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INVESTIGATING THE POSSIBILITY OF DEVELOPING ENAMEL CRACKS RELATED TO DEBONDING PROCEDURE

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Abstract

Aim: This study aimed to investigate whether cracks observed post-orthodontic treatment occur during bracket removal, if pre-existing cracks progress, or whether the normal lifestyle of untreated individuals causes more enamel cracks. **Materials and Methods:** The study included 54 individuals (27 experimental/treated, 27 control/untreated) with 216 upper incisors. Intraoral photographs were taken for the experimental group immediately before and after debonding, and for the control group at baseline and after 1 year. Photographs were captured using camera with macro lens and standardized settings (ISO 3200, F22, 1/160) with a polarizing lens and transillumination device. Crack lengths were measured using ImageJ software. Statistical analyses (Mann-Whitney U, Wilcoxon, Chi-square tests; $p<0.05$) were performed with SPSS 22.0. **Results:** The control group showed significantly higher new crack formation (8.3%) than the debonding group (0.9%) ($p<0.05$). Upper central incisors had higher crack susceptibility (14.8%) compared to lateral incisors (0%). No significant gender-based difference was observed ($p>0.05$). While crack counts remained unchanged in the experimental group pre-/post-debonding ($p>0.05$), the control group exhibited increased cracks after 1 year ($p<0.05$). **Conclusion:** Orthodontic debonding with appropriate techniques does not significantly increase enamel crack formation. Functional stresses and parafunctional habits are primary contributors to crack progression.

Keywords: Enamel Crack, Bracket Debonding, Dental Photograph.

INTRODUCTION

Enamel cracks, commonly referred to as 'craze lines,' are superficial and asymptomatic lines that appear on the surface of tooth enamel. These lines typically result from natural aging or factors such as bruxism. Cracked teeth can pose a diagnostic challenge for clinicians as they may mimic various conditions. Continuous physiological stress, along with pathological strains from trauma or iatrogenic causes, can lead to the formation of micro-cracks in teeth.

The persistence of intense stress on the tooth following the formation of these micro-cracks can complicates periodontal, pulpal, biomechanical, and dental treatments or can even lead to tooth fractures [1, 3]. Multiple morphological, physical, and iatrogenic factors can contribute to crack formation. Symptoms vary, making diagnosis difficult and potentially leading to long-term patient discomfort.

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Late diagnosis can worsen the prognosis, whereas early detection allows for tooth reinforcement using dental restorations. Possible etiological factors include repetitive heavy masticatory forces, premature occlusal contacts, physical trauma, resorptions, caries, weakened tooth structure due to substance loss, iatrogenic causes, and parafunctional habits [3, 5].

Objectively detecting the crack line or width clinically is quite challenging. Diagnosis and treatment planning include; symptoms, pulp and periodontal status, history of dental trauma, restorative procedures, parafunctional habits, and clinical visibility of the crack [5]. Although auxiliary methods like magnification, transillumination, and staining tests can aid diagnosis, estimating the width and direction of the crack is often not possible [6, 7].

Posterior teeth, particularly mandibular first and second molars, are most frequently affected. Symptoms can vary depending on the crack's depth, direction, and the involved tissues [6]. Tooth fractures, a common cause of tooth loss, often begin as enamel cracks [8].

Several classifications have been proposed, but no consensus exists due to inconsistent symptoms and the fact that cracks are often observed clinically, not microscopically [4, 9–11]. Some researchers suggest cracks confined to enamel do not require treatment, whereas those involving dentin are structural and requires treatment [12, 13]. The American Association of Endodontists' 2008 classification defines five types: craze line, cusp fracture, cracked tooth, split tooth, and vertical root fracture [14].

Preserving enamel surface structure is a key goal for orthodontists. Debonding and adhesive cleanup sequences are often empirically based, with limited scientific research on their effects. Enamel damage has been attributed to abrasive prophylaxis, acid-etching, enamel fractures during debonding [7, 15].

Orthodontic treatment has the potential to cause enamel damage through abrasive cleaning before etching, the acid-etching process itself, enamel fractures during forceful debonding, mechanical removal of composite residues with rotary instruments, and rebonding procedures and rotary instrument cleanup. Plaque accumulation can lead to demineralization, and ceramic brackets may cause wear.

