UNIVERSITY OF PITTSBURGH SCHOOL OF DENTAL MEDICINE

20 Research 25 Symposium

Celebrating 25 years

October 9, 2025
Alumni Hall
Connolly Ballroom

University of Pittsburgh
Health Sciences
School of Dental Medicine

WELCOME FROM THE DEAN AND ASSOCIATE DEAN FOR RESEARCH

It is our great privilege to welcome you to the Pitt Dental Medicine 2025 Research Symposium and to share our enthusiasm for the remarkable research being conducted across our school.

We are so very proud of this well-established highlight of the University of Pittsburgh School of Dental Medicine—this year we are celebrating its 25th anniversary.

It is inspiring to see the dedication and innovation that our faculty, staff, and students bring to the work done here. Our collective efforts not only advance knowledge but also address some of the most pressing challenges facing dental medicine, oral health, and beyond.

This symposium is a cornerstone of our academic year—it's where we celebrate the hard work and intellectual curiosity that drive our school forward. It's a privilege to see so many brilliant minds gathered here today, united by a shared passion for advancing dental science and medicine.

Marnie Oakley, DMD
Dean

Charles Sfeir, DDS, PhD
Associate Dean, Research

THIS IS A DAY FOR DISCOVERY, DIALOGUE, AND COLLABORATION. I ENCOURAGE EVERYONE TO ASK QUESTIONS, CHALLENGE IDEAS, AND FIND OPPORTUNITIES TO CONNECT WITH COLLEAGUES. YOUR NEXT BIG BREAKTHROUGH MIGHT BE SPARKED IN A CONVERSATION YOU HAVE HERE TODAY.

Research is the lifeblood of our profession. It's what allows us to advance the existing limits of patient care, develop new technologies, and uncover novel treatments for some of the most challenging oral health issues. The presentations you'll see today are more than just posters or data sets; they are the seeds of many ongoing and future innovations that will directly improve the lives of our patients.

We want to extend a special thanks to all of our presenters, especially our students and residents. The work you've accomplished is a testament to your dedication and skill. You are not only learning the current standards of care—you are actively creating the new ones that build our future.

This is a day for discovery, dialogue, and collaboration. We encourage everyone to ask questions, challenge ideas, and find opportunities to connect with colleagues. Your next big breakthrough might be sparked in a conversation you have here today.

Thank you for your commitment to excellence and for making our school a vibrant hub of research and innovation. We look forward to the incredible work that will be shared today.


2025
Research
Symposium

RESEARCH SYMPOSIUM 2025 AGENDA

9:00 – 9:15 AM

Opening Remarks and Introduction to the Program

Marnie Oakley, DMD
Dean
University of Pittsburgh
School of Dental Medicine

Charles Sfeir, DDS, PhD
Associate Dean of Research
University of Pittsburgh
School of Dental Medicine

9:15 - 10:15 AM

Keynote Speaker

Titanium particle-mediated Peri-implantitis and the Harm Associated with Empirical Dental Treatments

Georgios Kotsakis, DDS, MS
Professor & Assistant Dean for Clinical Research
Director of Research, Rutgers School of Dental Medicine

10:15 -10:30 AM

Break

10:30 – 10:45 AM

Pitt Dental Medicine Research Highlights Introduction

Charles Sfeir DDS, PhD, and Mary L. Marazita, PhD

10:45 – 11:00 AM

Periodontal Disease Elicits Alzheimer's Disease Pathology in Mouse Models

Sangmin Park
Student, DMD Class of 2029

11:00 – 11:15 AM

Not All Bleeding on Probing Indicates Disease: Single-Cell Transcriptomics Reveal Distinct Immunologic Signatures Associated with Bleeding Severity Around Dental Implants

Haipei Luan, DDS
Resident

11:15 – 11:30 AM

Nitric Oxide Synergizes with TGF-β1 in Healing of Segmental Bone Defects

Gabrielle Lorenz
PhD Candidate

11:30 – 11:45 AM

Pleiotropy-informed GWAS Identifies Orofacial Cleft Risk Loci

Noah Herrick, PhD
Postdoctoral Associate

11:45 -12:15 PM

Student Research Awards

Fatima N. Syed, MSE, PhD
Director of Student Research

12:15 – 1:00 PM

Luncheon

1:00 – 3:00 PM

Poster Session

MEET OUR KEYNOTE SPEAKER

TITANIUM PARTICLE-MEDIATED PERI-IMPLANTITIS AND THE HARM ASSOCIATED WITH EMPIRICAL DENTAL TREATMENTS

Georgios Kotsakis DDS MS

Professor & Assistant Dean for Clinical Research
Director of Research, Rutgers School of Dental Medicine

Dr. George Kotsakis is a board-certified periodontist and a fellow of the ITI. He received his DDS from the University of Athens. He practiced in Athens, Greece prior to coming to the US. He then completed his residency in periodontics and MS in science at the University of Minnesota when he first got involved in peri-implantitis research. Following his training, he became an assistant professor in the Department of Periodontics at the University of Washington, Seattle, WA and then moved to UTHealth San Antonio as an associate professor and held the Roland Meffert Endowed Professorship in Implant Dentistry. He currently is a professor of oral biology and Assistant Dean for Clinical Research at Rutgers School of Dental Medicine. He directs the NIH-funded Translational Periodontal Research Lab, conducting research on the biological mechanisms underlying peri-implant bone loss and developing novel treatments for dental and biomedical implants.

Dr. Kotsakis has published more than 100 peer-reviewed scientific articles and textbook chapters with more than 25,000 citations of his work (h-index: 35). He is one of the few dental researchers to have been published in prestigious medical publications, such as the *Lancet*, the *BMJ* and *PNAS*.

He has been the recipient of multiple career and research awards from the American Academy of Periodontology, the Academy of Periodontology Foundation, the IADR, and other organizations. Additionally, he serves as an associate editor for the *Journal of Periodontology*, and for *Clinical Implant Dentistry and Related Research*. He lectures frequently and gives hands-on courses both nationally and internationally on peri-implantitis therapy.

PRESENTATION ABSTRACTS

The following include today's oral presentation abstracts followed by poster abstracts.

Periodontal Disease Elicits Alzheimer's Disease Pathology in Mouse Models.

Park S[1], Campos T[1], Diacou A[1], Clark D[1,2]

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Alzheimer's Disease (AD) is characterized by cognitive decline, pathologic Aβ plaque accumulation, and neuroinflammation in the brain. AD presents a major healthcare and economic burden, affecting over 50 million people. However, our understanding of AD pathology is limited. Recent work has focused on peripheral inflammation in AD pathogenicity. Periodontal disease (PD) is a peripheral inflammatory condition that demonstrates an epidemiological association with AD. This study investigated the extent to which PD contributes to AD pathology.

PD was induced via a ligature induction technique in mouse models of AD (APP/PS1). Three experimental groups were evaluated; control group without PD (APP-C), PD-induced group (APP-PD), and a PD-re-induction group where PD was induced, the mouse was allowed to recover, then induced PD again (APP-RD) (n=7/group). AD pathology was assessed in the brain via immunohistochemistry analysis of Aβ plaque burden, microglial response and morphology, and via bulk RNA sequencing.

Aβ plaque accumulation and microglia activation increased in PD groups compared to APP-C ($p<0.05$). APP-RD exhibited significantly increased microgliosis and plaque-associated microglia count, while APP-PD exhibited significantly elevated non-plaque-associated microglia count. The non-plaque associated microglia in APP-PD demonstrated distinct morphological differences suggesting an altered microglia response ($p<0.05$). Transcriptomics analysis of brain tissue identified 2517 genes significantly differentially regulated in PD versus control. Gene ontology analysis identified dysregulation in mitochondrial regulation with mitochondrial fusion (Opa1), fission (Fis1), and regulation (Pink1, Bax, Clu) genes significantly altered. Redox genes that neutralize oxidative stress were downregulated and genes maintaining blood-brain barrier (BBB) integrity were also dysregulated.

