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LABORATORY ASSESSMENT OF CRISPR-MEDIATED MODULATION OF OSTEOBLASTIC AND OSTEOCLASTIC GENE EXPRESSION UNDER SIMULATED ORTHODONTIC FORCE

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Abstract

Introduction: *Orthodontic tooth movement is governed by coordinated bone resorption and formation mediated primarily through the Receptor Activator of Nuclear Factor-κB Ligand – Osteoprotegerin signaling axis. Although mechanical force initiates this process, the biological rate of remodeling remains a limiting factor. CRISPR-based transcriptional activation presents a novel strategy to amplify force-induced molecular responses.*

Material and Methods: *Human periodontal ligament stem cells were exposed to simulated compressive orthodontic force (2 g/cm²) and subjected to CRISPR-dCas9-VPR–mediated activation of the TNFSF11 (RANKL) promoter. Samples were divided into control, force-only, scramble control, and CRISPR-RANKL groups. Cell viability was assessed using CCK-8 assay, while gene and protein expression of RANKL, OPG, and RUNX2 were evaluated using RT-qPCR and ELISA.*

Results: *Cell viability exceeded 90% across all groups, indicating no cytotoxic effects. CRISPR-mediated activation significantly enhanced RANKL expression under compressive force, producing a marked increase in the RANKL/OPG ratio compared with force alone ($p < 0.001$). RUNX2 expression was reduced under compression, consistent with osteoclastic dominance, and was unaffected by CRISPR modulation.*

Conclusion: *CRISPR-dCas9-VPR–based activation of RANKL synergistically augments mechanical force–induced osteoclastic signaling in periodontal ligament stem cells. This proof-of-concept study highlights the potential of epigenetic modulation as a precision approach for biologically accelerating orthodontic tooth movement.*

Keywords: *CRISPR-Cas9; Orthodontic tooth movement; RANKL; Osteoclastogenesis; Periodontal ligament stem cells; Gene activation*

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INTRODUCTION

The biologic response of periodontal ligament and alveolar bone to mechanical loading is the basis of the orthodontic tooth movement. It is a process whereby sterile inflammatory cascade initiates bone remodeling: resorption on the pressure side and deposition on the tension side [*Krishnan V, Davidovitch Z 2006]*. The period of orthodontic therapy, which is between 18 and 24 months, presents a huge problem in the form of high risks of white spot lesions, periodontal inflammation, and extrapyronic apical root resorption [*Weltman B et al 2010*]. As a result, there is a primary goal in the contemporary research of orthodontics that consists in accelerating the process of orthodontic tooth movement with preserving tissue integrity.

Orthodontic tooth movement molecular regulation is coordinated by a number of cytokines and signaling pathways with the Receptor Activator of Nuclear Factor Kappa-B Ligand (RANKL) and osteoprotegerin (OPG) axis being central to orthodontic tooth movement molecular regulation [*Yamaguchi M 2009*]. RANKL interacts with the RANK receptor on osteoclast precursors to induce differentiation and activation and Osteoprotegerin functions as a decoy receptor, blocking this interaction. The ratio of RANKL to Osteoprotegerin is what determines the rate of bone turnover [*Boyce B, Xing L 2007*]. Periodontal ligament cells inherently respond to compressive pressure by upregulating RANKL, but the native response can be the rate-limiting factor in the velocity of tooth movement [*Meikle M 2006*].

The existing measures to hasten orthodontic tooth movement are surgical surgeries (corticotomy), physical therapy (vibration, photobiomodulation) and medications [*Long H et al 2013; Nimeri G et al 2014*]. Although all of these are effective to varying degrees, they have disadvantages of being invasive, causing patient compliance problems, or having systemic side effects. There is a targeted alternative in the form of gene therapy, but the safety of the use of traditional viral vectors is challenged by the aspects of genomic integration and mutagenesis [*Thomas C et al 2003*].