Structural damage can also occur during interproximal enamel reduction (stripping) [16]. Debonding methods involve deforming bracket wings/bases or applying shear force to the adhesive. Some clinicians consider ligature cutters for metal brackets to cause the least damage [17, 18]. The cumulative effect of pumice prophylaxis, bonding, debonding, and cleanup can lead to enamel loss and is considered a primary factor in the formation or propagation of enamel cracks [19].

Ideally debonding should not cause cracks or fractures; techniques promoting failure at the bracket-adhesive interface are optimal as residual adhesive minimizes enamel loss risk [28]. However, if a pre-existing crack is present at the adhesive-enamel interface, enamel loss can occur during debonding [20, 21].

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METHOD

This clinical study was designed as a prospective comparative analysis. Ethical approval was granted by the Istanbul Okan University Ethics Committee (Decision No: 176, Date: 03.04.2024). All participants provided written informed consent prior to enrolment.

A total of 54 individuals (28 male, 26 female) with a mean age of 23.5 ± 5.67 years (range: 12-35) were included. Participants were allocated into two groups: Debonding Group (Experimental): Twenty-seven patients (13 male, 14 female; mean age 21.89 ± 5.90) who had completed fixed orthodontic treatment with metal brackets bonded to the maxillary incisors. Control Group: Twenty-seven individuals (15 males, 12 females; mean age 24.52 ± 4.95) with no history of orthodontic treatment. For both groups, only the four maxillary incisors (teeth #11, #12, #21, #22) were evaluated, in a total of 216 teeth analyzed. Inclusion criteria for the Debonding Group were: systemically healthy individuals, fully erupted maxillary incisors and completion of fixed orthodontic treatment.

Exclusion criteria for all participants included: contraindications to orthodontic treatment, presence of parafunctional habits (e.g., bruksism) and existing restorations or endodontic treatment on the maxillary incisors.

Debonding Procedure

Metal brackets (American Orthodontics, Sheboygan, WI, USA) had been bonded using a light-cured composite adhesive (BracePaste® Adhesive, American Orthodontics). Debonding was performed by a single experienced orthodontist using a Griffin debonding plier.

A controlled vertical force was applied in a gingivo-incisal direction to debond the bracket from the tooth surface at the bracket-adhesive interface (Fig. 1, 2). Critically, adhesive remnant was not removed from the enamel surface prior to post-debonding photography to isolate the effect of the debonding force from subsequent cleaning procedures.

Figure (1): Griffin plier

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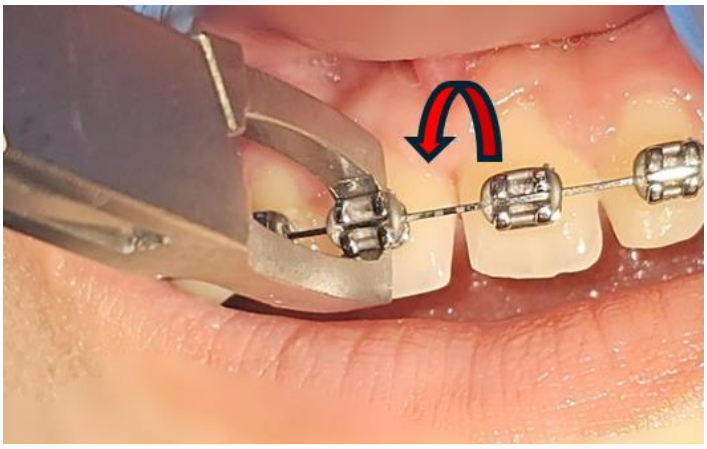

Figure (2): Vertical force (gingivo-incisal direction)

Photographic Documentation and Crack Visualization

Standardized intraoral photographs were captured using a digital SLR camera (EOS 700D, Canon Inc., Tokyo, Japan) equipped with a 100mm macro lens (Canon Inc.) and a polarizing filter (K&F Concept). Camera settings were fixed at manual focus (1:1 magnification), ISO 3200, F22 aperture, and 1/160s shutter speed. Cheek retractors were used for access, and the incisor surfaces were dried. To enhance the detection of enamel cracks, an external transillumination device (I.C. LERCHER DIA-STICK, I.C. Lercher GmbH, Austria) was employed instead of the camera flash (Fig. 3). The light source was positioned to illuminate the teeth from the proximal surfaces (mesial or distal), making cracks more visible as light-scattering lines (Fig. 4) [7, 22].

