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THE IMPACT OF FLUORIDE INTAKE AND FORCE MAGNITUDE ON ORTHODONTIC TOOTH MOVEMENT

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Abstract

Aim of the Study: This study evaluated the effects of systemic fluoride intake and orthodontic force magnitude on the rate of tooth movement during space closure. Material and Method: Thirty-six patients undergoing fixed orthodontic treatment were divided into four groups based on fluoride intake and applied force: Group 1 (fluoride, 300 g), Group 2 (fluoride, 50 g), Group 3 (no fluoride, 300 g), and Group 4 (no fluoride, 50 g). Tooth movement was measured over five weeks. Statistical analysis was performed using ANOVA, with significance at $p \leq 0.05$. Results: Mean tooth movement was highest in Group 1 (1.62 ± 0.82 mm) and lowest in Group 4 (1.03 ± 0.33 mm). Differences were statistically significant only between Group 1 and Group 4 ($p = 0.0035$). No significant differences were observed between heavy and light forces or between fluoride and non-fluoride groups. Conclusions: While heavier forces resulted in greater tooth movement, the differences were not statistically significant. Fluoride intake did not significantly impact movement, suggesting a complex role in bone remodeling. These findings highlight the importance of applying controlled orthodontic forces to optimize treatment outcomes while minimizing adverse effects.

Keywords: orthodontic tooth movement, fluoride intake, force magnitude, periodontal remodeling

Introduction

Cardiovascular diseases are one of the main causes of morbidity and mortality globally, having a major impact on the health system and on the quality of life of patients [1]. In particular, myocardial infarction and heart failure are conditions in which regeneration of heart tissue is essential for restoring heart function [1,2]. However, unlike other types of tissues, the adult myocardium has a limited capacity for regeneration. Mature cardiomyocytes show extremely low mitotic activity, which causes myocardial damage to be repaired predominantly by the formation of fibrotic tissue. This ineffective repair can lead to ventricular remodeling, decreased contractile performance, and ultimately progressive heart failure [3].

Orthodontic treatment shifts teeth within the alveolar bone by triggering remodeling processes and adaptive modifications in the surrounding periodontal tissues [1]. Applying an orthodontic force triggers a tissue response resulting from the disturbance caused by the orthodontic appliance and the modeling and remodeling of the alveolar bone [1]. The

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inflammatory reaction to orthodontic tooth movement is associated with the production and release of various cytokines [2], including transforming growth factor-beta (TGF-β) and interleukin-1 beta (IL-1β), which stimulate the differentiation, function, and survival of osteoclasts, thereby contributing to bone remodeling and tooth movement [3,4]. The stimulation of bone cells is mediated by various factors, including tumor necrosis factor (TNF), receptor activator of nuclear factor-kappa ligand (RANKL), receptor activator of nuclear factor-kappa (RANK), and osteoprotegerin (OPG) [4-8].

Osteopontin (OPN) is another protein associated with bone resorption, as it promotes osteoclast adhesion to the bone matrix [9]. Gingival crevicular fluid (GCF) serves as a medium for detecting molecules involved in bone modeling and remodeling during orthodontic tooth movement (OTM) [9].

The pace of tooth movement is influenced by various factors, such as the force's strength, orientation, spread, and application time. Additionally, it is affected by the tooth's initial shift and the biomechanical stresses, strains, and physiological changes occurring in the periodontium [10].

Fluoride is essential for dental health, particularly during adolescence—a critical period for tooth development. The recommended daily fluoride intake for adolescents aged 12 to 20 years is 3 mg for males and females [11]. Fluoride supplementation is typically considered for individuals residing in areas where the drinking water contains less than 0.6 parts per million (ppm) of fluoride [12]. Fluoride supplements are available in various forms, including chewable tablets, lozenges, and drops. The most commonly used form is sodium fluoride [11,12]. Chewable tablets are often preferred for older children and adolescents due to ease of use. Studies have demonstrated that supervised regular use of fluoride mouth rinses in children and adolescents is associated with a significant reduction in dental caries [12,13]. However, the necessity and dosage of fluoride supplements should be evaluated based on individual caries risk and existing fluoride exposure. Excessive fluoride intake can lead to dental fluorosis, and it's crucial to adhere to recommended dosages [13,14].