Our results demonstrate pathologic brain responses in mice as a function of PD, with increased Aβ plaque accumulation, microglia activation, and transcriptional dysregulation affecting mitochondria, oxidative stress, and BBB integrity. These findings underscore the need for further research on peripheral inflammation in AD pathogenesis.

Not All Bleeding on Probing Indicates Disease: Single-Cell Transcriptomics Reveal Distinct Immunologic Signatures Associated with Bleeding Severity Around Dental Implants

Haipei Luan, Debra Dias, Martinna Bertolini, Andrea Ravida

Introduction

Peri-implant mucositis is currently defined by any bleeding on probing (BOP), but this may over diagnose inflammation due to trauma from probing forces >0.25 N. Herrera et. al redefined it as profuse bleeding at one site or any bleeding at ≥2 sites, which still risks misclassifying mechanical bleeding. This study aimed to identify immunologic signatures linked to BOP severity around implants.

Methods

Twenty systemically healthy patients with implants restored before January 2021 and no radiographic bone loss were enrolled. They were stratified into four groups (n=5/group) based on modified bleeding index: 0=no bleeding, 1=isolated bleeding spot, 2=continuous line or drop of blood, 3=profuse bleeding. Clinical assessments were performed by two examiners. Peri-implant soft tissue biopsies were obtained from sites corresponding to recorded

BOP scores and analyzed via single-cell RNA sequencing using 10x Genomics Chromium platform followed by Illumina NovaSeq sequencing.

Results

Single-cell transcriptomic profiling revealed that only BOP scores 2 and 3 were associated with distinct enrichment of immune cell populations. Plasma cells, granulocytes, and monocytes were increased, alongside reduction in epithelial and fibroblast subsets, suggesting mucosal damage, particularly pronounced in BOP 3. In contrast, BOP scores 0 and 1 showed nearly identical immune cell compositions, consistent with a healthy immunological profile.

Additionally, genes associated with epithelial barrier function (e.g., LOR, CDSN) were downregulated in BOP 2 and 3, while inflammatory and immune mediators (e.g., CXCL8, IL17RA) were upregulated, especially in BOP 3, indicating activation of mucosal immune pathways.

Conclusion

These findings suggest that BOP scores 0 and 1 are immunologically similar and reflect a biologically healthy state, while BOP scores 2 and 3 indicate true inflammatory conditions. These results challenge current diagnostic paradigms and highlight the need for redefinition of peri-implant mucositis to improve diagnostic accuracy and prevent overdiagnosis.

Nitric Oxide Synergizes with TGF-β1 in Healing of Segmental Bone Defects

Gabrielle Lorenz[1], Daniel Zamith Miranda[2], Ingrid McNamara[1], Jennifer Cox[3], Jorge Pena[3], Braden Miller[4], Manuela Gaviria[5], Alexander Bowers[4,5], Clay Tucker[6], Alejandro Almarza[1], Andrew Draganski[6], Casey Sabbag[5], Joshua Nosanchuk[2], Juan Taboas[1]

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Objective

Delayed treatment of open bone fractures in low resource settings (LRSs) increases infection by 42% and chronic non-union rates by 6-12x. We have developed an injectable hydrogel containing TGF-β1 and nitric oxide ($NO^{\cdot}$) to prevent antimicrobial-resistant (AMR) infections and accelerate healing. $NO^{\cdot}$ is an endogenous antimicrobial that promotes bone regeneration; however, current $NO^{\cdot}$ donors are cytotoxic at required doses. Our team developed silica microparticles (MSNO) that prolong $NO^{\cdot}$ delivery. MSNO reduced MRSA (an AMR) biofilms in vitro (99%) and in vivo (femoral defects in rats). Herein, we study the effect of hydrogel type (RTC = rat tail collagen vs. PEG-GEL = gelatin azide/dibenzocyclooctyne-functionalized polyethylene glycol) and drug effect (+/- $NO^{\cdot}$ and +/- TGFB) on healing.

Experimental Methods

MSNO (10 mg/mL = 21.2 mM $NO^{\cdot}$) and TGF-β1 (100 mg/ml) were incorporated into 0.3% (w/v) RTC or 2% PEG-GEL. In vitro: Human bone marrow stromal cells (BMSC) were cultured with MSNO hydrogels and analyzed by RT-qPCR for stemness/differentiation markers. In vivo: 5.0 mm femoral segmental defects in Wistar rats (n=6) were treated 24 hours post-surgery (delayed intervention). Leukocyte infiltrate at 5-days was profiled by flow cytometry, and healing at 16 weeks assessed using X-rays, micro-CT, and histomorphometry.

Results

MSNO reduced expression of stemness (Sox2, Oct4) and chondrogenic (aggrecan) markers in BMSCs, while increasing osteogenic (osteocalcin, alkaline phosphatase). MSNO+TGF-β1 accelerated defect bridging (>50% by 9 weeks, >80% by 12 weeks), while controls with either alone showed no significant bridging. RTC increased CD4+ and cytotoxic (CD8+) T cells while decreasing granulocytes. Individually, MSNO and TGFβ1 suppressed $CD4^{+}$ and $CD8^{+}$ cells. Notably, TGF-β1 abrogated M1 macrophages, potentiating a reparative response.

Conclusion

$NO^{\cdot}$ and TGF-β1 synergistically promoted osteogenesis, potentially via induction of osteogenic differentiation and modulation of the immune response. PEG-GEL provided superior cytocompatibility, supporting its use as a therapeutic for contaminated bone wounds in LRSs.

Pleiotropy-informed GWAS Identifies Orofacial Cleft Risk Loci

Noah Herrick[1], Seppe Goovaerts[2,3], Alexandra Manchel[4], Myoung Keun Lee[1], Xinyi Zhang[5], Jenna C Carlson[5], Elizabeth J Leslie-Clarkson[6], Mary L Marazita[1,5], Justin Cotney[4], Peter Claes[2,3,8,9], John R Shaffer[1,7], Seth M Weinberg[1,7]

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9 Murdoch Children's Research Institute, Melbourne, Victoria, Australia

Several lines of evidence suggest that normal-range facial features and nonsyndromic orofacial clefts (OFCs) exhibit a shared genetic basis. Approaches designed to leverage this relationship hold the possibility of revealing new OFC risk loci by boosting discovery power. To test this idea, we applied a pleiotropy-informed GWAS method that uses conditional false discovery rate (cFDR-GWAS) analyses with summary statistics from large, independent European GWASs of normal facial shape (n=4,680; n=3,566), a subclinical facial phenotype (n=8,246), and nonsyndromic cleft lip with or without cleft palate (nsCL/P, n=3,969), revealing an interconnected genetic architecture between these traits greater than previously known. The cFDR approach identified 46 independent genomic loci associated with nsCL/P— over 9x the number identified in the original nsCL/P GWAS (n=5). Of these 46 loci, 27 were detected at 5% cFDR and 19 were detected at a suggestive FDR threshold (10% cFDR). Many of these loci were previously implicated in orofacial clefting (n=20) but were not identified in the original nsCL/P GWAS. The remaining discovered loci (n=21) represent novel regions. We looked at gene expression patterns for our top candidates in major cell types and spatial transcriptomics data using a single-nucleus RNA sequencing (snRNA-seq) atlas, which highlighted their role in craniofacial development. Ontology enrichment analysis resulted in significant terms for biological processes including morphogenesis and development of the embryonic skeletal system and mouse phenotypes including abnormal craniofacial development, cleft palate, and abnormal cartilage morphology. In conclusion, the application of an empirical Bayesian strategy to draw on association signals from genetically related traits can boost the power to better prioritize OFC risk loci missed by traditional gene mapping approaches. These results hold promise that the cFDR-GWAS approach may be able to enhance our understanding of the genetic architecture of other structural birth defects.

