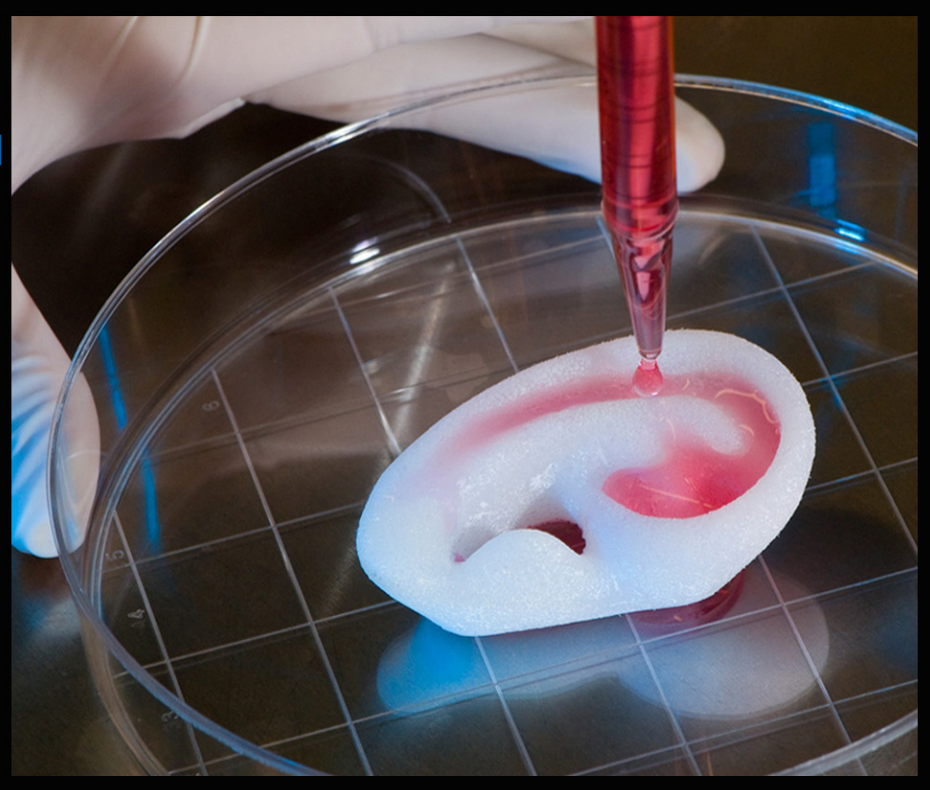
Boston University Henry M. Goldman
School of Dental Medicine

BOSTON UNIVERSITY

2012 SCIENCE DAY

Thursday, March 15
9 a.m. to 4:30 p.m.
670 Albany Street
BU Medical Campus

Keynote Lecture, 1 p.m.
670 Albany Street
Auditorium



"Regenerative Medicine: New Approaches to Healthcare" by Anthony Atala, M.D.

Dr. Atala is the Director of the Wake Forest Institute for Regenerative Medicine, and the W.H. Boyce Professor and Chair of the Department of Urology at Wake Forest University. Dr. Atala is a practicing surgeon and a researcher in the area of regenerative medicine.

Poster viewing
9 a.m. to 4:30p.m.
first floor hallways & foyer

Poster judging
10 a.m. to 1 p.m.
first floor hallways & foyer

Oral presentations
2 to 4:30 p.m.
rooms 107 and 108

- **Dental vendor exhibition, 9 a.m. to 2 p.m., 100 East Newton Street 1st floor hallways and cafeteria**
- **Complimentary lunch, noon to 1 p.m. 670 Albany Street, rooms 107 and 108**
- **Science Day is open to pre- and post-doctoral dental students, post-doctoral fellows, faculty and staff.**
- **Registration and abstract submission deadline: Tuesday, March 6 at 10 a.m.**
- **Online registration: http//gsdm.bumc.bu.edu/portal or contact Afaf Hourani at ahourani@bu.edu or 617-414-1048.**

Science Day is presented by the American Student Dental Association, the Student Research Group and the Office of the Associate Dean for Research at Boston University Henry M. Goldman School of Dental Medicine.

Images courtesy of Dr. Anthony Atala

bu.edu/dental/scienceday
bu.edu/dental-research/student-research/

I am honored to welcome not only students, staff, and faculty from the Henry M. Goldman School of Dental Medicine, but also students, staff, and faculty from across the Boston University Medical and Charles River Campuses, members of Boston University's Pre-Dental Society, Boston Medical Center CityLab Scholars, and friends and colleagues involved in scientific research throughout the Boston area.

Research is a critical component of both our students' education and our faculty's activities at the Henry M. Goldman School of Dental Medicine. Strengthening our research efforts and in particular, our collaborative interdisciplinary research is central to our and the University's strategic plan. It is absolutely a priority that we continue our efforts in this important area in order for us to maintain our competitiveness in obtaining grant funding, identifying and nurturing our best researchers within the Henry M. Goldman School of Dental Medicine, recruiting the best researchers from outside our Institution, and most important of all, maintaining our stature within the University as an integral member of what President Brown describes as a great urban research university.

Collaboration with other Schools and Colleges of the University will become increasingly important as we work to strengthen our position within the University and as we look to become the premier Center of Excellence in Education, Research and Patient Care in this nation.

The GSDM Science Day is a wonderful day of science and I applaud all who participated. I'd like to thank Dr. Maria Kukuruzinska and her staff for putting together such a comprehensive event, featuring cutting-edge research, and highlighting the work of our students and fellows. We are extremely honored to have Dr. Anthony Atala deliver the keynote address today at Science Day.

Dean

Jeffrey W. Hutter

Our selection of Dr. Anthony Atala as the keynote speaker to present his extraordinary research findings in the area of tissue regeneration and repair emphasizes how science drives clinical applications to healthcare. Dr. Atala's topic is timely, as scientists search for strategies to regenerate and repair damaged salivary glands following radiation treatment for head and neck cancers and in autoimmune exocrinopathy disorders, such as Sjogren's Syndrome.

Associate Dean for Research

Maria Kukuruzinska

(l-r) Dean Jeffrey W. Hutter, Dr. Anthony Atala, and Associate Dean for Research Maria Kukuruzinska

Boston University Henry M. Goldman
School of Dental Medicine

SCIENCE DAY 2012

Thursday, March 15

Regenerative Medicine: New Approaches to Healthcare by Anthony Atala, MD

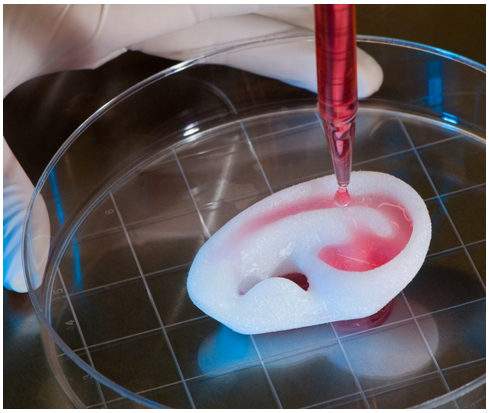


Image courtesy of Dr. Anthony Atala

Dr. Atala is the director of the Wake Forest Institute for Regenerative Medicine and the W.H. Boyce Professor and Chair of the Department of Urology at Wake Forest University. He is a practicing surgeon and researcher in the field of regenerative medicine.

Science Day is supported by the American Student Dental Association, the Student Research Group and the Office of the Associate Dean for Research at Boston University Henry M. Goldman School of Dental Medicine.

1 to 2 p.m.
670 Albany Street
Medical Campus

9 a.m. to 2 p.m.

vendor exhibition

100 East Newton Street, first floor hallway and cafeteria

3M/ESPE
A-Dec
Arcari Dental Laboratories
Colgate Oral Pharmaceuticals
Designs for Vision
Door to Door Dental
GlaxoSmithKline
Henry Schein Dental
Hu-Friedy
Johnson and Johnson
MIDMARK Corporation
Patterson Dental
Phillips Sonicare
Procter & Gamble Professional Oral Health (Crest OralB)
Surgitel
Triodent

1 to 2 p.m.

keynote research presentation

670 Albany Street, auditorium

Dr. Anthony Atala
Director, Wake Forest Institute for Regenerative Medicine
W.H. Boyce Professor and Chair, Department of Urology,
Wake Forest University

Regenerative Medicine: New Approaches to Healthcare

Patients with diseased or injured organs may be treated with transplanted organs. There is a severe shortage of donor organs which is worsening yearly due to the aging population. Regenerative medicine and tissue engineering apply the principles of cell transplantation, material sciences, and bioengineering to construct biological substitutes that may restore and maintain normal function in diseased and injured tissues. Stem cells may offer a potentially limitless source of cells for tissue engineering applications and are opening new options for therapy. Recent advances that have occurred in regenerative medicine will be reviewed and applications of these new technologies that may offer novel therapies for patients with end-stage tissue and organ failure will be described.

9 a.m. to 1 p.m.

poster presentations

670 Albany Street, first floor hallways

Pre-doctoral Students

Ella Botchevar, Lillelenny Santana, Jason Mahaffey, Josh Gilbert, Mona Haghani, Leslie Will, Wanda Wright, Sharron Rich and Judith Jones. Department of General Dentistry: "Oral Health Related Quality of Life in Orthodontic Patients."

Joshua Gilbert, Souichiro Oda and Leslie Will. Department of Orthodontics: "Comparison Between Preformed Archwires and Dental Arch Forms."

Sultan Muhammad, Gangli Liu, Pritam Sengupta, Basem Jamal, Meghan P. Bouchie and Maria A. Kukuruzinska. Department of Molecular and Cell Biology: "Upregulation of Cthrc1 N-glycoprotein Marks OSCC Tumor Spread."

Nathan Ng, Elizabeth Krall Kaye and Raul Garcia. Department of Health Policy & Health Services Research: "Coffee Consumption and Periodontal Disease in Men."

Post-doctoral Students

Reem Aljamaan, Yoshio Ohyama, Malcolm Snead and Yoshiyuki Mochida. Department of Periodontology and Oral Biology: "Expression of WDR72, Causative Gene for Amelogenesis Imperfecta in Ameloblasts."

Lea El Hachem, Eva Helmerhorst and Frank Oppenheim. Department of Periodontology & Oral Biology: "Histatin 5 Antifungal Activity Towards C. Albicans and C. Glabrata."