The highest fluoride concentration in the human body is found in calcified tissues, playing a crucial role in bone metabolism. Over time, the fluoride levels in these tissues tend to increase [11]. The role of fluoride in biological processes remains uncertain. Although it is widely recognized for its impact on osteoblast function [15], its influence on clast cell activity, critical for orthodontic tooth movements, remains a debated topic. Some research suggests that fluoride slows tooth movement in animal models [16], but its effects on humans have yet to be explored. This study seeks to determine whether differing fluoride concentration intake alters the rate of orthodontic tooth movement when subjected to both strong and gentle forces.

We used Discovery conventional brackets: manufactured by DENTAURUM GmbH & Co., distinguished by their durability, precision, and optimal bonding strength, making them a preferred choice in modern orthodontics [17].

Constructed from high-grade stainless steel, Discovery brackets offer superior strength and resistance to deformation, ensuring long-term stability. Their low-profile, rounded design enhances patient comfort, while a laser-structured base and torque-in-the-base technology facilitate precise tooth movement and optimal biomechanical control. Additionally, the brackets are designed to minimize soft tissue irritation, further improving the patient experience [17,18].

Engineered for efficient archwire sliding, the bracket slot reduces friction, optimizes tooth movement, and enhances treatment efficiency. Due to their reliability, biomechanical precision, and patient-friendly design, Discovery brackets are widely utilized in fixed orthodontic treatments and remain a preferred choice among orthodontists globally.

Material and method

The study evaluated 36 patients with fixed orthodontic appliances, in whom the sliding of the upper first premolar was initiated to close the remaining space after the extraction of the upper second premolar, in cases of maxillary DDM associated with various other anomalies.The patients were categorized into two groups based on whether they consumed fluoride. Each group was further divided to receive either a light or heavy orthodontic force, resulting in four subgroups with nine participants each: Group 1 (fluoride intake, heavy force), Group 2 (fluoride intake, light force), Group 3 (no fluoride intake, heavy force), and Group 4 (no fluoride intake, light force) (Fig. 1).

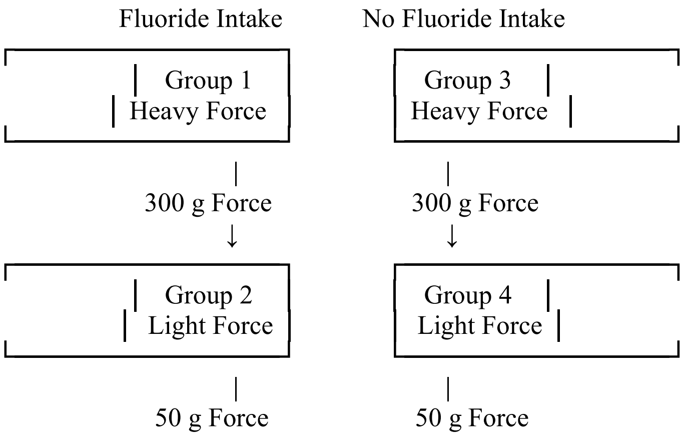

Fig.1 Group design

We utilized Discovery conventional brackets with a standard slot of 0.022” x 0.028” (inch), the Roth prescription, renowned for their exceptional quality and advanced orthodontic engineering [17]. Brackets were affixed to the buccal surfaces of the upper first permanent molars and first premolars. Brackets were uniformly applied to the enamel surfaces, ensuring proper alignment by adhering to positioning guidelines and centering them on the buccal surfaces.

Orthodontic forces in the study were categorized as light (50 g or 0.49 N) and heavy (300 g or 2.94 N) [19]. Forces were meticulously controlled to ensure consistency throughout the treatment process.

Tooth movement was induced by applying either a heavy (300 g in Groups 1 and 3) or a light (50 g in Groups 2 and 4) vestibular tipping force on the patient’s maxillary first premolars for five weeks.

Patients in Groups 2 and 4 received a 50 g buccally-directed force applied to their premolars via a 0.016-inch Beta III Titanium Cantilever Spring (3M Unitek, Monrovia, CA, USA). Meanwhile, subjects in Groups 1 and 3 were subjected to a 300 g buccally-directed force on their premolars, delivered by a 0.017 x 0.025-inch beta-titanium-molybdenum alloy cantilever spring (Beta III Titanium, 3M Unitek, Monrovia, CA, USA) [10].