Figure (3): (I.C. LERCHER DIA-STICK, I.C. Lercher GmbH, Austria)

Figure (4): The light source to illuminate the teeth from the proximal surfaces

Tianjin Daxue Xuebao (Ziran Kexue yu Gongcheng Jishu Ban)/
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Imaging Timeline

Debonding Group: Photographs were taken immediately before (T0) and after (T1) the bracket debonding procedure.

Control Group: Baseline photographs (T0) were taken, followed by a second set of photographs one year later (T1) to assess the natural incidence of crack formation/progression under functional conditions.

Crack Measurement Protocol

The open-source image analysis software ImageJ (National Institutes of Health, Bethesda, MD, USA) was utilized as a visualization and measurement aid to facilitate this comparison. Within the software, the freehand line tool was used to trace and highlight individual crack paths on the digital images, allowing for clear delineation and counting of each crack line (Figs. 5, 6). While the software permits quantitative length measurement in millimeters, its primary function in this study was to objectively confirm the presence of cracks and assist in the side-by-side evaluation of T0 and T1 states.

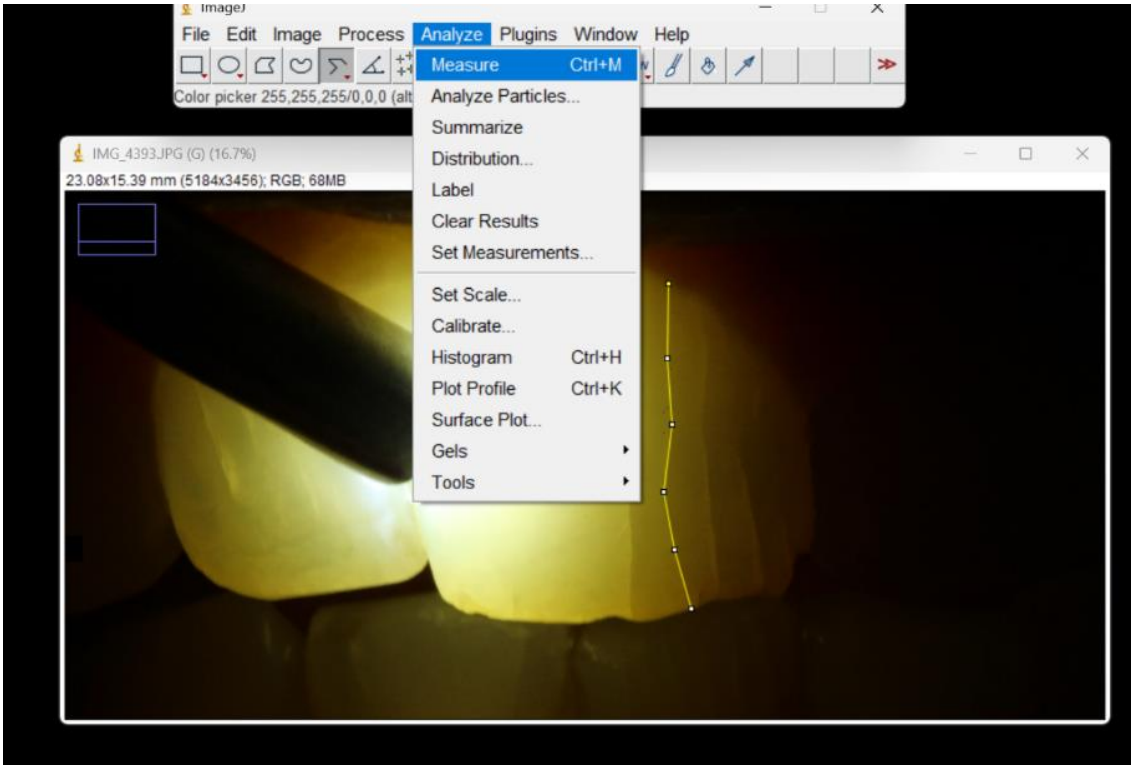

Figure (5): Freehand line tool to trace individual crack

Tianjin Daxue Xuebao (Ziran Kexue yu Gongcheng Jishu Ban)/
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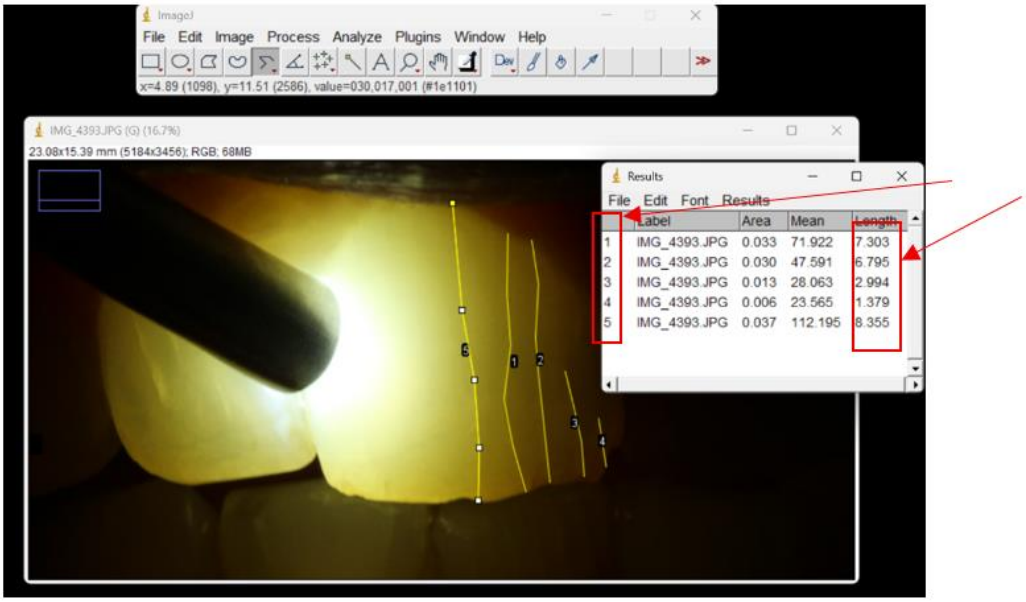

Figure (6): Many cracks selection with mm lengths

By superimposing the visual data from both time points, examiners could definitively categorize the outcome for each tooth. A crack was recorded as "new" only if it was absent in the T0 photograph but clearly present in T1. An "increase in length" was noted when a crack identifiable at T0 demonstrated visible extension at T1, as illustrated in the comparative example (Fig. 7). All assessments were performed by a single, calibrated examiner to ensure consistency.