Tone B. Enger, Sheede Khalil, Meghan P. Bouchie, Hilde K. Galtung, Kathrine Skarstein, Oyvind Palm, Janicke L. Jensen and Maria A. Kukuruzinska. Department of Molecular & Cell Biology: "Identification of Cell Polarity and Mechanosensing Defects in Sjogren's Syndrome."

Mark Jesin, Stephanie Rashewsky, William Tobler, Suresh Agarwal, Peter Burke and Andrew Salama. Department of Oral & Maxillofacial Surgery: "Analysis of Racial Disparities in Head and Neck Fractures."

Matt Steuer and Pushkar Mehra. Department of Oral & Maxillofacial Surgery: "TMJ Arthrocentesis: 10 year Experience Outcomes."

Gabriel Blumenkranz Sanchez, Guoxian Wei and Eva Helmerhorst. Department of Periodontology & Oral Biology: "A Comparison of Gliadin Degradation by Natural Colonizers of the Human Gastrointestinal Tract."

Nathan Turley, Pushkar Mehra, David Cottrell and Laishing Chou. Department of Restorative Sciences/Biomaterials: "Maxillary and Mandibular Reconstruction Surgery with Bone-Tissue Engineering."

Andres Jimenez Wolf, Na Tian, Josh Hansen, Dan Leffler, Ciaran Kelly and Eva Helmerhorst. Department of Periodontology & Oral Biology: "Clinical Study Design for Investigating Salivary Protein Composition in Healthy and Celiac Patients."

Post-doctoral Fellows

Balazs Nemeth and Miklós Sahin-Tóth. Department of Molecular & Cell Biology: "Trypsinogen Isoforms in the Mouse Pancreas."

Andras Szabo and Miklós Sahin-Tóth. Department of Molecular & Cell Biology: "The Chymotrypsin C-Sensitive Leu81-Glu82 Peptide Bond in Human Cationic Trypsin Exhibits Unusual Thermodynamic Stability."

2 to 4:30 p.m.

oral presentations

670 Albany Street, rooms 107 and 108

Pre-doctoral Students

Ray English, Meghan P. Bouchie and Maria A. Kukuruzinska. Department of Molecular & Cell Biology: "Hippo Signaling Pathway in Submandibular Gland Development: Potential Interplay with N-glycosylation."

Marc Horton, Ayesha Ghulam, Sharron Rich, Kathy Lituri, Patricia Whitworth, Heavenly Mitchell, Sheree Norquist and Michelle Henshaw. Department of Health Policy & Health Services Research: "Effect of Oral Health Promotion Provided by Public Health Nurses on the Behaviors of Mothers with Infants."

Post-doctoral Students

Ahmed Almehmadi, Yoshio Ohyama, Haytham Jaha, Sundharamani Venkitapathi, Reem Aljamaan, Masaru Kaku and Yoshiyuki Mochida. Department of Periodontology & Oral Biology: "The Use of Vwc2 Protein as a Novel Approach to Induce Bone Formation."

Debora Heller, Eva Helmerhorst, Bruce Paster and Frank Oppenheim. Department of Periodontology & Oral Biology: "Microarray Based Characterization of Early In Vivo Acquired Enamel Pellicle Colonizers."

Nedda Hifeda, Russell Giordano and Richard Pober. Department of Restorative Sciences/Biomaterials: "Bond Strength Between Veneer Porcelains and CAD/CAM Ceramic to Titanium."

Roozbeh Khosravi and Philip Trackman. Department of Periodontology & Oral Biology: "Novel Insights into Diabetic Bone Complications."

Olena Norris and Pushkar Mehra. Department of Oral & Maxillofacial Surgery. "Maxillofacial Gunshot Injuries at an Urban Level I Trauma Center - 10-year Analysis."

Andrea Schnur and Miklós Sahin-Tóth. Department of Molecular & Cell Biology: "Rare Cationic Trypsinogen Mutations Found in Subject with Pancreatitis are Harmless Variants."

Sundharamani Venkitapathi, Yoshio Ohyama, Haytham Jaha, Ahmad Almehmadi, Reem Aljamaan and Yoshiyuki Mochida. Department of Periodontology & Oral Biology: "Characterization of the Functions of Evc and Evc2 Proteins in Ellis -Van Creveld Syndrome."

Post-doctoral Fellows

Sebastian Beer and Miklós Sahin-Tóth. Department of Molecular & Cell Biology: "Functional Defects Caused by Chymotrypsin C (CTRC) Mutations in Chronic Pancreatitis."

Mohammed Nadershah and Pushkar Mehra. Department of Oral & Maxillofacial Surgery: "Efficacy of Anti-inflammatory Drugs in Third Molar Surgery Laboratory and Clinical Correlation."

Jiayi Zhou and Miklós Sahin-Tóth. Department of Molecular & Cell Biology: "A Potential Novel Pathogenetic Mechanism of Chronic Pancreatitis in Chymotrypsin C (CTRC) Mutants."

Judges on Science Day 2012

Salomon Amar
Srinivas Ayilavarapu
Eva Helmerhorst
Bo Hou
Richard D'Innocenzo
Judith Jones
Elizabeth Kaye
Yoshiyuki Mochida
Dan Nathanson
Miklos Sahin-Toth
Andrew Salama
Philip Trackman
Guoxian Wei
Leslie Will

Oral Health Related Quality OF Life in Orthodontic Patients

Lillelenny Santana, Jason Mahaffey, Josh Gilbert, Ella Botchevar, Mona Haghani, Leslie Will, Wanda Wright, Sharron Rich and Judith Jones
Department of General Dentistry

The purpose of this study is to ascertain the validity and reliability of the PedsQL (Pediatric Quality of Life Inventory) and TOQL (Teen Oral Health-related Quality of Life) in teens with and without malocclusion from the patients' and parents' perspectives. Additionally, this study aims to assess the effect of orthodontic treatment on patients' scores as an indication of oral and overall quality of life. The investigators hypothesize the following: H1: Severity of malocclusion is associated with scores on both the TOQL and PedsQL. H2: The magnitude of the association will be stronger for the oral-specific instrument, the TOQL, than the generic PedsQL. **METHODS: Design and IRB:** This is a cross-sectional, longitudinal study of teens' and parents' perception of the impact of teens' teeth and/or mouth on the teen's oral specific health-related quality of life. The parents and teens are also asked to rate the aesthetic appearance of the teen's teeth relative to the Aesthetic Component (AC) of the Index of Orthodontic Treatment Need (IOTN). This project was approved by the Internal Review Board at Boston University Medical Campus. All participants assented, and their guardians gave written informed consent. **Sample Selection and Inclusion Criteria:** Subjects selected for this study are teens aged 10-18 years old, who fall within the Grade 2 or greater category of the Dental Component (DC) of the IOTN. Patients with mental or physical disabilities, or patients who have previously undergone orthodontic treatment, were excluded from the study. Because interpreters were not available to ensure patient understanding of consent and survey, Spanish speaking subjects and/or guardians were also excluded. The comparison group is comprised of Pediatric Dentistry patients not undergoing orthodontic therapy. **Study Procedures:** Consents are obtained at the treatment planning appointment and both subject and guardian rate AC of the IOTN and complete the PedsQL and TOQL surveys. An Oral Health Screener form assessing the current oral health status of the subject is completed by the dentist during this visit. At completion of orthodontic treatment, the PedsQL and TOQL surveys are answered a second time by the subject and guardian. Subjects who consent to participate are offered $5.00 following completion of each survey. **Outcomes:** Outcomes of interest are the relationships between subjects' malocclusion and the overall oral health-related quality of life and overall quality of life. The oral health-related quality of life is measured using the TOQL Inventory. **Measures:** Both teen and parent complete the PedsQL, TOQL, and rate the subject's Aesthetic Component of IOTN. **Analyses:** Data from surveys is double entered into a database and analyzed using SAS® (*Statistical Analyses Software, Version 9.2, Cary, NC*). Using baseline data, the sample population is described with respect to age, gender, race and ethnicity, insurance, parent's and teen's education and treatment need. Baseline TOQL scores overall and by domain are computed and compared to teens not undergoing orthodontic treatment, matched by age and gender from previous studies (Varni et al., 1999; Huntington et al., 2011). Simple regressions are used to examine the relationships between TOQL (as the dependent variable) and IOTN scores. **Results**: Total participants (parents, teens) Descriptive, By IOTN scores

Comparison Between Preformed Archwires and Dental Arch Forms

Joshua Gilbert, Souod Oda and Leslie Will

Department of Orthodontics

Objectives: The objective of this study was to compare Japanese and Caucasian Class I and II maloclussion to commercially available preformed Nickel Titanium (NiTi) archwires. **Methods:** The results published by Nojima et al 2001 were used for the mandibular arch values of intercanine width, first molar width, canine depth, and first molar depth. 22 popular NiTi archwires that are commercially available in both Japanese and American markets were used for analysis. These archwires were scanned by using a flatbed scanner and digitally measured using ImageJ software. The archwire widths were measured at the level of the mean canine and first-molar depths, and then compared to Nojima's results. Measurements were repeated and the random error was analyzed by Dahlberg formula. Non-parametric Mann-Whitney test was used to compare archwire widths based on the Caucasian and Japanese dental arch depths. **Results:** Caucasians have a narrower and deeper canine measurement when compared to Japanese in both Class I and Class II malocclusion. The width of the preformed archwires at canine and first molar level were wider than the dental arches, in both Caucasian and Japanese, derived from the FA points of each tooth. Statistically significant difference was found between Japanese and Caucasian in archwire width of class I dental arch base. **Conclusions:** There was a large variation of archwire widths observed, and this variation does not coincide with the mandibular malocclusion dental arch. The mean difference of the width between preformed archwire and dental arch form in Caucasian appeared to be 1-2mm wider than Japanese due to Caucasian's deeper dental arch.