The magnitude of force was carefully measured and verified using a strain gauge (Dentaurum). To prevent occlusal trauma to the maxillary first premolars during the experiment, a light-cured composite resin (Herculite, Kerr Corporation, California, USA) was applied to the occlusal surfaces of the mandibular first permanent molars, effectively increasing the vertical dimension of the participants [10].

In all cases, the distance between the center of the bracket slot bonded to the first premolar and the mesial point of the molar tube was measured at the start of the traction and after 5 weeks.

All data analyses were conducted using the Statistical Package for the Social Sciences (SPSS for Windows, version 16, SPSS Inc., Chicago, IL, USA). A Univariate Analysis of Variance (ANOVA) was applied to compare group differences. A p-value of ≤ 0.05 was considered the threshold for statistical significance.

Results

The patients' ages ranged from 11.6 to 21.2 years, with a mean age of 15.2 years (Table 1).

Table 1. Tooth movement

	Minimum	Maximum	Mean	SD	SE
Group 1	0.37	3.17	1.62	0.82	0.07
Group 2	0.68	1.92	1.24	0.34	0.03
Group 3	0.75	2.89	1.31	0.51	0.06
Group 4	0.39	1.86	1.03	0.33	0.04

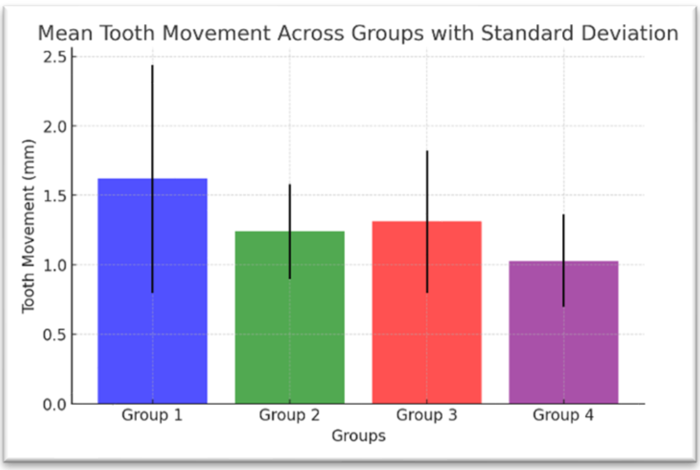

Fig.2 Mean tooth movements

Differences in tooth movement were significant only when comparing Groups 1 and 4 (p=0.0035). No significant differences were observed between heavy and light forces (Fig. 2).

Discussions

This study aimed to assess the influence of systemic fluoride intake and varying orthodontic force magnitudes on the rate of tooth movement during space closure. Our findings demonstrated that heavy forces (300 g) resulted in greater tooth displacement than light forces (50 g); however, the differences were not statistically significant. Additionally, systemic fluoride intake did not significantly impact tooth movement rate. These results align with some existing studies but contrast with others, highlighting the complex nature of orthodontic biomechanics and individual biological variability.

The rate of orthodontic tooth movement is primarily influenced by the magnitude of force applied to the teeth. Previous research suggests that higher forces can accelerate tooth displacement by stimulating osteoclastic activity in the periodontal ligament (PDL) and promoting alveolar bone resorption. Owman-Moll et al. reported that a fourfold increase in force resulted in a 50% increase in the rate of tooth movement [20]. However, our study did not observe a significant difference between the heavy and light force groups, suggesting that force magnitude alone may not be the sole determinant of orthodontic tooth movement.

One possible explanation for these findings is the phenomenon of the "optimal force threshold," wherein excessive forces may not always translate to faster movement due to increased hyalinization within the periodontal ligament (PDL). Studies have shown that excessive force application may induce areas of necrosis in the PDL, temporarily halting tooth movement and necessitating a period of tissue repair before movement resumes [21].

Fluoride is well-documented for its effects on dental hard tissues, particularly in enhancing enamel remineralization and reducing caries incidence. However, its impact on alveolar bone remodeling and orthodontic tooth movement remains controversial. Some animal studies have suggested that fluoride can slow tooth movement by inhibiting osteoclast activity, thereby reducing bone resorption [16]. Conversely, other studies have found minimal or no effect of fluoride on tooth movement in human subjects.