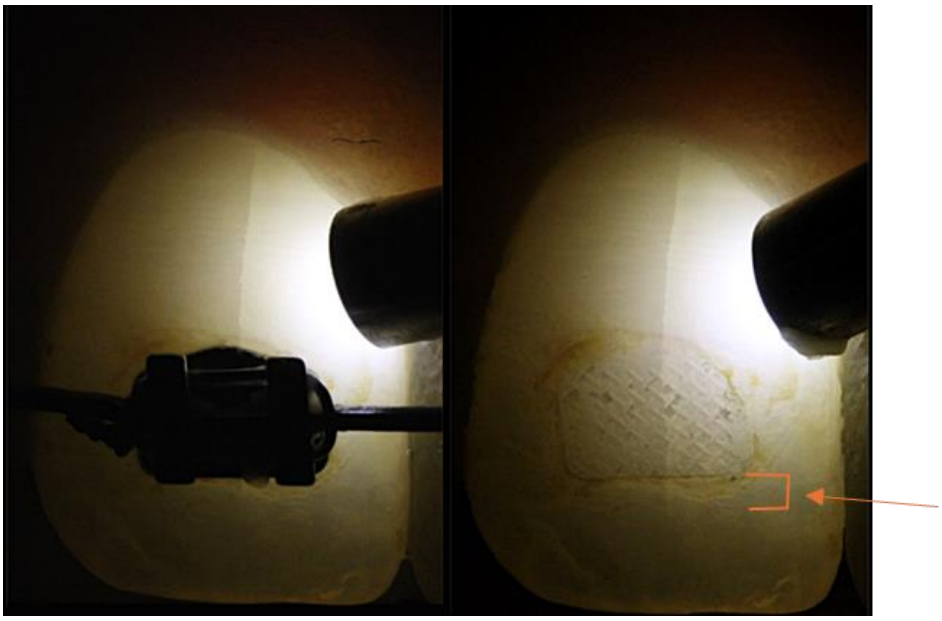

Figure (7): An increase in the crack length

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Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 22 (IBM Corp., Armonk, NY, USA). The normality of data distribution was assessed with the Kolmogorov-Smirnov test. Descriptive statistics (mean, standard deviation, median, frequency) were calculated. For inter-group comparisons of quantitative data, the Student t-test (normal distribution) or Mann-Whitney U test (non-normal distribution) was used. Intra-group comparisons (T0 vs. T1) were performed using the Wilcoxon signed-rank test. Comparisons of qualitative data (crack change categories) were conducted using the Chi-square test, Fisher-Freeman-Halton Exact test, or Yates' Continuity Correction, as appropriate. A p-value of less than 0.05 was considered statistically significant.

RESULTS

There was no statistically significant difference between the groups in terms of mean age and sex distribution (p>0.05), as seen in Table (1).

Table (2) shows that the crack counts at T0 and T1 times in the Control group were statistically significantly higher than those in the Experiment group (p:0.004; p:0.001; p<0.05). In the Experiment Group, no statistically significant change was observed at time T1 compared to the crack count at time T0 (p:0.317; p>0.05). In the Control Group, the increase observed at time T1 compared to the crack count at time T0 was statistically significant (p:0.003; p<0.05).

Table 1: Evaluation of Groups by Age and Gender

	Experimental	**Control**	**p**
Mean Age ± SD	21.89±5.90	24.52±4.95	[1]0.082
Sex, n (%)			
Male	13 (%48.1)	15 (%55.6)	[2]0.785
Female	14 (%51.9)	12 (%44.4)	

[1]Student’s t-test [2]Continuity (Yates) correction

Table 2: Evaluation of Groups by Number of Cracks

	Experimental(n=108)	**Control (n=108)**	**[1]p**
	Mean ± SD **(Min-Max)**	Mean ± SD **(Min-Max)**	
T0	0.94±1.18 (1) (0-7)	1.34±1.28 (1) (0-7)	0.004*
T1	0.94±1.22 (1) (0-7)	1.42±1.31 (1) (0-7)	0.001*
[2]p	0.317	0.003*	

[1]Mann Whitney U Test [2]Wilcoxon signed-rank test *p<0.05

While 94.4% of the teeth in the Experiment group had no new crack formation, crack length increased in 4.6% and a new crack formed in only 1 tooth (0.9%). Although 87% of the teeth in the Control group had no new crack formation, crack length increased in 4.6% and new cracks formed in 8.3%. There was a statistically significant difference between the groups in terms of crack changes at time T1 compared to time T0 (p:0.035;

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p<0.05). The rate of new crack formation in the Control group (8.3%) was significantly higher than that in the Experiment group (0.9%), Table (3).

Table (4) demonstrated that there was no statistically significant difference between the groups in terms of crack changes at T1 compared to T0 for tooth number 11 (p:0.474; p>0.05). In the Experiment group, 85.2% of tooth number 11 had no new crack formation, while crack length increased in 11.1% and a new crack formed in only 1 tooth (0.9%). In the Control group, 74.1% of tooth number 11 had no new crack formation, while crack length increased in 11.1% and new cracks formed in 14.8%.

There was no statistically significant difference between the groups in terms of crack changes at T1 compared to T0 for tooth number 12 (p:0.111; p>0.05). While none (100%) of tooth number 12 in the Experiment group had new crack formation, in the Control group, 85.2% of tooth number 12 had no new crack formation, crack length increased in 3.7% and new cracks formed in 11.1%.