Upregulation of Cthrc1 N-Glycoprotein Marks OSCC Tumor Spread

Sultan Muhammed, Gangli Liu, Pritam Sangupta, Basem Jamal, Meghan Bouchie and Maria Kukuruzinska
Department of Molecular and Cell Biology

The canonical Wnt signaling pathway and the metabolic pathway of protein N-glycosylation are known to control many developmental processes including cell proliferation and differentiation. In the canonical, or β-catenin-dependent, Wnt signaling pathway, Wnt ligands, such as Wnt3a, bind via co-receptor LRP5/6 to Frizzleds (Fzds) receptors which leads to stabilization of β-catenin. This, in turn, promotes translocation of β-catenin to the nucleus where it functions in transcriptional regulation of target genes. Previously, we reported that cellular discohesion in oral squamous cell carcinoma (OSCC) occurred, in part, due to inappropriate upregulation of DPAGT1, the first gene in the N-glycosylation pathway and its key regulator, which resulted in extensive N-glycosylation of E-cadherin. **Objective:** Recent work in our laboratory has shown that DPAGT1 is a target of canonical Wnt signaling. Activation of canonical Wnt leads to increased expression of DPAGT1 and N-glycosylation of Wnt proteins, Wnt3a and LRP5/6, leading to further upregulation of DPAGT1 in a positive feedback loop. Inappropriate activation of the canonical Wnt/DPAGT1 loop drives OSCC proliferation. Another Wnt pathway, the planar cell polarity (PCP) pathway, has been implicated in cytoskeletal reorganization, cell movement and polarity, and its disregulation has been linked to tumor spread. In the noncanonical Wnt pathway, Wnt5a binds Fzds receptor which initiates downstream events leading, in part, to the activation of RhoA and/or JNK and cell migration. Recently, the secreted glycoprotein collagen triple helix repeat containing 1 (cthrc1) has been identified as a novel component of the PCP pathway required for the clustering of Wnts and Fzds and activation of the pathway. **Results:** In this study, we show that cthrc1 is dramatically upregulated in OSCC specimens, localizing to the invasive front of tumor islands in vivo, and this correlates with its increased modification with complex N-glycans. **Conclusions:** Since N-glycosylation has been shown to be required for effective membrane anchoring of cthrc1, our studies suggest that the upregulation of DPAGT1 in OSCC is involved in the activation of the PCP pathway and oral tumor spread. *Supported by NIH grant RO1DE014437.*

Coffee Consumption and Periodontal Disease in Men

Nathan Ng, Elizabeth Krall Kaye, Raul Garcia
Department of Health Policy and Health Services Research

Objectives: Caffeine is a competitive inhibitor of phosphodiesterase which raises cellular cyclic adenosine monophosphate, activates protein kinase A, inhibits TNF-α synthesis and subsequently reduces inflammation in animal models. The purpose of this study was to determine if coffee intake, a major source of caffeine among older men, is associated with periodontal disease. **Methods:** Participants were 394 dentate, non-Hispanic white males in the VA Dental Longitudinal Study who attended 3 oral examinations between 1987 and 1998. Subjects are not VA patients but receive medical and dental care in the private sector. Mean age was 68 years. Probing pocket depth (PPD) was measured by calibrated examiners on each tooth and alveolar bone loss (ABL) was measured on radiographs in increments of 20% with a modified Schei ruler method. Bleeding on probing (BOP) was noted. Indices of moderate-to-severe periodontal disease were defined as cumulative numbers of teeth ever exhibiting PPD≥4mm or ABL ≥40%. Average coffee intake was obtained from food frequency questionnaires and the distribution was divided at the median (≤1 cup/day vs. >1 cup/day). Repeated measures generalized linear models estimated mean number of teeth with moderate-to-severe disease PD and BOP at each examination by coffee intake level. Covariates were age, alcohol consumption, body mass index, smoking, diabetes status, education, and frequencies of flossing, toothbrushing, periodontal treatment and cleanings. **Results:** There was no statistically significant difference in moderate-to-severe PPD between the low and high coffee intake groups. However, consumption of >1 cup of coffee/day was associated with fewer teeth overall with moderate-to-severe ABL ($p=0.02$) compared to ≤1 cup/day. Men who drank >1 cup of coffee/day also had fewer teeth with BOP ($p<0.09$). **Conclusions:** Caffeine, which is found in coffee, may have anti-inflammatory effects and reduce periodontal bone loss in humans. *Supported by the Massachusetts Veterans Epidemiology Research and Information Center and the US Department of Veterans Affairs Cooperative Studies Program.*

Expression of WDR72, Causative Gene for Amelogenesis Imperfecta in Ameloblasts

Reem Aljamaan, Yoshio Ohyama, Malcolm Snead, Yoshiyuki Mochida
Department of Periodontology and Oral Biology

Objective: Patients with Amelogenesis Imperfecta (AI) caused by WDR72 mutation display several dental phenotypes including enal hypomaturation. It has been reported that overexpression of WDR72 was localized at cytoplasm in non-odontogenic cells. However, the molecular/biological function of this intracellular protein was not fully examined in odontogenic cells. To further characterize the function of WDR72, as a preliminary step, the present study is sought to investigate the expression and localization of endogenous WDR72 in mouse tissues and ameloblast cell line, LS8. **Methods:** The expression of WDR72 transcript was analyzed using several mouse tissues by real time PCR. The full coding sequence of human WDR72 was subcloned into pcDNA3-Flag vector and used for a positive control. The specific antibody against WDR72 was generated and the protein expression was examined by immunoprecipitation (IP) - Western Blot (WB) techniques in LS8 cells. The localization of endogenous WDR72 protein was also visualized by immunofluorescence-based staining method in the same cell line. **Results:** The expression of WDR72 was highly detected in teeth and also observed in LS8 cells by real time PCR. The IP-WB analysis was performed using cell lysates and the endogenous WDR72 as well as Flag-WDR72 protein was clearly detected, demonstrating the successful antibody generation and feasibility of the experiment. The subcellular localization of endogenous WDR72 protein was visualized in LS8 cells and detected at the cytoplasm. **Conclusion:** We generated the specific antibody against WDR72 protein and the characterization of the antibody was successful. Our data demonstrated the intracellular expression/localization of WDR72 in odontogenic cell line, LS8 ameloblasts. This antibody will help us investigate the patho-physiological, and further molecular function of WDR72 in enamel formation/mineralization. *Supported by NIH grant DE019527 and Boston University School of Dental Medicine.*

Histatin 5 Antifungal Activity Towards C. albicans and C. Glabrata

Lea El Hachem, Eva Helmerhorst, Frank Oppenheim
Department of Periodontology and Oral Biology

Introduction: Candida albicans is an opportunistic fungus colonizing the oral cavity. Histatin 5, a basic histidine-rich protein, exerts fungistatic as well as fungicidal activities towards C. albicans. Another Candida species: C. glabrata showed a much lower sensitivity to histatin 5. The aim of this study was to compare C. albicans and C. glabrata blastoconidiaand spheroplast cell sensitivities to histatin 5 in growth inhibition and killing assays. **Methods:** C. albicans and C. glabrata cells were cultured in 20% Sabouraud Dextrose Broth at 30°C. After 24h cells were harvested, diluted in the same broth and exposed to a dilution series of histatin 5. After 24h incubation, growth inhibition was monitored spectrophotometrically at 620 nm, and expressed as the IC50 value. For the cell killing assay, the cultured cells were aliquoted into two portions. The first half was diluted in 10mM phosphate buffer containing 1.4M sorbitol. The second half was first converted into spheroplasts using zymolase. Blastoconidia and spheroplasts were mixed with a dilution series of histatin 5, and after 1.5h incubation, cells were diluted and plated on agar,followed by colony counting and LC50 value determination. **Results:** Histatin 5 showed IC50 values of 9.75±0.25 and >225 ug/ml and LC50 values of 3.2±0.2ug/ml and >200ug/ml towards C. albicans and C. glabrata, respectively. Our data confirm reduced sensitivity of C. glabrata in both growth inhibition and killing assays. Spheroplast formation of C. albicans and C. glabrata was successful, but due to low inherent viability of the preparations LC50 values could not yet be established. **Conclusion:** Fungal infections are important oral manifestations of immune suppression and naturally occurring salivary proteins are excellent candidates for controlling such infections. The most prevalent fungus in such infections, C. albicans, is susceptible to histatins, but infections caused by C. glabrata might need alternative antifungal agents for treatment.
Supported by NIDCR grants DE05672 and DE07652.

Identification of cell polarity and mechanosensing defects in Sjogren's Syndrome

Tone B. Enger, Sheede Khalil, Meghan P. Bouchie, Hilde K. Galtung, Kathrine Skarstein, Oyvind Palm, Janicke L. Jensen and Maria A. Kukuruzinska
Department of Molecular and Cell Biology

Sjogren's Syndrome (SS) is an autoimmune disease characterized by lymphocytic infiltration and secretory dysfunction of exocrine tissues, including salivary glands. Our initial findings revealed defects in E-cadherin- and ZO-1-mediated cell-cell junctions in a NOD mouse model of SS and in human SS specimens, suggesting that secretory dysfunction was a consequence of structural defects and loss of cell polarity. Indeed, previous studies using a NOD mouse model of SS showed no apparent correlation between lymphocytic infiltration and secretory dysfunction. **Objective:** To identify cell biological defects in SS, we assessed the localization of TAZ, a transcriptional coactivator with key roles in cell polarity and in mechanosensing the extracellular matrix (ECM) stiffness. We aligned changes in TAZ localization with those in fibronectin, a component of the ECM, and in matrix metalloproteinase 9 (MMP9) that functions in the remodeling of the ECM. **Methods:** Formalin-fixed and paraffin-embedded minor labial salivary gland SS specimens and non-compatible controls were stained for ZO-1, E-cadherin, β-catenin, MMP-9, fibronectin and TAZ using indirect immunofluorescence and analyzed by confocal microscopy. **Results:** In a subset of SS specimens, E-cadherin, β-catenin and ZO-1 exhibited disrupted staining. Fibronectin organization was less prominent, displaying a punctate pattern surrounding the acini. In contrast, MMP-9 was more pronounced in the basal regions of acini and colocalized with nuclei. Importantly, in SS specimens TAZ was less localized to cell-cell borders, exhibiting more nuclear and cytoplasmic staining, suggesting that the observed changes in the ECM and polarity are linked to its altered distribution. **Conclusion:** Our studies show for the first time that structural defects in a subset of SS phenotypes may be associated with changes in the distribution of TAZ, a regulator of cell polarity and a sensor of mechanical cues. *Supported by NIH grant RO1DE014437.*

Analysis of Racial Disparities in Head and Neck Fractures

Mark Jesin, Stephanie Rashewsky, William Tobler, Suresh Agarwal, Peter Burke and Andrew Salama
Department of Oral and Maxillofacial Surgery