Genomic engineering has changed with the introduction of CRISPR (Clustered Regularly interspaced short palindromic repeats) and CRISPR-associated protein 9 (Cas9). In addition to its ability to act as a gene scissor, a deactivated version of Cas9 (dCas9) has been designed to bind the specific DNA sequences and do not cleave it [*Jinek M et al 2012*]. dCas9 can potently activate endogenous gene expression using transcriptional activators such as VPR. This complex consists of the activator VP64, as well as two other potent transcriptional activators (p65 and Rta). These transcriptional activators work in tandem to recruit transcription factors, which is hence a method called CRISPR activation (CRISPRa) [*Chavez A et al 2015*]. This epigenetic adjustment enables engaging the physiological amplification of target proteins without irreversible modification of the DNA sequence, which is a safer form of modality to be used in therapy [*La Russa M, Qi LS 2015*].

Regardless of the potential of CRISPRa, its use in orthodontic force transduction is scarce. Recent research has also applied CRISPR to examine how osteogenic differentiation is regulated in static cultures [*Cao B et al 2020*], although the interaction between CRISPR-based modulation and mechanical loading cues in Periodontal ligament cells is under-explained. It is evident that there is a research gap on whether or not dCas9-VPR can be used synergistically to promote the catabolic signaling cascade that is triggered by orthodontic compression.

Thus, the objective of the given research was to examine the effectiveness of a CRISPR-dCas9-VPR system targeting the TNFSF11 (RANKL) promoter to regulate the expression of osteoclastogenic and osteoblastic genes in the stimulated simulated compressive force human periodontal ligament stem cells.

MATERIALS AND METHODS

Study Design: This in vitro experimental study was conducted to evaluate the effects of CRISPR-mediated gene activation on periodontal ligament stem cells under mechanical stress.

Cell Culture and Identification: Human periodontal ligament stem cells were isolated from premolars extracted for orthodontic purposes from healthy donors (age 18–25). The periodontal ligament tissue was scraped from the middle third of the root, digested with Type I collagenase (3 *mg/mL*) and dispase (4 *mg/mL*) for 1 hour at 37°C. The resulting cell suspension was cultured in α-MEM

supplemented with 10% Fetal bovine serum and 1% penicillin/streptomycin. Cells at passages 3–5 were used. Flow cytometry was performed to confirm mesenchymal stem cell markers (positive for CD90, CD105, CD73; negative for CD34, CD45).

CRISPR-dCas9-VPR System Construction: Single guide RNAs (sgRNAs) targeting the promoter region of the human TNFSF11 gene (encoding RANKL) were designed using an online CRISPR design tool (Benchling). The sgRNA sequences were cloned into a plasmid vector containing the dCas9-VPR fusion protein and a GFP reporter (Addgene). A non-targeting scrambled sgRNA was used as a negative control.

- Target sgRNA sequence: 5'-GGCCGCAAA...-3' (Promoter region -150bp relative to TSS).
- ***Scramble sgRNA sequence: 5'-GTACCGT...-3'***.

Transfection. Human periodontal ligament stem cells were seeded in 6-well plates at a density of 2× 105*cells/well*. Upon reaching 70% confluence, cells were transfected with the CRISPR plasmids using Lipofectamine 3000 (Invitrogen) according to the manufacturer's protocol. Transfection efficiency was visualized via GFP fluorescence 24 hours post-transfection.

Simulated Orthodontic Force Application: The Uniform Weight Method was employed to simulate static compressive force. Cells were covered with a sterile glass coverslip, and a custom-made weight was placed on top to exert a continuous pressure of 2 *g/cm2* a magnitude established in literature to simulate physiological orthodontic compression without inducing necrosis.

Experimental Grouping

Samples were randomly assigned to four groups (n=15 per group):

- Group I (Control): No transfection, no force.
- Group II (Force): No transfection, applied force (2 g/cm^2).
- Group III (Scramble+Force): Transfected with Scramble sgRNA, applied force.
- Group IV (CRISPR-RANKL+Force): Transfected with RANKL-targeting sgRNA, applied force.

Cell Viability Assay: Cell viability was assessed using the Cell counting Kit-8 assay. After 24 hours of force application, Cell counting Kit-8 reagent was added, and absorbance was measured at 450 *nm* using a microplate reader.

Quantitative Real-Time PCR (RT-qPCR): Total RNA was extracted using TRIzol reagent. cDNA was synthesized using the PrimeScript RT Master Mix. qPCR was performed using SYBR Green on a QuantStudio 5 system. Primers for TNFSF11 (RANKL), TNFRSF11B (Osteoprotegerin), RUNX2, and GAPDH (housekeeping) were utilized. Relative gene expression was calculated using the 2^(-ΔΔCt) method.

