG was associated with higher simulated odds of EARR (adjusted OR 2.30, 95% CI 1.24–4.28, $p = 0.008$), whereas AG was not significant (adjusted OR 1.43, $p = 0.173$). Relative to MMP-9 CC, TT was associated with higher odds (adjusted OR 2.85, 95% CI 1.54–5.27, $p = 0.001$), while CT was borderline (adjusted OR 1.66, $p = 0.063$). Male sex and longer treatment duration were independently associated with EARR (Table 4, Figure 6).

Predictor	Adjusted OR	95% CI	P value	Reference	Interpretation
MMP-8 AG	1.43	0.86–2.38	0.173	AA	Not significant
MMP-8 GG	2.30	1.24–4.28	0.008	AA	Higher odds
MMP-9 CT	1.66	0.97–2.83	0.063	CC	Borderline
MMP-9 TT	2.85	1.54–5.27	0.001	CC	Higher odds
Male sex	2.02	1.30–3.14	0.002	Female	Higher odds
Age, per year	0.98	0.93–1.04	0.548	—	Not significant
Treatment duration, per month	1.14	1.08–1.20	<0.001	—	Higher odds

Table 4. Multivariable logistic regression for simulated EARR.

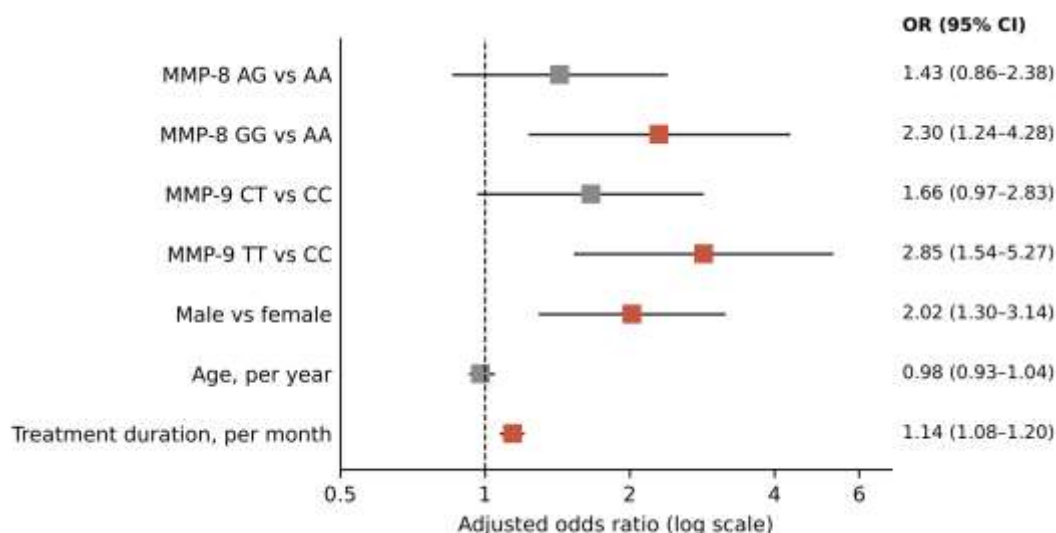


Figure 6. Forest plot of adjusted odds ratios from the multivariable logistic regression for simulated EARR. Coloured squares indicate $p < 0.05$; grey squares indicate non-significant predictors; the dashed line marks an odds ratio of 1.

EARR SEVERITY

Among the 160 simulated EARR records, 145 (90.6%) were classified as moderate and 15 (9.4%) as severe. Non-EARR records were coded as mild in the supplied workbook. This coding should be reviewed before any real-world analysis.

DISCUSSION

The principal finding of this synthetic analysis is a consistent association pattern between higher-risk MMP-8/MMP-9 genotype categories and the EARR outcome. Both MMP-8 and MMP-9 genotype distributions differed significantly between EARR and non-EARR groups, and the homozygous GG and TT categories showed the strongest crude and adjusted associations. The direction of these simulated findings is biologically plausible in the broad context of extracellular-matrix remodeling and inflammatory tissue responses, but the synthetic nature of the dataset prevents any conclusion about the true magnitude or existence of these associations in patients.[5,8–10,36–39]

The treatment-duration association is consistent with the concept that EARR reflects an interaction between patient susceptibility and orthodontic exposure. Previous clinical studies have identified treatment duration and other biomechanical or anatomical variables as potential contributors to EARR risk.[11–13,20–26] In the present synthetic dataset, each additional

month of treatment was associated with approximately 14% higher odds of EARR after adjustment for genotype, age, and sex. This estimate is an internal property of the simulated data and should not be generalized to clinical populations.