Introduction: Studies have shown that racial disparities exist throughout the spectrum of health care, including access to care, pain control, oncology, cardiology, transplantation, and trauma.1 Minority race and lack of insurance are predictive of increased morbidity and mortality in trauma patients.2-3 The objective of this study was to determine the impact of race and insurance status on outcomes in patients admitted to Boston Medical Center following head and neck fractures. **Methods:** We performed a retrospective cross-sectional analysis of patients with head and neck fractures using data from the Boston Medical Center Trauma Registry (November 2001-November 2010). Multivariate analysis was used to adjust for age, sex, mechanism of injury, injury severity score, and shock while evaluating for outcomes; hospital length of stay, number of procedures performed, discharge status, mortality and mortality by race and insurance status. **Results:** 2357 patients met the inclusion criteria. 1045 patients were admitted to the intensive care unit; 312 skull fractures, 214 cervical fractures, 264 facial fractures, and 255 multiple fractures of the head and neck. Fracture categories were evenly distributed for ICU patients (skull: 29.9%, facial: 25.3%, cervical: 20.5%,and multiple: 24.4%), whereas 62% of non-ICU patients had isolated facial fractures. When potential confounding variables were accounted for, there were no differences in mortality based on race or insurance status. Blacks and Hispanics sustained proportionately more gun shot wounds (GSW) than Whites, 16X and 7X respectively. More male patients (76.5%) experienced head and neck fractures than females (23.5%), with the largest disparity for Blacks and Hispanics, 5:1 and 4:1, respectively. The most powerful predictors of outcome were shock on admission, Injury Severity Score (ISS) and mechanism of injury specifically GSW. **Conclusions:** These findings indicate demographic differences amongst races with regard to incidence. There were notable disparities in race, gender, injury pattern, but few statistically significant differences in outcome measures. Cause specific mortality was strongly related to age, GSW as a mechanism of injury, increasing ISS, and shock on admission. Our study adds to the literature by Millham et al 2009, by finding that shock, GCS, and ISS were the most powerful predictors of outcome across all analyses.3 Research needs to continue to investigate racial and other disparities in health care in order to elucidate targeted prevention strategies for equitable healthcare.

TMJ Arthrocentesis: 10 year Experience Outcomes

Matthew Steuer and Mehra, Pushkar
Department of Oral and Maxillofacial Surgery

Objective: Evaluate efficacy of TMJ arthrocentesis and assess differences in outcomes between performing the procedure under intravenous sedation versus general anesthesia. **Methods:** Review of records of all patients from 2000-2010 undergoing arthrocentesis of the TMJ by a single surgeon. All patients had preoperative MRI examinations and were assigned a Wilkes grade based on clinical and radiographic findings. Arthrocentesis was performed using a two-needle technique with concomitant jaw manipulation. Clinical examinations were performed at presurgery (T1), immediately post-surgery (T2), and longest follow-up (T3/LFU) intervals. Numerical analog scales were used for subjective evaluation of TMJ pain and headaches, jaw function, diet, and disability. Objective evaluation of maximum incisal opening (MIO), lateral excursions (LE), and presence of TMJ crepitus were recorded at the T1 and T3 visits. **Results:** 226 patients underwent a total of 407 procedures. Group 1 included 77 patients (57 bilateral) and Group 2 included 149 patients (124 bilateral). At LFU, all patients had statistically significant reduction in the incidence and severity of TMJ pain and headaches. Mean MIO increased post-surgery and the changes were statistically significant in the closed lock group. Lateral excursions increased minimally post-surgery. Significant improvements occurred in Dietary Restrictions, Jaw Function restrictions and Disability. 92 % of patients still had residual clicking/popping and joint noises postsurgically. **Conclusions:** Arthrocentesis is a very effective, minimally invasive technique for treatment of TMJ pain and acute closed lock conditions, irrespective of the grade of Wilkes classification. It is almost 100% effective in treating closed lock and provides significant reduction in pain levels. Up to 95% of patients report immediate resolution/reduction of pain within the first postoperative week. Approximately 32% of these patients require repeat procedures at an average of 18 months postoperatively (Range 6-27 months). The technique seems to have better results under general anesthesia as compared to intravenous sedation. Volume of saline lavage does not seem to be critical beyond 150 cc and jaw manipulation by itself appears to be very beneficial even when adequate saline lavage is not possible due to lack of adequate inflow-outflow ports *References: 1. Brennan, P; Arthrocentesis for Temporomandibular Joint Pain Dysfunction Syndrome . JOMS 64: 949-951; June 2006 2. Yura, S; Totsuka, Y: Relationship of effectiveness of arthrocentesis under sufficient pressure and conditions of TMJ. JOMS 63: 225-228; 2008*

A Comparison of Gliadin Degradation by Natural Colonizers of the Human Gastrointestinal Tract

Gabriel Blumenkranz Sanchez, Guoxian Wei, Eva Helmerhorst
Department of Periodontology and Oral Biology

Celiac disease (CD) is a lifelong inflammatory enteropathy of the small intestine. It only affects certain genetically predisposed individuals. The disease is triggered upon ingestion of wheat gluten and similar proteins found in other cereals such as barley and rye. Once diagnosed, celiac patients must adhere strictly to a gluten free diet since no true therapy is available. The ingested gluten, specifically the gliadin fractions, is highly resistant to degradation by mammalian gastrointestinal proteases. For this reason, external proteases that can cleave gliadins have been considered as potential therapeutic drugs, since they may cleave and neutralize gliadins into non-toxic fragments. One source of such proteases is Rothia species, which are natural colonizers of the oral cavity. **Methods:** In this study we compare a commercially available probiotic dietary supplement with Rothia mucilaginosa (OD620=1.2; 13.7 x107 CFU/ml) for their capability to cleave gliadins. Activities were assessed towards several substrates including tripeptide paranitroanilide-derivatized substrates, a gliadin-derived 33-mer peptide and mixed gliadins in gel and solution. **Results:** The results indicated that the oral bacterium is far superior in gliadin degradation than the commercial probiotic. **Conclusion:** The data suggest the potential of Rothia mucilaginosa to be exploited as a probiotic supplement in gluten-sensitivity and celiac disease. *Supported by NIAID grant AI087803.*

Maxillary and Mandibular Reconstruction Surgery with Bone-Tissue Engineering

Nathan Turley, Pushkar Mehra, David Cottrell and Laishing Chou
Department of Restorative Sciences/Biomaterials

Background of Study: "Tissue Engineering has the potential to offer practitioners the option to reconstruct tissues in-vitro, for use as grafts for replacement of damaged and diseased body parts. Besides eliminating donor site morbidity, this concept offers the ability to precisely fabricate customized patient-specific replacements. Successful application of the technique involves a controlled, coordinated, and timed triad of cells, signaling molecules, and scaffolds in an appropriate environment. We have previously demonstrated the capacity for bone formation using biomimetically designed scaffolds and human osteoblasts in a mice model within 8 weeks. The purpose of this prospective study was to test the effectiveness of this technique in human subjects. Materials and Methods: Implementation of our protocol was done with selected patients where harvest of conventional bone grafts were not an option due to systemic disorders. Patients underwent harvest of small autogenous bone core from the third molar area under local anesthesia (approximately 5 mm3). Osteoblast cell cultures were expanded in a laboratory and challenged with Vitamin D to confirm osteocalcin and alkaline phosphatase expression using immunohistochemical techniques, thereby verifying active cell lines for bone formation. Cells were grown for a total of 6-8 weeks in the laboratory before transplantation into the patients. Scaffolds were biomimetically designed with a combination of well-tested inorganic elements and PLGA through a melt-molding process. Our previous studies have demonstrated signaling molecule expression, in vivo, associated with this scaffold design. Results: Patient 1: A 26 year old male patient with osteopetrosis suffering from congenital maxillary hypodontia with associated bilateral maxillary alveolar hypoplasia, and a history of multiple long bone fractures. The patient underwent intraoral transplantation of engineered cells using "bead-shaped" implantable scaffolds to the deficient maxilla followed by delayed endosseous dental implant placement. Core biopsies were taken at 6 months postoperatively and histological examination revealed excellent lamellar bone formation. Six dental implants were placed into the reconstructed maxilla and all implants osseointegrated without complications. The patient was successfully rehabilitated with an implant-supported prosthesis. The patient was followed up for nine years postoperatively and no significant bone loss was noted in any of the implants. Patient 2: A 51 year old male patient with a genetically-associated unknown craniofacial syndrome presented with multiple mandible fractures that had occurred spontaneously during mastication. He gave a history of multiple long bone fractures, and had a severely atrophic mandible. He underwent extraoral augmentation/reconstruction of the atrophic mandible with tissue engineered cells interspersed on customized scaffolds fabricated through a wax-up on a stereolithographic 3-D model. CT scans were obtained postoperatively at 1,2 and 3 year intervals and they revelaed consolidation and maintenance of well-ossified bone. The patient was followed up for six years postoperatively and suffered no further mandible fractures. Conclusion: This report demonstrates successful application of a novel tissue engineering technique for hard tissue (bone) formation utilizing biomimetically designed scaffolds and autogenous cell lines. The technique allows for production of lamellar bone of excellent quality in a relatively short period of time (8 weeks) which is also optimal for successful osseointegration of endosseous dental implants and healing of bone defects.