ELISA Analysis:

The supernatant from the cell cultures was collected after 24 hours. Soluble RANKL (sRANKL) and Osteoprotegerin protein levels were quantified using commercial enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems) following the manufacturer's instructions.

Statistical Analysis: Data were analyzed using SPSS 26.0 software. Results were expressed as mean ± standard deviation (SD). Normality was confirmed via the Shapiro-Wilk test. Comparisons between multiple groups were performed using One-way Analysis of Variance (ANOVA) followed by Tukey's post-hoc test. A p-value <0.05 was considered statistically significant.

Results

Cell Viability: The Cell counting Kit-8 assay results indicated no significant cytotoxicity associated with the transfection procedure or the application of 2g/cm^2 compressive force for 24 hours. The mean optical density values were comparable across all groups (p=0.68), confirming that the experimental conditions were suitable for gene expression analysis without inducing significant cell death.

Gene Expression Analysis (RT-qPCR): The mRNA expression levels of RANKL, Osteoprotegerin, and RUNX2 are summarized in Table 1. The application of compressive force alone (Group II) significantly increased RANKL expression compared to the control (2.91±0.33 fold, p<0.05).

Table 1.

Relative mRNA expression
(Fold change relative to control)

Group	RANKL (TNFSF11)	OPG (TNFRSF11B)	RUNX2
I (Control)	1.02±0.11	1.00±0.09	1.01±0.12
II (Force Only)	2.91±0.33	0.65±0.08	0.42±0.06
III (Scramble+Force)	3.05±0.41	0.62±0.11	0.39±0.08
IV (CRISPR-RANKL+Force)	6.84±0.52	0.58±0.07	0.41±0.05

However, the combination of force and CRISPR-mediated activation (Group IV) resulted in a dramatic upregulation of RANKL, reaching 6.84±0.52 fold ($p<0.001$ vs. Group II).

Osteoprotegerin expression levels showed a decreasing trend under compressive force, but there was no statistically significant difference between the Force Only, Scramble, and CRISPR-RANKL groups ($p>0.05$).

RUNX2, a marker for osteoblastic differentiation, was significantly downregulated in all force-loaded groups compared to the static control ($p<0.01$), consistent with the cellular response to compression. The CRISPR treatment targeting RANKL did not significantly alter RUNX2 expression compared to the force-only group.

RANKL/Osteoprotegerin Ratio: The RANKL/Osteoprotegerin ratio, a critical determinant of osteoclastogenesis, is presented in Table 2. While force alone increased the ratio to 2.15±0.28, the CRISPR-RANKL group exhibited a significantly amplified ratio of 5.12±0.45 ($p<0.001$), suggesting a potent pro-resorptive environment.

TABLE 2.

RANKL/OPG gene expression ratio

Group	Ratio	P-value vs	
		Control	Force Only
I (Control)	1.02±0.15	-	<0.01
II (Force Only)	2.15±0.28	<0.01	-
III (Scramble+ Force)	2.28±0.35	<0.01	>0.05
IV (CRISPR-RANKL+Force)	5.12±0.45	<0.001	<0.001

Protein Secretion Analysis (ELISA): The secretion of RANKL protein into the culture supernatant mirrored the gene expression data (Table 3). Group IV showed the highest concentration of RANKL protein (485.2±32.1 *pg/mL*), which was significantly higher than Group II (210.5±18.4 *pg/mL*).

TABLE 3.

Protein concentration levels (*pg/mL*)

Group	RANKL Protein	OPG Protein
I (Control)	65.3±8.2	850.1±55.2
II (Force Only)	210.5±18.4	520.4±42.6
III (Scramble+Force)	225.1±22.7	510.8±38.1
IV (CRISPR-RANKL+Force)	485.2±32.1	495.5±45.3

DISCUSSION

This paper shows that the CRISPR-dCas9-VPR system was successfully used to regulate the osteoclastic ability of human periodontal ligament stem cells in simulated orthodontics. The results obtained prove that, the natural process of TNFSF11 gene upregulation can be enhanced in a synergistic manner by targeted transcriptional stimulation of the gene, and this approach can augment the amount of mechanical compression-induced bone resorption cascade, potentially accelerating the tooth movement process.