There was no statistically significant difference between the groups in terms of crack changes at T1 compared to T0 for tooth number 21 (p:0.610; p>0.05). In the Experiment group, 92.6% of tooth number 21 had no new crack formation, while crack length increased in 7.4%. In the Control group, 88.9% of tooth number 21 had no new crack formation, while crack length increased in 3.7% and new cracks formed in 7.4%. None (100%) of tooth number 22 in both the Experiment and Control groups had new crack formation. Within the Experiment group, as indicated in Table (5), there was no statistically significant difference between tooth groups in terms of crack changes at T1 compared to T0 (p:0.080; p>0.05). In tooth number 11, 85.2% had no new crack formation, while crack length increased in 11.1% and new cracks formed in 3.7%. In tooth number 21, 92.6% had no new crack formation, while crack length increased in 7.4%. In teeth numbers 12 and 22, none had new crack formation. For the Control group, there was no statistically significant difference between tooth groups in terms of crack changes at T1 compared to T0 (p:0.139; p>0.05). In tooth number 11, 74.1% had no new crack formation, while crack length increased in 11.1% and new cracks formed in 14.8%. In tooth number 12, 85.2% had no new crack formation, while crack length increased in 3.7% and new cracks formed in 11.1%. In tooth number 21, 88.9% had no new crack formation, while crack length increased in 3.7% and new cracks formed in 7.4%. In tooth number 22, none had new crack formation.

Table (6) reveals that among males, there was no statistically significant difference between the groups in terms of crack changes at T1 compared to T0 in teeth (p:0.090; p>0.05). In the Experiment group, 94.2% of teeth had no new crack formation, while crack length increased in 5.8%. In the Control group, 88.3% of teeth had no new crack formation, while crack length increased in 3.3% and new cracks formed in 8.3%. Among females, there was no statistically significant difference between the groups in terms of crack changes at T1 compared to T0 in teeth (p:0.244; p>0.05). In the Experiment group, 94.6% of teeth had no new crack formation, while crack length increased in 3.6% and new cracks formed in 1.8%. In the Control group, 85.4% of teeth had no new crack formation, while crack length increased in 6.3% and new cracks formed in 8.3%.

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Within the Experiment group (Table 7), there was no statistically significant difference between sexes in terms of crack changes at T1 compared to T0 in teeth (p:0.830; p>0.05). In males, 94.2% had no new crack formation, while crack length increased in 5.8%. In females, 94.6% had no new crack formation, while crack length increased in 3.6% and new cracks formed in 1.8%. Within the Control group, there was no statistically significant difference between sexes in terms of crack changes at T1 compared to T0 in teeth (p:0.830; p>0.05). In males, 88.3% had no new crack formation, while crack length increased in 3.3% and new cracks formed in 8.3%. In females, 85.4% had no new crack formation, while crack length increased in 6.3% and new cracks formed in 8.3%.

Table 3: Evaluation of Crack Changes from Time T0 to Time T1 Between Groups

Change from T0-T1	Experimental (n=108)	Control (n=108)	p
	n (%)	n (%)	
No new crack formation	102 (%94.4)	94 (%87.0)	
Increase in crack length	5 (%4.6)	5 (%4.6)	0.035*
New crack formation	1 (%0.9)	9 (%8.3)	

Chi-square test *p<0.05

Table 4: Evaluation of Crack Changes from Time T0 to Time T1 by Individual Teeth in Each Group

Tooth no.		Experimental (n=27)	Control (n=27)	p
		n (%)	n (%)	
11	No new crack formation	23 (%85.2)	20 (%74.1)	
	Increase in crack length	3 (%11.1)	3 (%11.1)	0.474
	New crack formation	1 (%3.7)	4 (%14.8)	
12	No new crack formation	27 (%100)	23 (%85.2)	
	Increase in crack length	0 (%0)	1 (%3.7)	0.111
	New crack formation	0 (%0)	3 (%11.1)	
21	No new crack formation	25 (%92.6)	24 (%88.9)	
	Increase in crack length	2 (%7.4)	1 (%3.7)	0.610
	New crack formation	0 (%0)	2 (%7.4)	
22	No new crack formation	27 (%100)	27 (%100)	-

Fisher Freeman Halton Exact Test

Table 5: Evaluation of Crack Changes from Time T0 to Time T1 by Tooth Within Each Group