Clinical Study Design for Investigating Salivary Protein Composition in Healthy and Celiac Patients

Andres Jimenez Wolf, Na Tian, Josh Hansen, Dan Leffler, Ciaren Kelly and Eva Helmerhorst
Department of Periodontology and Oral Biology

Celiac Disease is a gluten-sensitive enteropathy characterized by an inflammatory reaction in the small intestine triggered by gluten proteins. Clinical consequences include malabsorption of nutrients due to the loss of absorptive villi and crypt hyperplasia. Certain similarities are found between the primary structures of gluten and salivary proline-rich proteins. These structural similarities can give an interesting perspective to the manner salivary proteins can be regarded in the celiac disease mechanism. Salivary proteome analyses have revealed a great potential for the diagnosis of systemic diseases. The concept of saliva as a diagnostic fluid is alluring because it is easily collected in a non-invasive manner, and the number of potential applications is rapidly increasing. **Objective:** The main focus of our study is to compare saliva composition, with emphasis on proline-rich proteins, in samples collected from healthy and celiac patients. A multi-site clinical study was initiated, in collaboration with the Department of Medicine and Division of Gastroenterology at Beth Israel Deaconess Medical Center, Boston, MA. **Methods:** Healthy and celiac patients are being recruited, consented, and asked to fill out three questionnaires probing oral health, overall gastrointestinal health and symptoms characteristic of celiac disease. Of all patients 10 ml of stimulated whole saliva and parotid saliva are being collected as well as two buccal epithelial swab samples. Salivary flow rate is being monitored throughout collection. Collected whole saliva samples are aliquoted, centrifuged and separated into supernatant and pellet fractions. All samples are being stored at -80 C. This is an ongoing clinical study. Patients are actively being enrolled. So far, samples from 12 celiac patients have been collected and processed. Future biological analysis will include proteome composition determination by SDS-PAGE and RP-HPLC, enzyme activity analysis, salivary microbiome analysis, and exon sequencing of mammalian genes of interest. *Supported by NIAID Grant AI087803.*

Trypsinogen Isoforms in the Mouse Pancreas

Balazs Nemeth and Miklós Sahin-Tóth
Department of Molecular and Cell Biology

Background: Mutations that stimulate autoactivation of the digestive proenzyme cationic trypsinogen cause hereditary pancreatitis in humans. To gain insight into pathogenesis, mouse models that mimic the human the disease would be valuable. However, expression and activation of mouse trypsinogens are poorly characterized. **Aims:** Our aim was to identify which trypsinogen isoforms are expressed in the mouse pancreas; to characterize the autoactivation of mouse trypsinogens and study their interaction with chymotrypsin C, which regulates activation of human cationic trypsinogen. **Methods:** Trypsinogens were purified from the mouse pancreas by affinity and ion-exchange chromatography. Isoforms were identified by N-terminal sequencing and mass spectrometry. Isoforms T7, T8, T9, and T20 were expressed recombinantly and purified by affinity chromatography. Trypsinogen activation was studied by enzyme activity assays and SDS-PAGE. **Results:** The mouse pancreas expresses 4 trypsinogen isoforms to high levels, T7, T8, T9 and T20. Compared to human cationic trypsinogens, all mouse trypsinogens isoforms exhibited markedly slower autoactivation, particularly in 1 mM CaCl2 that is characteristic of pancreatic juice. Chymotrypsin C strongly inhibited autoactivation of T8 and T9 mouse trypsinogens. **Conclusions:** Mouse trypsinogens undergo autoactivation at a much slower rate than human cationic trypsinogen, which suggests that the mouse is not an appropriate model to recapitulate the high autoactivation rates observed with human pancreatitis-associated trypsinogen mutants. *Supported by NIH grants R01DK058088, R01DK082412 and R01DK082412-S2 (ARRA).*

The Chymotrypsin C-Sensitive Leu81-Glu82 PeptideB in Human Cationic Trypsin Exhibits Unusual Thermodynamic Stability

András Szabó and Miklós Sahin-Tóth
Department of Molecular and Cell Biology

Background and aim: The digestive enzyme chymotrypsin C (CTRC) promotes inactivation of human cationic trypsin by cleaving the Leu81-Glu82 peptide bond in the calcium-binding loop of trypsin. Our aim was to investigate the thermodynamic properties of the Leu81-Glu82 peptide bond hydrolysis reaction as preliminary observations suggested this peptide bond may exhibit unusually high stability. **Methods:** Recombinantly expressed CTRC and human cationic trypsinogen were purified to homogeneity using affinity chromatography. Trypsinogen was activated to trypsin with enteropeptidase. The trypsin constructs contained the S200A inactivating mutation, which eliminates the catalytic serine residue. Trypsin digestion by CTRC was followed by SDS-PAGE. Kinetic parameters of CTRC were determined using the Suc-Ala-Ala-Pro-Phe-pNA substrate. **Results:** We found that digestion of trypsin at Leu81 by CTRC did not proceed to completion but an equilibrium was attained, which in the absence of calcium contained 50% uncleaved and 50% cleaved trypsin forms. This unusual thermodynamic equilibrium was confirmed by demonstrating that the cleaved Leu81-Glu82 peptide bond was re-synthesized by CTRC to reach the same equilibrium as observed in the forward cleavage reaction. Mutagenesis of the calcium coordinating glutamic acid (Glu82 and Glu85) residues to alanine shifted the equilibrium toward full digestion, indicating that structural integrity of the calcium binding loop is essential for the high thermodynamic stability of the Leu81-Glu82 peptide bond. On the other hand, mutations of Glu79 to alanine or lysine further stabilized the peptide bond and shifted the equilibrium towards the uncleaved form. Finally, using kinetic analysis of competitive inhibition, we found that trypsin cleaved at Leu81 had higher affinity (Ki: 0.3 μM) towards CTRC than intact, uncleaved trypsin (Ki: 2.9 μM). **Conclusion:** The chymotrypsin C-sensitive Leu81-Glu82 peptide bond in the calcium binding loop of human cationic trypsin exhibits high thermodynamic stability; favoring the uncleaved form in the presence of calcium. Although this phenomenon seems extraordinary, it may indicate that thermodynamically stable peptide bonds may be more widespread than previously appreciated. *Supported by NIH grants R01DK058088, R01DK082412 and R01DK082412-S2 (ARRA).*

Hippo Signaling Pathway in Submandibular Gland Development: Potential Interplay with N-Glycosylation

Ray English, Meghan Bouchie and Maria Kukuruzinska
Department of Molecular and Cell Biology

Abstract: Mouse submandibular gland (SMG) begins its development at embryonic day 11 when oral epithelium grows into the underlying mesenchyme. The metabolic pathway of protein N-glycosylation is critical for SMG development and partial inhibition of DPAGT1, the gene that initiates N-glycosylation, drives cytodifferentiation of ductal structures. We have shown that DPAGT1 is a target of the canonical Wnt signaling pathway and that DPAGT1 regulates E-cadherin-mediated cell-cell adhesion, required for the survival of differentiating duct cells. Recently, the Hippo pathway has been shown to be critical for tissue development by regulating Wnt signaling and establishing apical-basal polarity. TAZ is a Hippo pathway transcription factor and polarity enhancer that also serves as a mechanosensor of the extracellular matrix. **Objective:** We investigated whether the Hippo pathway participated in SMG development and if it interacted with the metabolic pathway of N-glycosylation. **Methods:** SMGs were dissected from mice at embryonic day 13.5, epithelial rudiments were separated from the mesenchyme and grown in vitro in the presence and absence of siRNA to DPAGT1. After 48 hours, SMGs were processed for immunofluorescence staining for F-actin, ZO-1 and TAZ and analyzed by confocal microscopy. **Results:** Confocal imaging showed colocalization of F-actin and ZO-1 extending further into the ductal region in DPAGT1-silenced glands compared to controls. TAZ was detected at the apical-lateral surfaces of differentiating duct cells, suggesting its role in polarity. Moreover, in DPAGT1 silenced glands, TAZ exhibited enhanced localization to intercellular junctions. **Conclusion**: Our findings show that inhibition of N-glycosylation promotes establishment of apical domains in ductal progenitors by ZO-1. In addition, the Hippo pathway component, TAZ, is expressed in the embryonic SMG, localizing to intercellular junctions in ductal cells, which is enhanced by partial inhibition of DPAGT1. These data suggest an inverse relationship between N-glycosylation and the Hippo pathway. *Supported by NIH grant RO1DE014437.*

Effect of Oral Health Promotion Provided by Public Health Nurses on the Behaviors of Mothers with Infants

Marc Horton, Ayesha Ghulam, Sharron Rich, Kathy Lituri, Patricia Whitworth, Heavenly Mitchell, Sheree Norquist, Michelle Henshaw
Health Policy and Health Services Research, Boston University Henry M.Goldman School of Dental Medicine, 560 Harrison Avenue, Healthy Baby/ Healthy Child, Boston Public Health Commission, 1010 Massachusetts Avenue, Boston.

The Boston Public Health Commission's Healthy Baby/Healthy Child Program (HBHC) is a perinatal home-visiting program that targets women at high risk for adverse birth outcomes. The purpose of this study was to explore the feasibility of incorporating a patient-centered oral health promotion model, delivered by home-visiting nurses, into HBHC and to assess the program's impact on participant's risk factors for early childhood caries (ECC). **Methods:** All nurses were trained in patient-centered counseling and oral health. For participating families, nurses incorporated oral health visits into their home visiting schedule. After each visit a dental hygienist conducted a follow-up oral health assessment and administered a questionnaire. Data was obtained from 18 mother-child controls and from 37 mother-child pairs who received home-based oral health counseling provided by the HBHC nurses. Chi-square tests and t-tests were performed. The participants were Black (59.5%), Hispanic (27.0%), Medicaid-eligible (70.3%), and 21.6% did not graduate high school. **Results:** The results indicated an increase in the percentage of mothers who reported wiping their child's gums or brushing their teeth (55.6% of controls; 86.5% at the first assessment; 100.0% at the third assessment; $p < 0.05$ for both) and in the percentage who reported that their child had a dentist (0.0% of controls; 35.1% at the third assessment; $p < 0.05$). **Conclusion:** These findings support the use of patient-centered counseling, provided by home visiting nurses, as an effective approach to prevent ECC among those most at risk and least likely to receive dental care. More research should be conducted in this promising area.