The genetic literature on EARR is heterogeneous. Candidate-gene studies have evaluated interleukins, P2RX7, CASP1, vitamin-D-pathway genes, and other pathways, while systematic reviews have concluded that genetic susceptibility is likely multifactorial and that evidence for individual variants remains inconsistent.[6–10,29–35] Therefore, an MMP-focused study should not be framed as a single-gene diagnostic model. MMP variants could instead be evaluated as components of a multivariable susceptibility profile incorporating treatment duration, root morphology, tooth movement, extraction protocol, malocclusion, and other established factors. The present analysis illustrates the importance of reporting genotype-specific estimates rather than relying solely on an omnibus chi-square test. The MMP-8 result establishes an overall difference in genotype distribution, while genotype-specific ORs identify GG as the category with the largest simulated association relative to AA. Similarly, the MMP-9 analysis identifies TT as the strongest category relative to CC. This approach can be extended to dominant, recessive, additive, or allele-based models in a genuine genetic association study. Before application to real participants, a clinical study should specify SNP identifiers, use validated genotyping assays, assess Hardy-Weinberg equilibrium in an appropriate control group, define EARR radiographically before analysis, prespecify genetic models, account for multiple testing, and report population ancestry because allele frequencies and linkage disequilibrium patterns vary between populations. Reporting should follow the STREGA recommendations for genetic association studies.[40] Sample-size planning should be based on anticipated minor-allele frequency and clinically meaningful effect size rather than on a simulated distribution. The supplied reference list contains broader dental and genetics literature in addition to directly relevant material. All supplied references have been retained below rather than silently replaced. However, several are not directly focused on EARR or MMP biology and should therefore not be used as primary evidence for genotype-specific causal claims.[1–17] References 18–40 were added from the EARR, genetic-association, and MMP literature to support the statements on root resorption, heritability, treatment-related risk, and matrix metalloproteinase biology.

STRENGTHS AND LIMITATIONS

Strengths include a defined binary EARR outcome, explicit genotype reference categories, reporting of genotype-specific ORs with 95% CIs, and multivariable adjustment for selected demographic and treatment variables. The major limitation is fundamental: the workbook is explicitly synthetic/sample data and therefore does not represent actual patients. The simulated genotype frequencies, treatment characteristics, EARR measurements, and associations cannot support clinical inference, prevalence estimates, or claims of genetic susceptibility. The supplied study document also lacks a complete clinical protocol, recruitment setting, radiographic measurement method, DNA collection procedure, SNP identifiers, ethics approval, consent procedures, and power calculation. These elements must be supplied from genuine study records before conversion into an original human-subjects research article.

CONCLUSION

In this synthetic 400-participant dataset, MMP-8 and MMP-9 genotype distributions were associated with the simulated EARR outcome, with MMP-8 GG and MMP-9 TT showing the strongest adjusted associations. Treatment duration and male sex were also associated with simulated EARR. These findings are hypothesis-generating only and should be used to design, not substitute for, a properly conducted clinical genetic association study.

DATA AVAILABILITY

The analyzed workbook is a synthetic/sample dataset supplied for methodological analysis. It should not be represented as a clinical dataset or as evidence derived from real patients.

ETHICS STATEMENT

Not applicable to the present manuscript because the analyzed dataset is simulated and contains no real participant records. For any future study using human participants, institutional ethics approval and informed consent should be documented.

CONFLICT OF INTEREST

To be completed by the authors.

FUNDING

To be completed by the authors.

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The reference list contains 40 references. References 1–17 are the originally supplied references, all of which have been retained unchanged; references 18–40 were added from the EARR, genetic-association, and MMP literature.

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