		Tooth 11	Tooth 12	Tooth 21	Tooth 22	p
		n (%)	n (%)	n (%)	n (%)	
Experimental	No new crack formation	23 (%85.2)	27 (%100)	25 (%92.6)	27 (%100)	
	Increase in crack length	3 (%11.1)	0 (%0)	2 (%7.4)	0 (%0)	0.080
	New crack formation	1 (%3.7)	0 (%0)	0 (%0)	0 (%0)	
Control	No new crack formation	20 (%74.1)	23 (%85.2)	24 (%88.9)	27 (%100)	
	Increase in crack length	3 (%11.1)	1 (%3.7)	1 (%3.7)	0 (%0)	0.139
	New crack formation	4 (%14.8)	3 (%11.1)	2 (%7.4)	0 (%0)	

Fisher Freeman Halton Exact Test

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Table 6: Evaluation of Crack Changes from Time T0 to Time T1 in Groups by Sex

		Experimental (n=52)	Control (n=60)	
	T0-T1	**n (%)**	**n (%)**	**p**
Male	No new crack formation	49 (%94.2)	53 (%88.3)	
	Increase in crack length	3 (%5.8)	2 (%3.3)	0.090
	New crack formation	0 (%0)	5 (%8.3)	
		Experimental (n=56)	**Control (n=48)**	
Female	No new crack formation	53 (%94.6)	41 (%85.4)	
	Increase in crack length	2 (%3.6)	3 (%6.3)	0.244
	New crack formation	1 (%1.8)	4 (%8.3)	

Fisher Freeman Halton Exact Test

Table 7: Evaluation of Crack Changes from Time T0 to Time T1 by Sex Within Each Group

T0-T1		**Male (n=52)**	**Female (n=56)**	**p**
		n (%)	**n (%)**	
Experimental	No new crack formation	49 (%94.2)	53 (%94.6)	
	Increase in crack length	3 (%5.8)	2 (%3.6)	0.830
	New crack formation	0 (%0)	1 (%1.8)	
		Male (n=60)	**Female (n=48)**	
Control	No new crack formation	53 (%88.3)	41 (%85.4)	
	Increase in crack length	2 (%3.3)	3 (%6.3)	0.830
	New crack formation	5 (%8.3)	4 (%8.3)	

Fisher Freeman Halton Exact Test

DISCUSSION

This clinical study aimed to evaluate the significance of visible enamel cracks following orthodontic treatment and to investigate their relationship with the bracket debonding procedure. Enamel cracks remain a frequently discussed topic in the literature, yet their precise pathophysiology and clinical outcomes are not fully elucidated [2, 23, 24]. A central question for clinicians is whether cracks observed post-treatment result from the progression of pre-existing flaws or are directly induced by the debonding process itself [3, 5]. While numerous investigations have utilized invasive and high-cost methods like scanning electron microscopy (SEM) to examine microscopic damage [17, 18], their findings often have limited applicability in daily clinical practice. Our study focused specifically on macroscopic cracks detectable under routine clinical conditions using non-invasive methods like transillumination and high-resolution photographic analysis. This approach is critical for standardizing diagnostic criteria in general dentistry. The literature presents conflicting findings, with some reporting no significant difference in crack formation between different debonding techniques [25], while others indicate that ceramic

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brackets cause more enamel damage than metal ones [26, 27]. Our study aimed to contribute reliable methodological data to clarify these discrepancies.

Various methods exist for assessing enamel cracks. Earlier studies by Zachrisson et al. evaluated cracks after adhesive cleanup using fiber-optic transillumination, methylene blue dye, and visual shadowing [7, 15]. We omitted methylene blue due to its potential for intense staining in deep cracks, difficulty of removal, risk of temporary patient discoloration, and inconsistent performance in superficial cracks. Alternative methods like stereomicroscopy, SEM, polarized light microscopy, and AI-based digital imaging offer high resolution and objectivity [8, 9, 28]. However, their requirements for expensive equipment, time-consuming preparation, and non-clinical settings limit practical utility. Prioritizing patient comfort, clinical applicability, and reliability, we selected transillumination as our primary diagnostic aid.

Our methodological choices differ from several previous studies. First, we exclusively evaluated metal brackets. Some studies found that ceramic brackets caused more enamel damage than metal [27, 29, 30]; thus, our findings are not directly comparable to studies involving ceramic brackets. Second, unlike many in vitro studies on extracted teeth [17, 18, 22, 25], our in vivo design accounts for the natural hydration and organic matrix content of vital enamel, which influences its resistance and provides more clinically relevant data. Third, a key difference is the timing of assessment. While some studies evaluated enamel after both debonding and adhesive remnant removal [7, 17, 25], we photographed teeth immediately after debonding before any cleaning procedure. This eliminated the potential confounding effect of rotary instruments, isolating the impact of the debonding force itself.