The Use of Vwc2 Protein as a Novel Approach to Induce Bone Formation

Ahmed Almehmadi1 ,Yoshio Ohyama1,Haytham Jaha1,Sundharamani Venkitapathi1,Reem Aljamaan1,Masaru Kaku2,Yoshiyuki Mochida1 1 Department of Periodontology and Oral Biology, Boston University, Henry M. Goldman School of Dental Medicine, USA 2 Niigata University Graduate School of Medical and Dental Sciences, Division of Bio-Prosthodontics, JAPAN

Objective: We have recently reported that a novel secretary protein, von Willebrand domain containing 2 like (Vwc2l), promotes osteoblast mineralization in vitro. In the present study, Vwc2, a highly homologous protein to Vwc2l was characterized. The objective of our study is to investigate the effect of Vwc2 on bone formation. **Methods:** The expression of Vwc2 in MC3T3-E1 (MC) osteoblastic cell line was investigated by real time PCR at RNA level and by Western Blotting (WB) analysis at protein level. The presence of Vwc2 protein in bone was also examined by immunohistochemistry (IHC) and WB analysis. To characterize the function of Vwc2 protein,recombinant Vwc2 protein (rVwc2) was added into mouse calvaria ex vivo cultures and the extent of newly synthesized bone formation was analysed. **Results:** Vwc2 transcript is detected in MC cells with the highest expression observed at day 7 during biomineralization. The expression pattern of Vwc2 protein in the cultured media from MC cells was similar to that of Vwc2 transcript. The localization of Vwc2 protein was observed by IHC in mouse developing maxilla. The effect of rVwc2 on mouse calvaria bone formation was examined and the results showed increased bone formation. **Conclusion:** Our data demonstrated that Vwc2 is expressed in osteoblasts/bone and has the ability to increase bone formation. The data obtained from this study may provide insights to the biological functions of this novel protein and help to develop a new molecular design for bone loss therapies. *Supported by NIH grant DE019527,AR057451 and Boston University School of Dental Medicine.*

Microarray Based Characterization Of Early In Vivo Acquired Enamel Pellicle Colonizers

Debora Heller, Eva Helmerhorst, Bruce Paster, and Frank Oppenheim
Department of Periodontology and Oral Biology

The salivary protein derived acquired enamel pellicle (AEP) is the substrate for the earliest phase of bacterial attachment. The elucidation of this process is critical for understanding the pathogenesis of oral diseases. The characterization of this phase of early biofilm formation has become amenable to a broader investigation with the advent of the Human Oral Microbe Identification Microarray (HOMIM). **Objectives:** To obtain insight into the sequential pattern of AEP colonization. **Methods:** Samples of tooth integuments were collected from 12 healthy individuals at 0, 2, 4 or 6 hours after thorough removal of plaque and pellicle from buccal tooth surfaces. The HOMIM approach was used to analyze the samples for the presence of over 300 species. Statistical significance was assessed using Paired Wilcoxon Signed-Rank Test and Spearman Correlation Coefficient with a significance level of $p<0.05$. **Results:** Of the possible 422 target probes, 125 were detected in the samples. Species or phylotypes identified were as follows: Streptococcus anginosus or S. intermedius, Streptococcus oralis, Streptococcus sanguinis, Streptococcus Cluster II-III and Actinomyces spp. were predominant at 0 hr. Capnocytophaga sputigena, Campylobacter gracilis, Kingella oralis and Gemella haemolysans were more prominent after 2 to 4 hours. Significant increases in colonization over time were noted for Gemella haemolysans ($p=0.0005$), Streptococcus anginosus or S. intermedius ($p=0.03$), Streptococcus mitis bv2 ($p=0.004$), Streptococcus oralis ($p=0.0003$) and Streptococcus Cluster I ($p=0.0092$). **Conclusion:** Species of Streptococcus and Actinomyces were the early colonizers of AEP. However after 6 h of colonization, species of Gemella, Granulicatella, Prevotella, Haemophilus and additional species of Streptococcus were predominant. These in vivo data reveal detailed and characteristic pattern of the early phases of dental biofilm formation important for plaque maturation and pathogenesis of disease. *Supported by NIDCR grants DE05672 and DE07652.*

Bond Strength Between Veneer Porcelains and CAD/CAM Ceramic to Titanium

Nedda Hifeda, Russell Giordano, Richard Pober
Restorative Sciences and Biomaterials Department- Goldman School of Dental Medicine- Boston University

Objectives: The aim of this study was to evaluate the bond strength of three different veneering porcelains fired on CP Titanium and a CAD/CAM ceramic cemented to CP Titanium. **Methods:** sixty-four bars of CP Titanium grade II (25x 3x 0.5 mm) were divided randomly into four groups: Group 1 Titankeramic (Vita Zahnfabrik), 2 Triceram (Dentaurum), 3 Initial Ti (GC), and Group 4 MKII blocks (Vita). Each veneer porcelain (groups 1-3) was applied with dimensions limited to 8x3x1mm and fired on CP Ti bars following the manufacture's instructions and ISO 9693 recommendations. Group 4, Vita Mark II blocks were sectioned into sixteen bars (8x3x1mm) and cemented with Multilink Implant cement (Ivoclar) on CP Ti bars. Fifteen Specimens from each group were tested for bond strength by Schwickerath crack initiation test (ISO/DIS 9693) using an Instron machine; the mode of failure and bond interface was evaluated by SEM/EDS, N =15. **Results:** Bond Strength of Veneer Porcelain and MKII to CP Ti. Group Bond Strength (MPa) Significant Difference Group 1: Titankeramik/CP Ti 22.3 ± 8.5 A Group 2: Triceram/CP Ti 16.6 ± 9.9 AB Group 3: Initial Ti/CP Ti 10.0 ± 6.9 B Group 4: Mark II CP Ti 59.0 ± 17.7 C The data was analyzed statistically using ANOVA and Tukey at $p< 0.5$. Groups with the same letter are not significantly different. Group 4 is significantly higher than the other groups. Group 1 is significantly higher than group 3, but similar to group 2. SEM/EDS analysis reveals that the mode of failure for groups 1 - 3 are a mixture of adhesive, mixed, and cohesive failures. **Conclusion:** Cementing a milled ceramic to CP Ti produces bond strength significantly greater than conventional veneering porcelains.

Novel Insights into Diabetic Bone Complications

Roozbeh Khosravi and Philip Trackman
Department of Periodontology and Oral Biology

Diabetes doubles the risk of bone fracture. Organic and inorganic components of bone matrix determine bone strength, and poor quality of bone in diabetes is independent of bone mineral density. Studies indicated that in diabetes, glycation of collagen, the most abundant protein in bone matrix, prompts abnormal arrangement of collagen molecules leading to fragile bones. Moreover, diabetic bone osteopenia is attributed to lower enzymatic collagen cross-links. What remains unknown is whether diabetes down regulates lysyl oxidase (LOX), which is made by bone forming cells (osteoblasts), consequently reducing enzymatic collagen cross-links. **Methods:** We used primary calvarial rodent osteoblasts to examine collagen and glycated collagen regulation of LOX. Our findings indicate that collagen up-regulates LOX in osteoblasts, while glycated collagen fails to induce LOX. To determine the mechanism of collagen up-regulation of LOX, we investigated roles for collagen receptors, namely integrins and Discoidin Domain-Receptor 2 (DDR2). Inhibitor and knockdown studies suggest that collagen up-regulates LOX through DDR2, and independent of integrins. Additionally, we assessed diabetes regulation of LOX in a mouse calvarial bone-healing model. **Results:** Our in vivo experiments show that diabetes up-regulates LOX mRNA, protein and enzyme activity in partially healed bone (day 7). To further examine factors resulting in up-regulation of LOX in diabetes, we performed histological analysis. Our observations after 7 and 14 days of bone healing suggest that hematomas, which form in the initial stage of bone healing, do not resolve in diabetic mice. **Conclusion:** Because hematomas are a rich source of growth factors, we suspect that unresolved hematomas in diabetic healing bone may promote cell proliferation at the expense of osteoblast differentiation and mineralization. *Supported by NIH/NIDCR Grant RO1DE14066.*

Maxillofacial Gunshot Injuries at an Urban Level I Trauma Center - 10-year Analysis

Olena Norris and Pushkar Mehra
Department of Oral and Maxillofacial Surgery

Purpose: Optimal management of facial gunshot trauma remains controversial in terms of timing and reconstruction techniques. To analyze current trends in surgical management, a 10-year retrospective study of patients admitted with facial gunshot wounds was undertaken. Data with respect to length of hospitalization, patient demographics, treatment cost and payments to hospital were reviewed to evaluate treatment management and socio-economic disparity. **Materials:** Retrospective analysis of facial gunshot injuries in patients treated at Boston Medical Center, by the Department of Oral and Maxillofacial Surgery, from 2001 to 2011. Data was obtained from the institutional trauma registry and hospital records. **Results:** During the study period, there were total of 1957 patients admitted with gunshot wounds to Boston Medical Center, with 136 (6.9%) involving the facial region. 55 patients met inclusion criteria and were selected for the study. Age ranged from 16 to 61 years, with mean age being 25 years for men and 34 for women. 48/55 (87%) were males and 7/55 (13%) were females. The most common injury was to neck zone III; mandible fractures were encountered in 26 (47%) patients. Fractures were treated within 72 hours from admission for the majority of patients. 12 (22%) patients returned for secondary treatment. 20% patients had associated neurological injuries 9% had cervical spine fractures. Angiography was performed in 33 (60%) patients with 7 (13%) requiring embolization. 38 (70%) patients required airway management. Overall mortality was 9%, and most cases were associated with brain injury or severe bleeding from chest or abdominal injuries; no death occurred from isolated facial gunshot injuries. 18% of patients had private third party medical insurance, 45% had public insurance, and 23% had no insurance. Estimated hospital profit (not including the physician charges) was 11% from treating patients with private insurances. In contrast, there was a loss of 50% while treating patients with public insurance; and a 100% loss when treating uninsured patients. Conclusions: Airway compromise was the most life-threatening early problems; requiring establishment of definitive airway upon assessment. Brain, vascular and cervical spine injuries were common and warranted further investigation. Patients admitted with higher stages of shock and lower mental status, due to brain, vascular and/or spinal cord injuries: correlated with prolonged hospitalization, increased treatment costs and extended rehabilitation. We advocated early intervention (less than 72 hours), conservative approach, one-stage reconstruction of all involved bony and soft tissue injuries. In our study, African Americans were more frequently injured as compared to other ethnic groups; this was due to the geographic location of our hospital and mission to serve the underprivileged/ low income patients. The majority of the facial gunshot injuries were not presented as life threatening; but typically resulted in significant morbidity. With respect to the cost of healthcare, the vast majority of patients relied on public aid and had no insurance; in all cases the cost of care was more than the reimbursement provided.