Demographic and Clinical Characteristics of Median Rhomboid Glossitis: A 20-Year Case Series

Dr. Mobolaji Moore *(UPMC)*, Dr. Yareen J. Alawneh *(University of Pittsburgh)*, Dr. Kurt Summersgill *(University of Pittsburgh School of Dental Medicine and UPMC)*

Introduction

Median rhomboid glossitis (MRG) is a rare erythematous lesion located on the dorsum of the tongue, often associated with Candida infection. The lesion is typically asymptomatic; however, it may present with pain or burning in some cases. Risk factors for MRG include smoking, immunosuppression, and poor oral hygiene. Despite its established link to Candida, MRG's etiology remains multifactorial and underreported. This study aims to evaluate the prevalence of Candida in biopsy-confirmed MRG cases and analyze associated demographic, systemic, and lifestyle factors to improve diagnostic and management strategies.

Materials and Methods

We conducted a natural language search of UPMC and University of Pittsburgh databases for MRG biopsied from 2006 to 2024. Patient demographics, clinical features, and histopathological findings were collected and analyzed. Biopsy slides were reviewed to confirm Candida presence and assess histologic features, with diagnostic disagreements resolved through consensus review.

Results

Fifty-seven cases were identified. Most patients were female (53%) and White (63%), with a mean age of 56 years. MRG was asymptomatic in 88% of cases, with few reports of pain. Candida pseudohyphae

were identified in 70% of cases, while hyperplastic tissue was observed in 63%. Only 19% of cases exhibited nodular variants of MRG. Smoking was reported in 10% of patients. Medical histories were frequently incomplete, limiting analysis of systemic associations.

Conclusion

In our study, MRG biopsies were predominantly of asymptomatic middle-aged females. While Candida is frequently implicated, its absence in 30% of cases highlights the multifactorial nature of MRG's etiology or the need for a more sensitive method of identification. The findings emphasize the need for heightened clinician awareness of MRG's diverse clinical presentations to avoid unnecessary biopsies. Broader studies are necessary to better characterize this condition and its regional variations.

Dental Anomalies in Alagille Syndrome

Mariam Amr,[1] Weehan Choi,[1] Jiehua Zhang,[1,2,3] Simona Hankeva,[4] Noemi Van Hul,[4] Alice Goodwin,[4,6] Emma Andersson,[7] and Andrew Jheon[4,6*]

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The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Abstract

Alagille syndrome (ALGS1; Online Mendelian Inheritance in Man/OMIM no. 118450, and ALGS2, OMIM no. 610205) is a multisystem genetic disorder characterized by chronic cholestasis, cardiovascular anomalies, ocular abnormalities, vertebral defects, and distinctive craniofacial features. It is the most common rare cholestatic liver disease seen by pediatric hepatologists. Despite its varied and multisystem presentation, in a recent survey by the Alagille Syndrome Alliance (ALGSA; alagille.org), 50% of children with ALGS reported that their teeth are a major cause of concern (e.g., misshapen, crooked, discolored teeth). Yet there are only a handful of case reports involving teeth and ALGS, and the dental manifestations of ALGS remain poorly understood. Notch signaling, a highly conserved cell–cell communication pathway, is essential for development and homeostasis. Mutations in the Notch ligand JAGGED1 (JAG1) underlie ALGS1, and the Jag1Ndr/Ndr missense (H268Q) mouse model closely recapitulates the human phenotype. To investigate dental involvement in ALGS1, we examined teeth from adult Jag1Ndr/Ndr (Ndr) mice alongside human deciduous incisors using histology, scanning electron microscopy (SEM), nanoindentation hardness testing, and digital color analysis. Both Ndr mouse and human ALGS teeth exhibited multiple abnormalities, including greenish discoloration, altered molar morphology, partial detachment between ameloblasts and the enamel matrix, and disrupted ameloblast–stratum intermedium interactions. SEM revealed enamel rod mineralization defects, with potential differences at the dentin–enamel junction. Hardness testing confirmed reduced enamel and dentin strength in ALGS teeth compared with controls. These findings contribute to the characterization of dental defects in ALGS1 and underscore the critical role of Notch signaling in dental development.

Loss of Trem2 leads to increased mouse incisor dentin

W. Choi, J. Yin, A. Diacou, D. Clark, A.H.Jheon

Trem2 is an immunoreceptor expressed on osteoclasts and microglia, and its loss of function causes Nasu-Hakola disease and increased risk of Alzheimer's disease. Although the role of Trem2 on inflammation and neurodegenerative diseases has been studied previously, relatively little is known regarding changes in teeth. To better understand the dental manifestation of Trem2 inactivation, we analyzed the hemi-mandibles of 4-monthold Trem2-/- mice using micro-computed tomography (microCT). Preliminary micro-CT images of Trem2-/- mice showed significantly higher dentin volume with lower pulp volume in the incisor, while molars showed no significant differences. We performed histology to analyze the localization of osteoclast and its associated markers. Fewer osteoclasts were observed in Trem2 null mice along with little or no

difference in dentin sialophosphoprotein (Dspp) expression, suggesting that the significantly higher dentin volume is due to less resorption rather than increased formation. Surprisingly, we observed significantly fewer osteoclasts around the periodontal ligaments around the molar roots in Trem2-/- mice despite no visible morphological differences. This initial study suggests that Trem2 might play a significant role in dentin osteoclast survival and differentiation associated with dentin resorption.

X-chromosome-wide Meta-analysis Identifies Genetic Associations With Orofacial Clefts

Zeynep Erdogan-Yildirim[1], Seth M Weinberg[1], John R Shaffer[1,2], Claire Simpson[3,4], Joan Bailey-Wilson[3], Ingo Ruczinski[5], Mary L Marazita[1,2,6]

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Orofacial clefts (OFCs) are among the most common congenital defects and demonstrate sex-biased prevalence: cleft lip with or without cleft palate (CL/P) is twice as often in males as in females, whereas cleft palate (CP) is more common in females. This pattern along with emerging evidence implicating X-linked genes in OFC etiology suggests that the X chromosome may be a plausible contributor to non-syndromic OFCs. Although several X-linked genes like TBX22, MID1, and OFD1 are known to be causal for syndromic forms of OFCs, X-chromosomal markers implicated in non-syndromic OFC risk remains underexplored.

To address this gap, we conducted an X-chromosome-wide meta-analyses involving 5,609 individuals of multiethnic ancestry from two independent samples, genotyped with the Illumina Human610-Quadv.1_B BeadChip array and the Illumina GDA-8v1-0 array, respectively. Additional genotypes were imputed using the TOPMed reference panel r2 via TOPMed Imputation Server and minimac4. Variants with poor imputation quality (rsq < 0.8) were excluded. Separate logistic regression analyses for CL/P and CP were performed using the GENESIS package, adjusting for sex, the principal components of ancestry, and relatedness.

No variant reached the conventional genome-wide significance threshold of $< 5 \times 10^{-8}$. However, we observed suggestive associations that replicate previously reported X-linked loci: For CL/P, the strongest signal was found in the intergenic region between EFNB1 and PJA1 (chrX:69094776_G/T, p= 3.08×10^{-6}), 4kb away from known locus. For CP, the strongest signal mapped to the intergenic region near SPANXN4 (chrX:142884712_C/T, p=1.30×10^{-5}), and the second strongest signal was detected with the intronic variant in DMD (chrX:32953387_T/A, p=2.47×10^{-5}). Both loci were reported in previous OFC studies.

These findings underscore the potential involvement of X-chromosomal regions—especially EFNB1/PJA1, SPANXN4, and DMD—in non-syndromic OFC risk. To further refine our analysis and improve the discovery power, we are currently re-imputing the genotypes using the latest TOPMed reference panel r3, which has better variant coverage and hence, better imputation accuracy.