In our study, fluoride intake did not significantly alter the rate of tooth movement. Several factors may contribute to this, including the dosage and duration of fluoride consumption, individual metabolic differences, and the interaction of systemic and local factors in bone remodeling. Additionally, fluoride enhances osteoblast activity [3,15], which could offset its potential inhibitory effects on osteoclast function and neutral effects on tooth movement.

Orthodontic tooth movement is a biologically complex process that depends on multiple factors beyond force magnitude and fluoride intake. These include genetic predisposition, systemic conditions, and metabolic factors that can significantly impact how patients respond to orthodontic forces. Younger patients typically experience faster tooth movement due to increased bone turnover and higher cellular activity in the PDL. Cytokines such as IL-1β, TNF-α, and RANKL mediate osteoclast activity and bone resorption. The balance between pro-resorptive (RANKL) and anti-resorptive (OPG) factors determines the efficiency of tooth movement [22]. Excessive forces can lead to localized ischemia and tissue necrosis in the PDL, temporarily delaying tooth movement until repair processes restore normal function.

The results of this study provide valuable insights for orthodontic treatment planning. While heavier forces may offer a theoretical advantage in accelerating space closure, they do not necessarily produce significantly greater movement in clinical practice. This supports moderate, controlled forces to minimize adverse effects such as root resorption and patient discomfort while achieving effective treatment outcomes. Findings suggest that standard fluoride intake supplements do not interfere with orthodontic tooth movement. However, excessive fluoride consumption should still be monitored, as high systemic fluoride levels could alter bone metabolism in ways that may not be immediately apparent.

Despite the insights gained, our study has some limitations that should be acknowledged: a larger cohort may be necessary to detect subtle differences in tooth movement between force magnitudes and fluoride intake groups. Future studies should assess varying fluoride concentrations to determine potential dose-dependent effects on tooth movement. A longer observation period could provide more comprehensive data on the cumulative impact of fluoride and force magnitude on orthodontic tooth movement.

Conclusions

This study found that while heavier forces resulted in greater tooth movement, differences were not significant. Fluoride intake also had no significant impact, indicating a complex role in bone remodeling. Force magnitude alone does not determine treatment outcomes, underscoring the need for controlled application to optimize movement and minimize risks. Fluoride's influence remains uncertain, requiring further research. Future studies should explore long-term and dose-dependent effects to refine orthodontic treatment strategies.

Bibliography

1. Krishnan V, Davidovitch Z. *Cellular, molecular, and tissue-level reactions to orthodontic force.* **Am J Orthod Dentofacial Orthop.** 2006;129(4):469.e1-32. doi:10.1016/j.ajodo.2005.10.007.
2. Ren Y, Hazemeijer H, de Haan B, Qu N, de Vos P. *Cytokine profiles in crevicular fluid during orthodontic tooth movement of short and long durations.* **J Periodontol.** 2007;78(3):453-458. doi:10.1902/jop.2007.060261.
3. Hofbauer LC, Lacey DL, Dunstan CR, Spelsberg TC, Riggs BL, Khosla S. *Interleukin-1beta and tumor necrosis factor-alpha, but not interleukin-6, stimulate osteoprotegerin ligand gene expression in human osteoblastic cells.* **Bone.** 1999;25(3):255-259. doi:10.1016/s8756-3282(99)00162-3.
4. Yao Z, Xing L, Qin C, Schwarz EM, Boyce BF. *Osteoclast precursor interaction with bone matrix induces osteoclast formation directly by an interleukin-1-mediated autocrine mechanism.* **J Biol Chem.** 2008;283(15):9917-9924. doi:10.1074/jbc.M706415200.
5. Yan T, Riggs BL, Boyle WJ, Khosla S. *Regulation of osteoclastogenesis and RANK expression by TGF-beta1.* **J Cell Biochem.** 2001;83(2):320-325. doi:10.1002/jcb.1200.
6. Nakao K, Goto T, Gunjigake KK, Konoo T, Kobayashi S, Yamaguchi K. *Intermittent force induces high RANKL expression in human periodontal ligament cells.* **J Dent Res.** 2007;86(7):623-628. doi:10.1177/154405910708600708.
7. Yamaguchi M. *RANK/RANKL/OPG during orthodontic tooth movement.* **Orthod Craniofac Res.** 2009;12(2):113-119. doi:10.1111/j.1601-6343.2009.01444.x.
8. Nishijima Y, Yamaguchi M, Kojima T, Aihara N, Nakajima R, Kasai K. *Levels of RANKL and OPG in gingival crevicular fluid during orthodontic tooth movement and*

effect of compression force on releases from periodontal ligament cells in vitro. **Orthod Craniofac Res.** 2006;9(2):63-70. doi:10.1111/j.1601-6343.2006.00340.x.