Rare Cationic Trypsinogen Mutations Found in Subject with Pancreatitis are Harmless Variants

Andrea Schnur and Miklós Sahin-Tóth
Department of Molecular and Cell Biology

Introduction: Mutations in the PRSS1 gene encoding human cationic trypsinogen cause hereditary pancreatitis. Disease-associated trypsinogen mutations increase activation or impair degradation and thereby lead to the development of intrapancreatic trypsin activity. Recently, numerous novel rare PRSS1 mutations with unknown clinical significance were identified in subjects with idiopathic chronic pancreatitis. Despite lack of evidence, some of these variants have been described as pancreatitis-associated. Objectives: The aim of this study was to characterize published novel trypsinogen variants functionally, in order to judge their possible pathogenic impact. **Methods:** Wild type and 9 mutant trypsinogens were expressed recombinantly and purified. Trypsinogen activation, trypsinogen/trypsin degradation by chymotrypsin C and enzyme kinetic parameters were studied by activity assays and gel electrophoresis. Cellular expression of trypsinogens was assessed by SDS-PAGE and trypsin activity assays of conditioned media from transfected HEK 293T cells. **Results:** None of the investigated mutants exhibited increased activation or impaired degradation; the gain of function phenotypes typical of disease-associated mutations. Surprisingly, 6 of 9 mutants showed loss of function either due to reduced secretion and/or increased degradation by chymotrypsin C. **Conclusions:** Rare cationic trypsinogen mutations found in subjects with chronic pancreatitis are harmless variants, most likely not associated with the disease. The loss-of-function trypsinogen variants may even have a protective effect. These results emphasize that classification of novel PRSS1 variants should be based on functional evidence. *Supported by NIH grants R01DK058088, R01DK082412 and R01DK082412-S2 (ARRA).*

Characterization of the Functions of Evc and Evc2 Proteins in Ellis -Van Creveld Syndrome

Sundharamani Venkitapathi, Yoshio ohyama, Haytham Jaha, Ahmad Almehmadi, Reem Aljaman and Yoshiyuki Mochida
Department of Periodontology and Oral Biology

Introduction: Ellis van Creveld syndrome (EVC) is an autosomal recessive skeletal dysplasia, with inter- and intra-familial variability, characterized by short ribs, short limbs, postaxial polydactyly, hyperplastic frena with shallow labial sulcus, peg shaped teeth, delayed eruption of teeth and dysplastic nails. Mutations in either of the genes EVC or EVC2 leads to the manifestation of the same clinical phenotype identified as EVC syndrome, though there is no significant sequence homology between them at the DNA or protein level. Reports suggest that Evc knockout mice show clinical phenotype similar to patients with EVC syndrome. Evc2 knockout mice model developed in our lab also has features demonstrating phenotypes similar to EVC syndrome.Objectives:Ellis van Creveld syndrome now belongs to a group of disorders classified as ciliopathies. Reports from literature suggest that Evc and Evc2 proteins localize to cilia. To characterize the biological functions of Evc and Evc2 proteins, the objective of our study is to examine the localization of these proteins and to decipher their roles in Hedgehog signaling. **Methods:** The gene expression (EVC) was analyzed using several mouse tissues and mouse cell lines by real time PCR. The localization of Evc and Evc2 proteins in Imcd3 cells were investigated using anti-Evc and anti-Evc2 proteins by immunofluorescent staining. Binding assay of Evc and Evc2 proteins were performed using Imcd3 cells. The role of Evc and Evc2 proteins in Hedgehog signaling was studied by gain-of-function method and Western blot was performed using anti-Xpress antibody. **Results:** We observed that Evc and Evc2 proteins localize to cilia and the binding assay suggests that Evc and Evc2 proteins interact with each other. The IP-WB analysis performed using cell lysates from 293 cells suggests that they also have a role in processing transcription factor Gli3 protein. **Conclusion:** We observe that Evc and Evc2 proteins have a role in modulating Hedgehog signaling pathway by regulating the processing of transcription factor Gli3 protein.

Functional Defects Caused by Chymotrypsin C (CTRC) Mutations in Chronic Pancreatitis

Sebastian Beer and Miklós Sahin-Tóth
Department of Periodontology and Oral Biology

Background and Aims: Chronic pancreatitis is a progressive inflammatory disorder of the pancreas. Inappropriate activation of the digestive proenzyme trypsinogen to trypsin in the pancreas has a central role in the disease mechanism. The digestive enzyme chymotrypsin C (CTRC) degrades trypsinogen and trypsin and thereby reduces trypsin activity. Clinically frequent CTRC mutations A73T, R254W, and K247_R254del cause loss of CTRC activity or secretion and increase the risk for chronic pancreatitis. A large number of rare CTRC variants have been found in patients with chronic pancreatitis, however, the functional and clinical significance of these remains unknown. To classify CTRC mutations according to their phenotype, we analyzed secretion and activity of 32 missense CTRC mutations. **Methods:** CTRC secretion from transiently transfected HEK 293T cells was measured using enzyme activity assays and SDS-PAGE. Mutants with reduced activity were purified and their ability to cleave a small peptide substrate and to degrade trypsinogen was characterized. Additionally, CTRC degradation by trypsin was investigated. **Results:** Seventeen of 32 mutants showed normal or nearly normal secretion and activity, whereas 15 mutants were functionally deficient in one or more aspects: secretion of 3 mutants was below detectable levels and 3 other mutants showed significantly reduced secretion. Four mutants had severely diminished activity (<5%), but were normally secreted (60-90%). Two mutants exhibited moderately impaired activity. Rapid degradation by trypsin was found in 3 mutants and 2 mutants were degraded at a slower rate. **Conclusion:** Although only three CTRC mutations are statistically associated with chronic pancreatitis, here we demonstrate that 12 additional mutations cause considerable functional impairment and therefore are likely to be pathogenic. We identified three distinct, but mutually non-exclusive loss of function mechanisms: secretion defect, catalytic deficit and degradation by trypsin. This phenotypic dataset may aid in the classification of the functional relevance of CTRC mutations identified in patients with chronic pancreatitis. *Supported by NIH grants R01DK058088, R01DK082412 and R01DK082412-S2 (ARRA).*

Efficacy of Anti-Inflammatory Drugs in Third Molar Surgery Labaratory and Clinical Correlation

Mohammed Nadershah and Pushkar Mehra
Department of Oral and Maxillofacial Surgery

Purpose: This is a double-blind randomized clinical trial to assess the effect of four commonly used pharmacological regimens on the level of prostaglandin E2 (PGE2) in urine and saliva and correlate the findings to the clinical postoperative course after removal of impacted lower third molars. **Patients and methods:** Eighty ASA 1 patients were randomly selected in this study. The inclusion criteria included: 1) Bilateral full-bony impaction of lower third molars;1) Between 18 to 30 years of age; 2) No systemic disease; 3) Taking no medications; 4) No allergies to any of the study drugs; and 5) Absence of local or systemic infection. All procedures were done by a senior oral surgery resident using local anesthesia and intravenous ambulatory general anesthesia using a combination of fentanyl, valium, and methohexital. Patients were randomly divided into the following four groups (20 patients per group): Group 1: Received immediate pre-operative placebo tablet, intra-operative Dexamethasone (8mg IV), and a placebo post-operative tablets every 6 hours for a week.; Group 2: Received immediate pre-operative placebo tablet, intra-operative normal saline (2cc IV), and a post-operative placebo tablets every 6 hours for a week; Group 3: Received immediate pre-operative Ibuprofen 600mg, which was continued every 6 hours post-operatively for a week in addition to intra-operative normal saline (2cc IV); and, Group 4: Received immediate pre-operative Ibuprofen 600mg, which was continued every 6 hours post-operatively for a week in addition to intra-operative Dexamethasone (8mg IV). All groups received 30 mg codeine tablets every 4 hours as needed for postoperative pain management. Clinical examination included: 1) Subjective evaluation using VAS scales for: Pain, Loss of Jaw Function, Swelling, Diet Restriction, and General Wellness; and 2) Objective evaluation of: Maximum interincisors opening (MIO) and lateral excursions (LE), presence or absence of any TMJ symptoms, and muscle tenderness. A note was also made of the number of codeine tablets consumed since last visit, and for the presence of dry socket or infection. Saliva and urine samples were taken from each patient pre-operatively and post-operatively at scheduled intervals (24 hours, 48 hours, 72 hours, and 168 hours). A multivariate analysis of variance was used to determine the statistical significance of the results between groups. A p-value less than 0.05 was considered to be statistically significant. **Results:** There were no significant differences in the results between the two sexes for any of the clinical parameters assessed in this study. Patients receiving anti-inflammatory therapy fared significantly better in most parameters, especially in the 3-day postsurgical period. A single dose of intravenous dexamethasone alone had a potent but transient beneficial effect when compared to the results with round-the clock non-steroidal oral anti-inflammatory medication (NSAIDS) which showed significant improvement in both subjective patient comfort as well as objective parameters related to jaw function throughout the postoperative course. **Conclusions:** There is a clear correlation between systemic prostaglandin levels and clinical symptomatology following third molar surgery. There seems to be distinct advantages of routinely using drugs to help pharmacologically reduce inflammation, and thus promote patient comfort after minor oral surgery. Preoperative NSAIDS before and after third molar surgery seem to be clearly beneficial. Use of a single dose of intravenous steroids perioperatively also helps suppress inflammation and reduce untoward sequelae, although to a lesser degree and for a shorter duration than continuous oral NSAIDS. Combining ibuprofen with perioperative intravenous dexamethasone added some benefit in some of the measured parameters but without a statistically significant advantage over using NSAIDS only.

A Potential Novel Pathogenetic Mechanism of Chronic Pancreatitis in Chymotrypsin C (CTRC) Mutants

Jiayi Zhou, and Miklos Sahin-Tóth
Department of Molecular and Cell Biology

Mutations in the chymotrypsinogen C (CTRC) gene have been identified as risk factors for chronic pancreatitis, but detailed pathogenetic mechanisms remain largely unknown. Under physiological conditions, premature active trypsin is inhibited by trypsin inhibitor SPINK1 in the pancreas. **Aims:** To examine whether the protective mechanism by SPINK1 is impaired following expression of CTRC mutants, and how SPINK1 is correlated with endoplasmic reticulum (ER) stress, which we have identified in CTRC mutants. **Methods:** 1. Dexamethasone-differentiated AR42J rat pancreatic acinar cells were infected with recombinant adenovirus carrying wild-type CTRC or mutants p.Q48R, p.G61R, p.A73T, p.R254W, and p.K247_R254del. CTRC secretion was measured by activity assays and SDS-PAGE. Messenger RNA levels of trypsin inhibitors SPINK1 and SPINK3 (rat homologs of human SPINK1), together with mRNA of ER chaperones BiP and calreticulin, were quantified by real time PCR. Splicing of XBP1 (XBP1s) was detected by PCR and agarose gel electrophoresis. 2. Messenger RNA levels of SPINK1 and SPINK3 were further measured following adenoviral expression of ER stress transcription factors ATF4, XBP1s and active form of ATF6 (ATF6a) in AR42J cells. 3. Conditional A73T CTRC mutant transgenic mice were generated, and mRNA level of SPINK3 (mouse homolog of human SPINK1) and XBP1 splicing were tested in primary acinar culture of the mice in the same way as described above. **Results:** 1. CTRC mutants p.Q48R, p.G61R and p.A73T were poorly secreted from AR42J cells and caused a significant downregulation of both SPINK1 and SPINK3 (bigger fold change than SPINK1) and an increase in ER stress markers including BiP and calreticulin mRNA levels and XBP1 splicing. The magnitudes of SPINK1 and SPINK3 seemed to correlate with the extent of the secretion defect and ER stress (p.G61R>p.A73T>p.Q48R). 2. Adenoviral expression of XBP1s caused a significant decrease of mRNA of both SPINK1 and SPINK3 in AR42J cells, and expression of ATF6a caused a decrease of mRNA of SPINK3, but not of SPINK1. 3. In primary acinar culture from A73T CTRC mutant transgenic mice, increased XBP1s was detected and the mRNA level of SPINK3 was significantly downregulated. **Conclusions:** 1. Downregulation of SPINK1 (SPINK1 and SPINK3 in rats, SPINK3 in mice) seems to be associated with CTRC mutants that exhibit a secretion defect and ER stress (p.Q48R, p.G61R and p.A73T). 2. Downregulation of SPINK1 seems to be an effect downstream of ER stress. 3. Downregulated expression of SPINK1 may impair its protective role, and provide a novel pathogenetic mechanism of chronic pancreatitis in certain CTRC mutants. *Supported by NIH grants R01DK058088, R01DK082412 and R01DK082412-S2 (ARRA).*