Not All Bleeding on Probing Indicates Disease: Validation of Protein Signatures Associated with Peri-Implant Mucosal Integrity and Inflammation

Alexandra McDonald, Haipei Luan, Debora Dias, Andrea Ravida, Martinna Bertolini

Objectives

Our group has done single-cell transcriptomic profiling of peri-implant mucosal tissues and revealed that only moderate to severe bleeding on probing (BOP 2–3) is associated with enrichment of immune cell populations and loss of epithelial barrier gene expression, whereas BOP 0–1 reflects a healthier immunologic state. The present study aimed to validate these findings at the protein level by visualizing epithelial integrity, immune infiltration, and inflammatory markers across BOP severities.

Methods

Circumferential peri-implant mucosal biopsies were obtained from 10 systemically healthy patients with implants restored prior to 2021. Samples were embedded in OCT, frozen at –80 °C, and cryosectioned at 10 µm. Sections were permeabilized,

blocked, stained with conjugated antibodies against selected proteins relevant to epithelial barrier function and immune activation, counterstained with Hoechst, and imaged using a Nikon NI-E widefield microscope at 20× magnification. Fields were stitched to generate complete views of the peri-implant mucosa, allowing visualization of the epithelial lining, connective tissue compartments, and gingival papilla tip architecture.

Results

Histological imaging demonstrated well-organized epithelial layers and low immune cell infiltration in BOP 0–1 sites, consistent with a non-inflammatory state. In contrast, BOP 2–3 sites showed disrupted epithelial integrity and increased presence of infiltrating immune cells within the peri-implant mucosa. Protein expression patterns validated the transcriptomic data.

Conclusions

These findings corroborate that BOP 0–1 represents a biologically healthy state, whereas BOP 2–3 reflects true peri-implant inflammation. Together, transcriptomic and protein-level validation challenge dichotomous definitions of peri-implant mucositis and support the need for refined diagnostic thresholds.

Future Directions

Further spatially resolved studies, including multiplex imaging and integration with single-cell datasets, will clarify cell–protein interactions in situ and guide the development of more precise clinical diagnostic criteria.

A Knock-in Strategy to Detect Prx1 Expressing Cells Shows Regeneration of the Periodontium by Means of Prx1-progeny Cells

Dalia Rasheed Issa[1]; Xue Hui Geng[1]; Charles Sfeir[1,2]; Giuseppe Intini[1,2,3]

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2 McGowan Institute for Regenerative Medicine University of Pittsburgh,

3 Hillman Cancer Center University of Pittsburgh, Pittsburgh, PA, United States

Objectives

In a transgenic mouse model, pnPrx1+ cells have been reported to reside within the continuously regenerating periodontal ligament (PDL) of mouse incisors and to contribute to the formation of the PDL in molars. However, their potential involvement in periodontal regeneration of mouse molars within this model remains unclear. In the present study, we aimed to investigate the role of pnPrx1+ cells in periodontal regeneration of mouse molars using a new Prx1-CreER Knock-in/TdTomato mouse model.

Methods

Tracing the contribution of pnPrx1+ cells during tissue homeostasis were assessed in transgenic (Prx1-CreER-GFP/TdTomato) and knock-in (PRX1-CreER-knock-in/TdTomato) mice following tamoxifen administration (one or two doses). Mandibles were collected at 0-, 2-, and 3-days post-injection. To evaluate the role of pnPrx1+ cells in periodontal regeneration, a periodontal fenestration defect was created on the buccal aspects of the mandible involving the roots of the first molar. Twelve mice (8–10 weeks old) were used (n = 6, transgenic; n = 6, knock-in). Each model was randomly divided into two groups: one control and one test group receiving tamoxifen. Mandibles were harvested at 7- and 30-days post-surgery for histological and fluorescence analysis of TdTomato+ cells.

Results

For tracing pnPrx1 cells, the Knock-in mouse model showed TdTomato+ fluorescent cells, which increased proportionally with two doses of tamoxifen. In contrast, no detectable TdTomato+ cells were observed in the transgenic model. In periodontal regeneration, TdTomato+ cells were concentrated within newly formed PDL and in areas adjacent to the periodontal defect of both roots of the first molar at 30 days post-surgery in the test group of Knock-in mice, compared with the transgenic mice. No fluorescent cells were detected in the control groups at 30 days.

Conclusion

pnPrx1 cells contribute to periodontal regeneration in knock-in mice. Incomplete formation of PDL and cementum was observed in both models throughout the observation period.

The Correlation Between BMI Percentile and the Dental Age in Males Across Differing Ethnicities

Emily Brooks, Fayrouz Bazina, Andrew Jheon, Wellington Rody, Alice Goodwin, Fayrooz Abdelkader

Introduction

Understanding the relationship between body mass index (BMI) percentile and dental age is crucial in orthodontics and pediatric dental research, as it influences the timing of treatment. The American

Association of Orthodontists recommends that children have their first orthodontic evaluation at 7 years to evaluate malocclusions, direction, and pattern of facial growth. Growth and development are impacted by both genetic and environmental factors, including BMI. As of 2020, the Centers for Disease Control and Prevention (CDC) estimates that 19.7% of children are classified as obese. Prior studies indicate that overweight and obese children experience accelerated dental development, while underweight children show delayed dental maturity.

Objectives

The relationship between BMI percentile and dental age in White, Black, Asian, and Hispanic male children has not been evaluated. This study aims to identify the possible correlation between BMI, ethnicity, and dental development in males aged 7-15 undergoing orthodontic treatment.

Methods

This retrospective study utilizes existing records of 400 male patients, aged 7 to 15 years, who were examined at the Orthodontic and Pediatric Dentistry Clinic at the University of Pittsburgh. Age, sex, ethnicity, height, weight, and date of panoramic radiographs will be obtained from the electronic patient records in the Orthodontics and Pediatric Dentistry graduate clinic. BMI will be calculated using CDC guidelines, based on height and weight obtained within six months of the radiograph. The Demirjian method will be used to evaluate patients' panoramic radiographs to assess dental maturity.

Statistical analysis will include unpaired t tests (BMI percentile vs ethnicity) and analysis of variance (ANOVA) (BMI vs calculated dental age). The multiple regression model will assess the predictive value of BMI percentile on dental age. Intra-rater reliability will be assessed by re-evaluating 10% of the sample with a Cronbach's alpha correlation.

Conclusion

Findings from this study may identify the influence of BMI and ethnicity on dental development, improving treatment planning in pediatric orthodontics.

Genetic Association of Single Nucleotide Variants in SIX6 Gene With Mandibular Retrognathia/class II Malocclusion and Cleft lip/palate

Rucha Tank, Kate Carnival, Dylan J. Baxter, Mariana Bezamat, Alice Goodwin

Introduction

Pierre Robin Sequence (PRS) is a rare congenital disorder characterized by a triad of clinical features including airway obstruction, glossoptosis, and micrognathia/retrognathia. Additionally, the majority of PRS individuals also have cleft palate. While isolated cases of PRS have mutations/deletions in the SOX9 gene, further RNA Seq experiments determined a differentially expressed gene, SIX6. SIX6 hemizygosity and homozygosity have been shown in several patients with 14q22-q23 deletion syndrome, a rare chromosomal syndrome associated with micrognathia/retrognathia and high arched palate/ cleft palate.

Objectives

The role of SIX6 in craniofacial development has not been established. The aim of this study was to investigate whether SIX6 may be a yet unidentified gene associated with PRS related craniofacial phenotypes. Specifically, the study examined a genetic association between SIX6 and mandibular retrognathia and cleft palate or lip using a candidate SNV approach in a human subject population.