9. Reinholt FP, Hultenby K, Oldberg A, Heinegård D. *Osteopontin--a possible anchor of osteoclasts to bone.* **Proc Natl Acad Sci U S A.** 1990;87(12):4473-4475. doi:10.1073/pnas.87.12.4473.
10. Karadeniz EI, Gonzales C, Elekdag-Turk S, et al. *The effect of fluoride on orthodontic tooth movement in humans. A two- and three-dimensional evaluation.* **Aust Orthod J.** 2011;27(2):94-101.
11. Stepnick RJ, Nakata TM, Zipkin I. *The effects of age and fluoride exposure on fluoride, citrate and carbonate content of human cementum.* **J Periodontol.** 1975;46(1):45-50. doi:10.1902/jop.1975.46.1.45.
12. Flood S, Asplund K, Hoffman B, Nye A, Zuckerman KE. *Fluoride Supplementation Adherence and Barriers in a Community Without Water Fluoridation.* **Acad Pediatr.** 2017;17(3):316-322. doi:10.1016/j.acap.2016.11.009.
13. Ismail AI, Hasson H. *Fluoride supplements, dental caries and fluorosis: a systematic review.* **J Am Dent Assoc.** 2008;139(11):1457-1468. doi:10.14219/jada.archive.2008.0071.
14. Ismail AI. *Fluoride supplements: current effectiveness, side effects, and recommendations.* **Community Dent Oral Epidemiol.** 1994;22(3):164-172. doi:10.1111/j.1600-0528.1994.tb01835.x.
15. Kassem M, Mosekilde L, Eriksen EF. *Effects of fluoride on human bone cells in vitro: differences in responsiveness between stromal osteoblast precursors and mature osteoblasts.* **Eur J Endocrinol.** 1994;130(4):381-386. doi:10.1530/eje.0.1300381.
16. Gonzales C, Hotokezaka H, Karadeniz EI, et al. *Effects of fluoride intake on orthodontic tooth movement and orthodontically induced root resorption.* **Am J Orthod Dentofacial Orthop.** 2011;139(2):196-205. doi:10.1016/j.ajodo.2009.05.029.
17. Redlich M, Mayer Y, Harari D, Lewinstein I. *In vitro study of frictional forces during sliding mechanics of "reduced-friction" brackets.* **Am J Orthod Dentofacial Orthop.** 2003;124(1):69-73. doi:10.1016/s0889-5406(03)00238-5.
18. Daratsianos N, Jäger A, Eliades T, Bourauel C. *In vitro cyclic shear fatigue of the bracket-adhesive complex: a pilot study.* **J Orofac Orthop.** 2012;73(2):138-150. doi:10.1007/s00056-011-0069-5.
19. Yee JA, Türk T, Elekdağ-Türk S, Cheng LL, Darendeliler MA. *Rate of tooth movement under heavy and light continuous orthodontic forces.* **Am J Orthod Dentofacial Orthop.** 2009;136(2):150.e1-150.e9. doi:10.1016/j.ajodo.2009.03.026.
20. Owman-Moll P, Kurol J, Lundgren D. *The effects of a four-fold increased orthodontic force magnitude on tooth movement and root resorptions: an intra-individual study in adolescents.* **Eur J Orthod.** 1996;18(3):287-294. doi:10.1093/ejo/18.3.287.
21. Gonzales C, Hotokezaka H, Yoshimatsu M, et al. *Force magnitude and duration effects on amount of tooth movement and root resorption in the rat molar.* **Angle Orthod.** 2008;78(3):502-509. doi:10.2319/052007-240.1.
22. Patkar S, Sharan R. *An optimization framework for network annotation.* **Bioinformatics.** 2018;34(13):i502-i508. doi:10.1093/bioinformatics/bty236.