Scanning electron microscope offers critical advantages for detecting micro-cracks and surface irregularities at high magnification [17, 18]. Our use of standardized dental photography with transillumination presents a practical, non-invasive, and clinically applicable alternative. The debonding technique—using a Griffin plier with a vertical gingivo-incisal force—was selected as a method reported to leave minimal enamel damage [18]. Regarding adhesive, we used a conventional composite resin (BracePaste®). While resin-modified glass ionomer cements (RMGIC) have been associated with less enamel damage upon ceramic bracket removal, their lower bond strength and higher clinical failure rate compared to composites make composites the preferred choice for reliable bracket fixation due to superior micromechanical retention and controlled polymerization [31, 32].

The statistical approach, utilizing parametric and non-parametric tests as appropriate, aligns with methodologies used in similar research [25]. The homogeneous distribution of age and gender between our groups minimized potential bias.

The mean participant age was 23.5 years, with the oldest being 35. Dumbryte et al. observed a more pronounced increase in crack size in individuals aged 35-54 [18]. Since our sample did not include this older demographic, the generalizability of our results is

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somewhat limited. Age-related thinning of enamel and reduction of its organic matrix may accelerate crack progression [33, 34], warranting investigation in older age groups.

Study showed no statistically significant differences in crack formation or progression between genders, either within or between groups. This contrasts with some studies. Baherimoghadam et al. reported a higher incidence of new cracks in women with fluorosed teeth [35], and Dumbryte et al. linked increased enamel fragility in postmenopausal women to hormonal changes [18]. Perez et al. found significant gender-based differences in crack distribution patterns [36]. Further research is needed to clarify the role of gender in enamel crack susceptibility.

Historically, studies reported a higher prevalence of enamel cracks in orthodontically treated patients compared to untreated controls. Zachrisson et al. found cracks in 73.8% of debonded teeth versus 59.7% in controls [7]. The high rate attributed to debonding in that era likely reflects less refined techniques. The similar distribution and localization of cracks across groups in their study suggested underlying non-iatrogenic factors. Yeom and Rhee reported an 18.5% increase in cracks across all groups, highlighting the protective effect of modern debonding methods [22]. Our finding of a significantly lower rate of new crack formation in the debonding group (0.9%) compared to the control group (8.3%) strongly suggests that contemporary metal bracket debonding is not a primary etiological factor for visible enamel cracks. The post-debonding adhesive cleanup phase is a recognized risk period. Pignatta et al. recommended a specific protocol for minimal damage [17], and other studies caution that ultrasonic or rotary instruments can induce micro-cracks if used with excessive pressure or duration [17, 37, 38]. By assessing the enamel surface immediately after debonding and before any cleanup, we evaluated the isolated effect of the debonding procedure, finding it to be minimal.

The higher susceptibility of upper central incisors to new crack formation (14.8% in CG), with no new cracks on lateral incisors, aligns with biomechanical expectations. Central incisors are subject to greater functional stress due to anterior guidance and have a thinner enamel layer compared to posterior teeth, facilitating crack formation. Their prominent role in aesthetics also makes cracks in this region a primary patient concern. The absence of new cracks on lateral incisors can be attributed to their smaller size, reduced functional load, and potentially more homogeneous enamel structure [7, 15]. Our exclusive focus on anterior teeth is justified by their aesthetic priority and the practical difficulty of obtaining equally clear photographic documentation of posterior surfaces. This finding underscores the need for clinicians to employ particularly careful debonding and cleaning protocols.

CONCLUSION

The findings of this clinical study suggest that modern debonding of metal brackets, when performed with appropriate technique, does not significantly contribute to the formation of new visible enamel cracks. The higher incidence observed in untreated individuals over time points to functional and physiological stressors as more dominant etiological factors.

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The anterior dentition, particularly maxillary central incisors, demonstrates the highest risk.

Recommendations

- Future research should investigate the effects of debonding ceramic brackets.
- Evaluate protocols for teeth with hypomineralized enamel.
- Include long-term follow-up to monitor crack progression.

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