Methods

Saliva samples collected from 330 participants through the Dental Registry and DNA Repository (DRDR) were included. A total of 81 cases with mandibular retrognathism (class II malocclusion, overjet more than 5mm), 9 cases with cleft lip and palate, and 240 participants with normal jaw relationship and no cleft palate were used as comparison. After gathering all samples, DNA extractions were conducted and genotyped for 10 candidate SNVs within the ~190Kb region of SIX6. Samples were amplified and analyzed with QuantStudio 6 Flex and allelic discrimination plots were generated based off the participant genotypes. Genotyping information was then run through PLINK 1.9, a genetic analysis software, to identify associations between genotypes and phenotypes.

Results

Two SNVs were significantly associated with mandibular retrognathia (rs10483727_C, P=0.016, OR=1.69 and rs17097602_T, P=0.013, OR=0.55). These SNVs have been previously associated with glaucoma cases and retinal nerve fiber alterations and association of the SIX6 gene with mandibular retrognathia is a novel discovery.

Body Mass Index Distribution in Male Children Across Different Ethnicities

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Objectives

The purpose of this study is to evaluate the body mass index (BMI) distribution among Caucasian and African American males aged 7-15 years old. This preliminary study is part of two larger studies to determine the correlation between BMI, ethnicity, and skeletal and dental maturity.

Methods

Age, sex, height, weight, and self-reported ethnicity were obtained from Axium EHR. BMI was derived from height and weight, converted to age- and sex-specific percentiles using the CDC Child and Teen BMI calculator, and categorized into "underweight," "healthy," "overweight," "obese," and "severely obese."

Statistical analyses were completed by Dr. James Hartsfield at the University of Kentucky College of Dentistry. As the Shapiro-Wilk test concluded that the chronological age for each ethnic group was not normally distributed, the Wilcoxon two-sample test was used to compare the groups. To compare the percentages of subjects reporting different ethnicities for each BMI category, a contingency table was created and analyzed with Fisher's Exact Test.

Results

A sample of 405 subjects with recorded BMI and ethnicity was obtained. Of the 405 patients included in the study, 42.5% (n=172) were African American and 57.5% (n=233) were Caucasian. The p-value for the Shapiro-Wilk test was significant at <0.0001, indicating the groups were not normally distributed. The p-value for the Wilcoxon two-sample test was 0.6096, which was not significant. Similarly, the p-value for Fisher's Exact Test, which was used to analyze the contingency table, was not significant at 0.6344.

Conclusions

Analysis of the chronological ages of the Caucasian and African American groups showed no significant difference in age distribution at the time height and weight were taken. Additional testing of the data revealed no significant difference in BMI distribution across both ethnicities. Next steps will involve analysis of skeletal and dental age data to investigate the correlation with BMI and ethnicity.

The Effect of Excess Folic Acid on Craniofacial Development in B9d1 Mouse Model

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Since 1998, fortification of cereal grains with folic acid (FA) has been mandatory in the United States to prevent Neural Tube Defects (NTDs) in embryos. Maternal exposure to environmental factors in early development can influence phenotypic variability and cause heritable epigenomic changes. While fortification reduced NTDs, growing FA consumption raises concerns about overexposure and its unknown effects on craniofacial development. Primary cilia are critical for embryonic signaling and craniofacial development and are thought to interact with FA. Ciliary defects have been shown to contribute to neural tube and craniofacial abnormalities, and FA has been shown to exacerbate NTD severity. Mice with null mutations in B9d1, a transition zone gene, on a high FA diet exhibit increased facial narrowing with a significant genotype-diet interaction compared to those on a low FA diet. In this study, heterozygote B9d1 mice were fed high FA (10 ppm) and low FA (2 ppm) diets for three generations. DNA and RNA

from third-generation E11.5 embryos were used for Enzymatic Methylation Sequencing (EMseq) and RNA sequencing (RNAseq). EMseq revealed increased methylation in high FA samples and identified several predicted and known genes with differential methylation. RNAseq uncovered many differentially expressed genes across interactions (genotype by diet, wildtype by heterozygote, wildtype by null) and raised concerns about the methodology behind generating the B9d1 mutation. Two-block Partial Least Squares regression between RNAseq and morphometric landmark data suggested early disruption in the basal plate may explain observed variance. Examination of B9d1 RNA expression in null genotypes also raised concerns of alternative splicing. These findings are currently being investigated along with confirmation of proper genotyping.

Quercetin's Effects on Skeletal Stem Cell Proliferation and Genome Maintenance

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Introduction

When faced with minor injuries, the calvaria can regenerate and repair itself regardless of cause. However, treating critical calvarial bone defects that arise from major trauma or surgical interventions needed to treat cancer, often require methods such as transplantation, matrix implantation, or other forms of bone tissue engineering. Given the limitations of these methods, a possible solution is to utilize compounds such as Quercetin to induce proliferation of skeletal stem cells (SSC) and sustain calvarial bone regeneration.

Question/Hypothesis

We hypothesized that Quercetin can induce proliferation of calvarial SSC (cSSC) and expression of key factors that may help the regeneration process.

Methods

We tested the effects of Quercetin at different concentrations in vitro, using an immortalized cell line of cSSC (1μM, 10 μM, and 100 μM). After treating the cells, we used qPCR (quantitative Polymerase Chain Reaction) to measure the activity of 4 genes that are known to be involved in cell proliferation and genome maintenance: MKI67, SIRT1, SIRT3, and SIRT5.

Results

The dose response analysis shows that 100μM of Quercetin induces a down regulation of MKI67, indicating that cell proliferation may be inhibited at this concentration. However, the same concentration of Quercetin induces expression of SIRT1 and SIRT3, while expression of SIRT5 remains unchanged.

Conclusions

Quercetin at a concentration of 100μM induces expression of certain Sirtuins. However, it may inhibit cell proliferation. Future studies will aim at determining whether the same effects can be replicated in vivo using a mouse calvarial bone regeneration model.

Immunofluorescence Studies (IF) of Maturing Enamel in Plastic Sections

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Abstract

Mature dental enamel, the hardest calcified tissue, contains only 1% of organic matrix. However, during the secretory stage of amelogenesis, enamel matrix (EM) comprises approximately 30% of enamel by weight. The EM is removed during the maturation stage via a coordinated proteolytic process; however, exact spatiotemporal details of this process remain poorly understood, partially because of the difficulties of preserving the integrity of maturing EM in decalcified sections. The objective of this study was to develop a method of visualizing maturing EM in fully calcified specimens. We focused our studies on Amelogenin (Amelx), the major enamel matrix protein with a single phosphorylation site at Serine-16. Phosphorylated Amelx (Amelx(+P) is the key regulator of enamel mineralization during the secretory stage of amelogenesis [1,2].

We developed a novel method for immunofluorescence (IF) studies of forming enamel in sections of murine incisor embedded in LR White resin sections, prepared using the Exakt system. Incisors of 4-week-old rats (n=3) were extracted, dehydrated in ethanol, and embedded in LR White. The blocks were sectioned and ground into <100 μm sections. The sections were etched with 3% lactic acid for 10s and subjected to IF staining using custom antibodies against Amelx(+P) according to the procedures developed in our lab [3].

Using this technique, Amelx(+P) signal was detected in the secretory and early maturation enamel. The signal first disappeared in the inner and mid-enamel layers, while in the outer enamel it extended to the mid-maturation stage, demonstrating a complex dephosphorylation profile.

Conclusion

We present here a novel method to investigate protein localization in maturing enamel without demineralizing the enamel tissue.