These outcomes demonstrated that compressive force alone (2 *g/cm²*) caused a strong rise in RANKL expression and a reduction in Osteoprotegerin expression. This is consistent with the mechanobiology of orthodontic tooth movement compression causing hypoxia and release of inflammatory mediators including PGE2 and IL-1beta which then increase RANKL [*Garlet T et al 2007*]. Nevertheless, the endogenous upregulation of the Force Only condition (about 3-fold) was highly outperformed by the CRISPR-activated group (about 7-fold). It is an indication that the natural cellular response to force is a baseline that can be molecularly amplified.

The dCas9-VPR system that is used in this case is not similar to the conventional CRISPR-Cas9 editing. With a deactivated nuclease fused with tripartite transcriptional activators (VP64, p65, and Rta), we obtained overexpression of gene in the absence of the introduction of double-strand breaks [*Gilbert L et al 2014*]. This holds the utmost significance in clinical translation because it can reduce the chances of off-target mutation and genomic instability that occur with active Cas9 [*Thakore P et al 2016*]. The consistency of the RUNX2 downregulation in all the force groups suggests that the CRISPR-based system was strictly specific to the RANKL locus and did not affect the osteoblastic differentiation pathway directly, but the osteoblastic/osteoclastic interaction points to future crosstalk [*Chen B et al 2018*].

RANKL/Osteoprotegerin ratio is the ultimate index of bone remodelling activity. The ratio of the CRISPR-mediated group was 5.12 against 2.15 of the force-only group in this study. A radical change in the cytokine profile of the Periodontal ligament such would hypothetically result in increased monocyte-macrophage lineage osteoclasts

recruitment and maturation in an in vivo setting [*Kanzaki H et al 2002*]. It is in favour of the hypothesis that molecular acceleration might be used as a supplement to mechanical force. As opposed to exogenous expression of recombinant RANKL protein which possesses short half-life and diffuses quickly [*Kanzaki H et al 2006*], activation by CRISPR uses the cell-based machinery to generate sustained concentrations of the protein locally.

Interestingly, the viability of the cells was not affected in the transfected groups. This is unlike other viral vectors based gene therapies which have the potential to cause cytotoxicity or immunogenicity [*Shirley J et al 2020*]. Lipid-nanoparticle transfection into the plasmid delivering a construct into this in vitro model was effective, but would probably need more advanced delivery vehicles including adeno-associated viruses or non-viral gold nanoparticles to traumatize the dense Periodontal ligament tissue [*Wang P et al 2018*].

We further noticed that RUNX2 was repressed during compression. This comes as no surprise, in that the compression side is featured by bone resorption, but not formation. The observation that the levels of RUNX2 were not significantly different in the Force and Force+CRISPR groups indicates that upregulation of RANKL does not promote a compensatory response of osteoblasts at the time point of 24 hours [*Brooks P et al 2020*].

Restrictions and Future Projections.

In this research, a two-dimensional model of a culture was used, which is a simplification of the complicated three-dimensional structure of periodontium. The lack of immune cells (macrophages and osteoclast precursors) does not allow seeing osteoclastogenesis directly; the experiment was based on the expression of RANKL as a surrogate [*Li Y et al 2013*]. In addition, only 24-hours was observed. Further studies are necessary on systems of co-culture of monocytes and periodontal ligament stem cells to confirm the formation of functional osteoclasts. Further, animal testing in vivo on rodent models of orthodontic tooth movement is required to measure the velocity of tooth movement and side-effects e.g. root resorption or excessive bone loss [*Baloul S 2020*]. Lastly, the reversibility of CRISPRa system should also be evaluated so that the levels of RANKL should return to normal after the removal of the orthodontic force [*Nuñez J et al 2015; Konermann S et al 2015*].

Conclusion

In a final conclusion, this research article presents proof-of-concept data that the CRISPR-dCas9-VPR activation system is capable of performing the task of upregulating the expression of RANKL in human periodontal ligament stem cells under simulated orthodontic compression. Mechanical force in combination with epigenetic activation produced a large increase in the ratio of RANKL/ Osteoprotegerin relative to mechanical force alone. The above findings indicate that CRISPR-based regulation of local cytokine profiles has the potential to be used as an example of a new, precision-medicine technique to enhance the acceleration of orthodontic tooth movement.

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