Boston University Henry M. Goldman
School of Dental Medicine

BOSTON UNIVERSITY

Science Day 2012 Awards Luncheon

TUESDAY, APRIL 10 in HIEBERT LOUNGE

Presented by Dean Jeffrey W. Hutter and Associate Dean for Research Maria Kukuruzinska

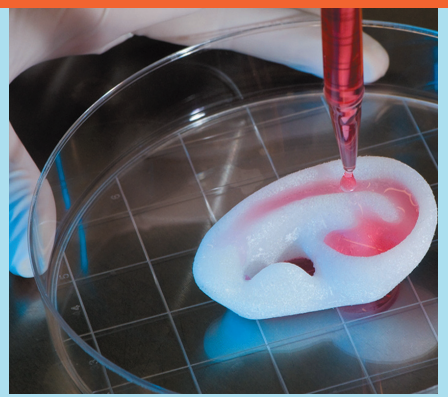


Image courtesy of Dr. Anthony Atala, director of the Wake Forest Institute for Regenerative Medicine, the W.H. Boyce Professor and Chair of the Department of Urology at Wake Forest University, and Science Day 2012 keynote speaker.

PROGRAM

WELCOME

Maria Kukuruzinska
Associate Dean for Research
Professor of Molecular and Cell Biology

Jeffrey W. Hutter
Dean and Spencer N. Frankl Professor in Dental Medicine

GSDM SCIENCE DAY 2012 AWARDS

Pre-doctoral Student, Poster Presentation
Sultan Muhammad, Gangli Liu, Pritam Sengupta, Basem Jamal, Meghan Bouchie and Maria Kukuruzinska. Department of Molecular & Cell Biology: "Upregulation of Cthrc1 N-glycoprotein Marks OSCC Tumor Spread."

Post-doctoral Student, Poster Presentation
Lea El Hachem, Eva Helmerhorst and Frank Oppenheim. Department of Periodontology & Oral Biology: "Histatin 5 Antifungal Activity Towards C. Albicans and C. Glabrata."

Post-doctoral Fellow, Poster Presentation
Balazs Nemeth, Miklós Sahin-Tóth. Department of Molecular & Cell Biology: "Trypsinogen Isoforms in the Mouse Pancreas."

Pre-doctoral Student, Oral Presentation
Ray English, Meghan Bouchie and Maria Kukuruzinska. Department of Molecular & Cell Biology: "Hippo Signaling Pathway in Submandibular Gland Development: Potential Interplay with N-glycosylation."

Post-doctoral Student, Oral Presentation
Debora Heller, Eva Helmerhorst, Bruce Paster and Frank Oppenheim. Department of Periodontology & Oral Biology: "Microarray Based Characterization of Early In Vivo Acquired Enamel Pellicle Colonizers."

Post-doctoral Fellow Oral Presentation
Sebastian Beer and Miklós Sahin-Tóth. Department of Molecular & Cell Biology: "Functional Defects Caused by Chymotrypsin C (CTRC) Mutations in Chronic Pancreatitis."

ADA/Dentsply Award
Nathan Ng, Elizabeth Krall Kaye and Raul Garcia. Department of Health Policy & Health Services Research: "Coffee Consumption and Periodontal Disease in Men."

ACKNOWLEDGEMENT OF OTHER AWARD WINNERS

Science & Engineering Research Symposium 2012
Dean's Award
Ahmed AlMehmadi, Yoshio Ohyama, Haytham Jaha, Sundharamani Venkitapathi, Reem Aljamaan, Masaru Kaku and Yoshiyuki Mochida. Department of Periodontology & Oral Biology: "The Use of VWC2 Protein as a Novel Approach to Induce Bone Formation."

YDC37 Post-doctoral Student 2012 Winner
Manuel Posada, Gurkan Goktug, Hideo Yamamoto. Department of Restorative Sciences/Biomaterials: "An Aesthetic Comparison of Different CAD/CAM Custom Abutment Materials on the Gingival Color: A Clinical Report."

Medical Research Scholars Program
Taylor Nicholas Snider, DMD'12 was selected to this new NIH launched program.

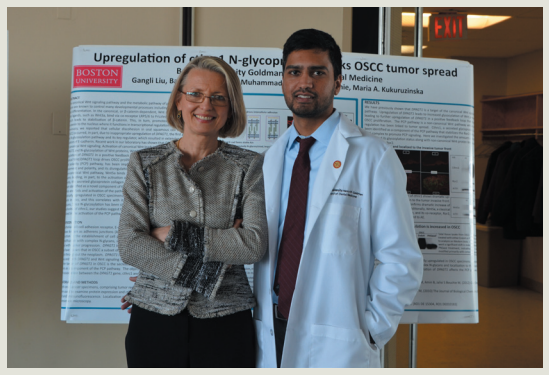
Hinman Student Research Symposium
Most Outstanding Presentation in Clinical Research Winner
Sultan Muhammad, Gangli Liu, Basem Jamal, Meghan Bouchie and Maria Kukuruzinska. Department of Molecular & Cell Biology: "Upregulation of Cthrc1 N-glycoprotein Marks OSCC Tumor Spread."

Congratulations!

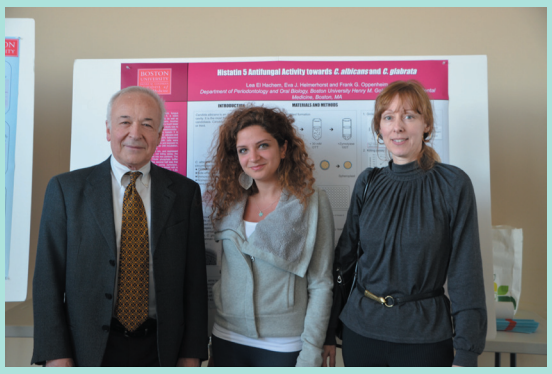
Boston University Henry M. Goldman School of Dental Medicine hosted Science Day 2012 featuring keynote speaker Dr. Anthony Atala March 22 and the Science Day Awards Luncheon April 10.

Dr. Anthony Atala, Associate Dean for Research Maria Kukuruzinska, and Dean Jeffrey W. Hutter

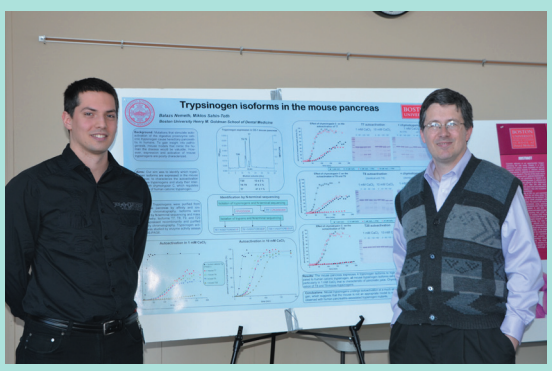
Pre-doctoral Student Poster Presentation winner Sultan Muhammad with Associate Dean for Research Maria Kukuruzinska



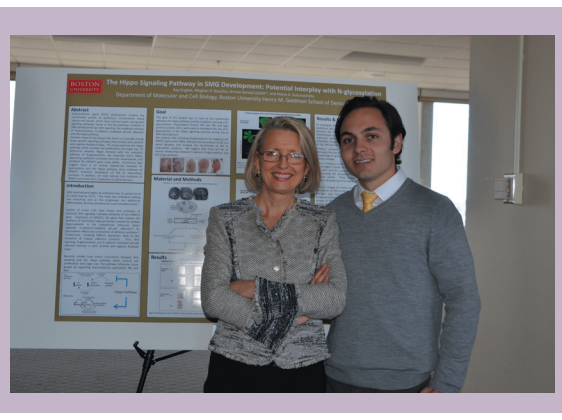
Post-doctoral Student Poster Presentation winner Lea El Hachem with Drs. Frank Oppenheim and Eva Helmerhorst



Post-doctoral Fellow Poster Presentation winner Balazs Nemeth with Dr. Miklós Sahin-Tóth



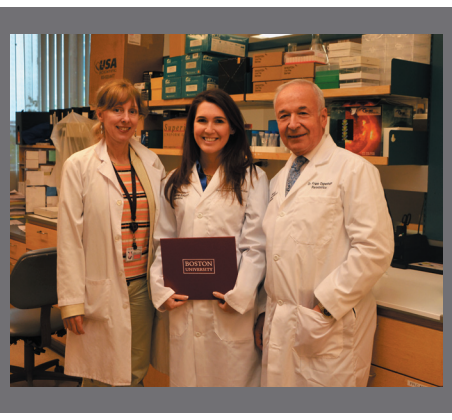
Pre-doctoral Student Oral Presentation winner Ray English with Associate Dean for Research Maria Kukuruzinska



Post-doctoral Fellow Oral Presentation winner Sebastian Beer with Dean Hutter

ADA/Dentsply Award winner Nathan Ng with Dean Hutter and Dentsply representative Joel Montero

Post-doctoral Student Oral Presentation winner Debora Heller with Drs. Eva Helmerhorst and Frank Oppenheim



Boston University Henry M. Goldman School of Dental Medicine

BOSTON UNIVERSITY