[1]- Shin et al., JBC, 2019, 295(7):1943-1959; [2]- Gabe et al., Matrix Biol, 2024:131:17-29; Yang et al., JBC, 2019, [3]- 294(48):18475-18487

Keywords

Amelogenin, Phosphorylated amelogenin, Mature enamel, immunofluorescence

A Multiomics Approach to Establishing Oral and Cardiovascular Disease Connection

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Bezamat Lab

Background

Oral diseases, including dental caries and periodontitis, have long been hypothesized to contribute to systemic inflammation and increased atherosclerotic cardiovascular disease (ASCVD) risk. However, the underlying mechanisms have yet to be completely elucidated. Data from three complementary studies was integrated to evaluate epidemiological, genomic, and metabolomic links between oral health and cardiovascular outcomes.

Methods

Two large dental/medical datasets were analyzed for epidemiological analysis: the Dental Registry and DNA Repository (DRDR; n = 2,247) and the National Health and Nutrition Examination Survey (NHANES; n = 3,202), assessing independent associations between caries indices [decayed, missing, filled teeth (DMFT) and surfaces (DMFS)] and self-reported CVD. To explore biological impacts, the Dental/Heart SCORE cohort (n = 552; age 45–75 years) was analyzed which contains comprehensive oral exams, subclinical atherosclerosis measures [carotid intima-media thickness (CIMT), coronary artery calcium (CAC)], untargeted serum metabolomics, and genome-wide genotyping. Logistic and linear regression models were used, adjusting for traditional risk factors. GWAS significance was defined at $p < 5 \times 10^{-8}$.

Results

In the DRDR, higher DMFS was independently associated with CVD ($p < 0.006$), and in NHANES, DMFT was similarly associated ($p < 0.0001$), even after adjusting for age, sex, smoking, income, and periodontal measures. In the Dental/Heart SCORE cohort, metabolomic profiling revealed overlapping lipid pathways—including sphingolipid and lysophospholipid metabolism—associated with both gingivitis and subclinical atherosclerosis. In contrast, GWAS analyses identified distinct genetic associations for oral and cardiovascular traits (e.g., DMFT near CDC73/KCNT2, CAC near CNTNAP2).

Discussion & Future Directions

Together, these studies demonstrate that higher caries burden is linked to cardiovascular disease risk at the population level, but shared inherited genetics do not completely explain association. Instead, overlapping metabolic pathways, particularly involving lipid metabolism, may represent mechanistic bridges. Our future work will integrate multiomics and larger, multi-ethnic cohorts to clarify causal pathways and evaluate the potential of oral health measures as predictors in cardiovascular risk stratification

Dentin-pulp Organoids as a Model to Study Dental Repair Processes

Reem Alhalabe, Fatima N. Syed

Keywords

Odontoblasts, pulp cells, reparative dentin, inflammation, organoids

The dentin-pulp complex maintains tooth vitality and defends against microbial invasion. Upon bacterial invasion, odontoblasts, at the dentin pulp-interface, initiate a reparative process by synthesizing a new layer of dentin, called reparative dentin. Although these natural repair processes exist, these mechanisms have limited repair capacity. So better models are required to study it. Here, three-dimensional dentin-pulp organoids derived from human dental pulp stem/progenitor cells are used as innovative model to capture the spatiotemporal progression of odontoblast differentiation and mineralization under bacterial stimulation, clarifying the role of odontoblasts in dental repair.

Organoids were engineered and cultured in odontogenic medium and examined at two stages: day 1 (no differentiated odontoblasts; "–odontoblasts") and day 14 (peripheral dentin-like and central pulp-like regions with differentiated odontoblasts; "+odontoblasts"). Organoids were exposed for 24 hours to Porphyromonas gingivalis lipopolysaccharide (LPS). In parallel, 2D cultures of pulp-like DPSCs and DPSCs differentiated to odontoblast-like cells were established to compare responses to LPS. Alizarin Red and quantitative gene expression assays assessed mineral deposition and odontogenic gene expression.

Alizarin Red staining showed mineralized dentin-like tissue in +odontoblast organoids but not in –odontoblast organoids. Also, LPS induced a dose-dependent increase in mineralization in +odontoblast organoids. Additionally, in separated 2D cultures, odontoblast-like cells exhibited higher mRNA expression of RUNX2 and DMP-1 compared to pulp-like cells in response to LPS. Importantly, RUNX2 and DMP-1 expression in odontoblast-like cells increased significantly in a dose-dependent manner in response to LPS, further supporting that the odontoblast-like cells have a distinct response to bacterial LPS characteristic of a reparative phenotype relative to pulp-like cells.

These results suggest that dentin-pulp organoid model recapitulates the biological responses of the natural dentin–pulp complex, particularly its reparative response to bacterial challenge. This organoid platform offers new way to study endodontic diseases, such as pulpitis, and holds potential for screening therapeutic drugs.

Effects of Conditional CK2α1 Knockout on Bone, Dentin, and Enamel in Mice.

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Abstract

Protein kinase CK2 (CK2) is a serine/threonine protein kinase that phosphorylates various substrates containing the acidic motifs S/T-X-X-E/D/pS/p. CK2 regulates cell survival, proliferation, and cellular apoptosis across multiple cell types. However, the role of CK2 in mineralization is poorly understood. We hypothesized that CK2 is required for the formation of hard mineralized tissue. We generated a conditional knockout(cKO) mouse by crossing CK2α1fl/fl with Sp7-Cre mice. We characterized dental and skeletal phenotypes of CK2α1 cKO mice by micro-computed tomography (α-CT) and histological staining in 4-week-old male and female mice. The depletion of CK2α1 in osteogenic cells impaired skeletal growth development, characterized by a lower body weight and length compared to CK2α1fl/fl control mice. The trabecular bone of the femur exhibited severely reduced bone volume fraction, connectivity density, and thickness in cKO mice compared to control mice. The cortical bone was significantly reduced, more porous, and less dense in cKO mice, whereas tissue mineral density remained unchanged compared to control mice. The histological staining using Alizarin red and Von Kossa in undecalcified sections showed fewer calcium and phosphate deposits in cKO compared to control mice. We detected fewer type I and III collagen bundles and a reduced number of collagen networks by picrosirius red staining in cKO sections imaged under both bright and polarized lights, compared to control mice. The dentoalveolar structures showed thinner alveolar bone, shorter roots with thinner dentin and cementum, reduced enamel thickness, and an enlarged pulp chamber in cKO compared to control mice. Our data showed impaired mineralization in dental and skeletal tissues in the absence of CK2, which suggests the requirement of CK2 for hard tissue formation and homeostasis. We plan to investigate the molecular mechanisms by which CK2 regulates mineralization by identifying CK2's phosphoproteomes and intracellular pathways modulated by the kinase during skeletal and dental development.

Source of Funding

Center for Craniofacial Regeneration, University of Pittsburgh

Local Sustained Magnesium Delivery Modulates Immune Response and Uncouples Host Microbiome Interactions

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Magnesium (Mg) is a degradable biometal that releases bioactive Mg2+, the fourth most abundant intracellular cation, with immunomodulatory properties shown to promote anti-inflammatory M2 macrophage polarization. Mg deficiency has been associated with multiple chronic diseases such as diabetes, Alzheimer's disease, rheumatoid arthritis and periodontal disease. Chronic inflammatory diseases are often linked to disrupted host-microbe homeostasis, leading to immune-mediated tissue destruction. Macrophages are key players in this excessive and destructive host response as effector cells of the innate immune system. They have a dual function: defending against pathogens and promoting tissue repair which makes them a promising therapeutic target for managing chronic inflammatory diseases. In this study, we demonstrate the feasibility of a novel controlled Mg delivery strategy for targeting macrophages and its impact on the microbiome. We used periodontal disease (PD) and peri-implantitis (PI) as chronic inflammatory disease models to modulate the immune response for the prevention and treatment of murine PD and PI. Our data show that local sustained delivery of Mg inhibited inflammatory bone loss and polarized M2 macrophages within the surrounding tissues. Intriguingly, 16S-rRNA sequencing showed persistent microbial dysbiosis despite the improved disease outcomes, suggesting an uncoupling of the host-microbiome interactions. In addition, single cell RNA sequencing showed that Mg delivery reversed disease-associated inflammatory pathways via downregulating signal transducer and activator of transcription 3 (STAT3) pathway in monocytes and T-cells. Our findings suggest that local sustained Mg delivery is a feasible therapy for the prevention and treatment of chronic inflammatory diseases characterized by the disruption of host-microbe homeostasis and subsequent immune response-driven tissue damage.

ALC–MDPC Coculture and OCCM Models for Studying Amelogenin and Emdogain Regulation of Mineralization

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Abstract

Mineralization of dental tissues relies on interactions among apical papilla–derived stemlike cells (ALC), mouse dental pulp cells (MDPC), and immortalized mouse cementoblasts (OCCM-30). Extracellular proteins, including amelogenin and Emdogain, regulate these processes, but their effects across in vitro models require further study. This study aimed to develop coculture and cementoblast models to evaluate how coatings and protein treatments influence proliferation and mineralization.

ALC–MDPC coculture models were established using 24-well transwell inserts, with ALC seeded in the insert and MDPC cultured in the lower chamber. OCCM protein regulation studies were performed in 48-well plates. Collagen, laminin, and matrigel coatings were compared, and recombinant porcine amelogenins (P173, rP172) and Emdogain were tested. Readouts included growth curves, Picogreen assays, and Von Kossa staining at multiple time points.

In coculture models, collagen coating enhanced mineral deposition, but reduced proliferation compared with uncoated conditions. Optimal seeding density for ALC was 4,000 cells per insert, reaching confluence by Day 3. Recombinant amelogenins promoted mineralization, with rP172 producing greater effects than P173. Coculture with MDPC further increased mineralization but reduced ALC proliferation, likely due to nutrient competition.

In OCCM experiments, 10,000 cells per well was optimal for assessing proliferation. In the absence of coating, rP172 slightly enhanced proliferation compared with P173, whereas under collagen-coated conditions, P173 showed a modest advantage over

rP172. Regardless of coating status, Emdogain consistently produced the greatest increase in proliferation. Preliminary Von Kossa assays showed more robust mineral deposition by Day 16 compared with Day 11, indicating that longer culture improves detection of OCCM mineralization, though further validation is required.

In conclusion, collagen coating, recombinant amelogenins, and Emdogain differentially regulate proliferation and mineralization in dental cell models. These findings describe coculture and cementoblast models that enable investigation of protein regulation in dental cell proliferation and mineralization.

Bone Marrow Adipose Tissue Depletion in Obese Mice Reduces Osteoclast Differentiation of Myeloid Cells

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Introduction

Bone marrow adipocytes (BMAds) are critical for bone homeostasis, but our understanding of the pathophysiology associated with bone marrow adipose tissue (BMAT) expansion is limited. Our previous studies revealed that obese C57BL/6J mice had increased BM myeloid cells and osteoclast precursors. These findings were coupled with an increase in osteoclasts that resulted in decreased trabecular bone volume and cortical area. We hypothesized that BMAT expansion promoted osteoclastogenesis of myeloid cells, which led to obesity-related bone loss.

Methods

BMAT-depleted mice (BMAd-Pparg KO) and littermate controls (BMAd-Pparg WT) were fed a high-fat diet (HFD; 60% kcal) or sucrose-matched low-fat diet (10% kcal) for 12 weeks starting at 8 weeks old. BMAT-depleted mice were generated by breeding Osterix-FLPo with FLPo-activated Adiponectin Cre mice, which generated a BMAd-specific Cre recombinase (BMAd-Cre). BMAd-Cre mice were then bred with Ppargflox/flox mice to impair BM adipogenesis. RNA was isolated from BMAds for RT-qPCR. Flow cytometric analyses measured immune and osteoclast precursors. Tibial trabecular and cortical bone volume were measured through µCT, and femoral histomorphometric analyses measured bone cell numbers and function.

Results

BMAds isolated from HFD-fed BMAd-Pparg KO mice had decreased MCP-1 expression, a key mediator of osteoclastogenesis and myeloid accumulation. Compared to BMAd-Pparg WT mice, HFDfed BMAd-Pparg KO mice had a decrease in myeloid cells and osteoclast precursors. HFD-fed BMAdPparg KO mice also had decreased serum CTX and TRAP levels, markers indicative of bone resorption. These results coincided with a decrease in osteoclasts and eroded surfaces, which resulted in HFD-fed BMAd-Pparg mice having increased trabecular bone volume.

Conclusions

BMAT depletion prevented obesity-induced trabecular bone loss. These results indicated that BMAT expansion is critical for myeloid cell commitment and differentiation to osteoclasts. Clinically, these results suggest targeting BM adipogenesis could ameliorate abnormal osteoclast formation, which could be of value for the treatment of obesity-related bone loss.

Integrating Clinical, Social, and Genetic Determinants Towards Elucidating Dental-systemic Disease Relationships

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Low-grade, chronic inflammation resulting from dental disease can contribute to elevated systemic levels of inflammation associated with adverse health conditions like diabetes and cardiovascular disease. Previous studies have identified genetic and epidemiological factors as suggestive evidence for a bidirectional relationship between systemic disease and dental disease, but lack dedicated integration of dental disease records and genomic

information, precluding identification of causal factors. Here we leverage electronic health record (EHR), whole genome sequencing (WGS), and survey data from >450K participants in the All of Us Research Program to identify epidemiological and genetic factors contributing to combined dental-systemic disease phenotypes. SNOMED codes corresponding to dental and systemic disease terms (N=13 and 10, respectively) were used to define dental, systemic, and dental + systemic disease cohorts of participants for whom genomic data was available in All of Us (N=3,503, 83,190, and 6,341, respectively). Genome-wide association analysis was conducted on the cohorts with demographically-matched controls sampled from srWGS participants in All of Us (N=414,820), using logistic regression on Hail MatrixTables stratified by both sex and ancestry. Association analysis of the combined dental + systemic disease phenotype identified one genome-wide significant locus (rs115408863, $p < 5 \times 10\text{-}8$) intronic to the gene CPS1 and numerous additional loci with marginal genome-wide significance ($p < 5 \times 10\text{-}6$). Sex-stratified analysis identified loci for female participants ($p < 5 \times 10\text{-}6$) in multiple genes involved in immune response signaling. We then cross-referenced our cohorts with available data in the "Overall Health", "Lifestyle", "Healthcare Access and Utilization", and "Social Factors of Health" surveys (i.e., SDOH) to identify SDOH showing statistically significant stratification and co-variation. These analyses are ongoing, with participants in the dental + systemic cohort with the greatest shift relative to the control in identified SDOHs selected for targeted analysis of genes implicated in the previous GWAS.

Interpositional Implant for Physeal Injury

Seema Patel, DMD Candidate, Class of 2029

Abstract

Physeal (growth plate) injuries in pediatric patients can lead to the formation of bony tethers, resulting in growth deformities such as limb length discrepancies and angular misalignment. Current treatment approaches—including fat grafting, epiphysiodesis, and surgical distraction—often show limited and inconsistent success in preventing re-ossification of growth plate defects. To address this unmet clinical need, this project aims to develop a practical, self-crosslinking hydrogel implant designed to inhibit mineralization within physeal defects and help restore normal growth plate function.

This effort builds upon a previously validated light-activated hydrogel prototype that demonstrated prevention of mineralization in a large animal model. However, light-based crosslinking presents significant barriers in the surgical setting, including limited tissue penetration and the lack of accessible curing equipment in the operating room. To overcome these challenges, we propose reformulating the hydrogel using strain-promoted azide-alkyne cycloaddition (SPAAC), a biocompatible, bio-orthogonal chemistry that enables rapid gelation under physiological conditions.

The reformulated hydrogel will be evaluated for key mechanical properties—such as gelation time and compressive stiffness—as well as its biological performance in preventing mineralization by stem cells and chondrocytes in vivo. In parallel with technical development, we will conduct a targeted market survey of pediatric orthopedic surgeons to evaluate current clinical workflows, unmet needs, and design preferences. This feedback will guide product refinement and support the definition of a clear clinical use.

This project is supported by the University of Pittsburgh's Commercialization Gap Fund, which provides critical resources to validate both the technical feasibility and commercial viability of promising innovations, thereby accelerating their path toward clinical translation and patient impact.

FACULTY PUBLICATIONS

The following Pitt Dental Medicine publications were published between July 1, 2024 and August 15, 2025. They are presented in order of their date of publication.

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Goldfaden JS, **Prasad JL**, Demirturk H, Licht SC, Vargo RJ. An incidentally discovered interradicular radiolucency of the anterior mandible. *The Journal of the American Dental Association*. 2025 Aug 5.

Kobyra JA, Pezzillo M, Bentley ER, Balmert SC, **Sfeir C**, Little SR. Encapsulation of Therapeutic, Low-Molecular-Weight Chemokines Using a Single Emulsion, Microfluidic, Continuous Manufacturing Process. *Pharmaceutics*. 2025 Aug 14;17(8):1056.

PITT DENTAL MEDICINE RESEARCH GRANTS

The following are new Pitt Dental Medicine grants received between July 1, 2024 and August 15, 2025.

Total Grant Awards: $9,330,486.00

Alejandro Almarza, PhD,
Principle Investigator

Juliane Rolim de Lavor,
Post Doctotal Student

Comparing pain phenotypes across lower back, knee, and TMJ

17-Sept. 17, 2024-Aug. 31, 2025
Total: $356,270.00

Alejandro Almarza, PhD,
Principle Investigator

Mairobys Socorro Gonzalez, DDS, PhD,
Co-investigator

TMJ Disc Regeneration

Aug. 8, 2024-July 31, 2028
Year one: $337,875.00
Total: $1,351,500.00

Alice Goodwin, DDS, PhD,
Principle Investigator

Mariana Bezemat Chappel, DDS, PhD,
Co-investigator

SIX6; a new candidate gene in the etiopathogenesis of Pierre Robin sequence

Oct. 1, 2024-Sept. 30, 2025
Total: $30,000.00

Andrea Ravida, DDS, MS, PhD,
Principle Investigator

Martinna Bertolini, DDS, MS, PhD;
Debora Dias, DDS. MSC,
Co-investigators
Maryia Karaban, Post Doctotal Student

Comparative Analysis of Three Types of Collagen Membranes Exposed to the Oral Environment: A randomized clinical trial

Jan. 30, 2025-Jan. 29, 2026
Total: $18,412.00

Ariadne Letra, DDS, MS, PhD,
Principle Investigator

Leveraging whole genome sequencing and functional genomic characterization to improve NSCLP gene discovery

May 1, 2025-Feb. 28, 2030
Year one: $645,034.00
Total: $3,022,042.00

Bavya Mavila Chathoth,
Principle Investigator

The function of PTHR1 signaling in the development, abnormalities, and regeneration of craniofacial and skeletal structures

July, 1, 2024-June 30, 2025
Total: $9,981.00

Brent Vasquez,
Principle Investigator

Investigating Interactions Between Phosphorylated Amelogenin and Acid Phosphatase 4 Expression

Aug 1, 2024-July 31, 2025
Total: $54,774.00

Gina DeLeonibus,
Principle Investigator

Alice Goodwin, DDS, PhD,
Co-investigator

SIX6: A New Candidate Genee for Pierre Robin Sequence

July 1, 2025-June 30, 2026
Total: $6,000.00

Giuseppe Intini, DDS, MS, PhD,
Principle Investigator

Generation and maintenance of novel transgenic immunocompetent osteosarcoma mouse lines

Dec. 1, 2024-Nov. 30, 2025
Total: $17,770.00

Giuseppe Intini, DDS, MS, PhD,
Principle Investigator

Dobrawa Napierala, PhD,
Co-investigator

Luigi Mancinelli, Roberta Di Carlo, Dalia Rasheed Issa,
Post Doctotal Students

Harnessing PRX1 expressing cells for endogenous periodontal regeneration

Aug. 12, 2024-May 31, 2029
Year one: $565,228.00
Total: $2,846,262.00

Ildeu Andrade Jr., DDS, PhD,
Principle Investigator

3D-Printed versus Laboratory-Fabricated Hyrax Expanders: A

Randomized Controlled Clinical Trial

July 1, 2024-June 30, 2025
Total: $20,000.00

Juan Taboas, MS, PhD,
Principle Investigator

Herbert Ray, Co-investigator

Commercialization of the Vital-Dent Regenerative Pulp Therapy

Aug. 1, 2024-July 31, 2026
Total: $236,271.00

Kenneth Stephen,
Principle Investigator

Impact of model base design and 3D print settings on the quality of orthodontic retainers

July 1, 2024-June 30, 2025
Total: $6,000.00

Lias Alberti Ferreira,
Principle Investigator

Ariadne Letra, DDS, MS, PhD; Danilo Cassiano Ferraz, Letícia Chaves de Souza, DDS, MS, PhD
Co-investigators

Understanding the role of SPP1 in apical periodontitis

Jan. 1, 2025-Dec. 31, 2025
Total: $15,176.00

Mairobys Socorro,
Principle Investigator

Molecular Signaling in Dentinogenesis and Dental Caries Risk

Sept. 19, 2024-Sept. 18, 2026
Total: $296,734.00

Martinna Bertolini, DDS, MS, PhD,
Principle Investigator

Tracking Keratin 6+ Cell Expansion in Oropharyngeal Mucosal Sites Post-Candida albicans Infection: Determining a spatial and temporal relationship

Sept. 1, 2024-Aug. 31, 2025
Total: $10,000.00

Rebecca Green, PhD,
Principle Investigator

Epigenotype- Genotype -Phenotype interactions in facial development

July 1, 2024-June 30, 2026
Year one: $154,602.00
Total: $309,326.00

Renato Silva, DDS, MS, PhD,
Principle Investigator

Ariadne Letra, DDS, MS, PhD; Danilo Cassiano Ferraz, Letícia Chaves de Souza, DDS, MS, PhD
Co-investigators

Exploring the role of CCL2 in apical periodontitis

Jan. 0, 2025-Dec. 31, 2025
Total: $45,434.00

Robert Weyant, MS, DMD, DrPh,
Principle Investigator

Elizabeth Pawlowicz, DMD;
Lawrence Mauro, DMD, FAGD, MBA,
Co-investigators

Postdoctoral Training in General, Pediatric and Public Health Dentistry and Dental Hygiene

July 1, 2025-June 30, 2030
Year one: $76,820.00
Total: $453,212.00

Yejin Cho,
Principle Investigator

Local Delivery of CCL2 to Reverse Peri-implant Bone Loss in Murine Peri-implantitis

Sept. 1, 2024-Aug. 31, 2027
Year one: $54,774.00
Total: $164,322.00

Debra Polk, AB, PhD,
Principle Investigator

Health Sciences Bridge Funding (HSBF)

March 1, 2025-Feb. 28, 2026
Total: $40,000.00

Juan Taboas, MS, PhD,
Principle Investigator

Interpositional Implant for Physeal Injury

June 5, 2025-May 31, 2026
Total: $15,000.00

Arshia Ashjaei,
Principle Investigator

Andrew Jheon, DDS.. PhD,
Co-investigator

EPITHELIAL RESTS

July 1, 2025-June 30, 2026
Total: $6,000.00

UNIVERSITY OF PITTSBURGH SCHOOL OF DENTAL MEDICINE

University of Pittsburgh

Health Sciences
School of Dental Medicine