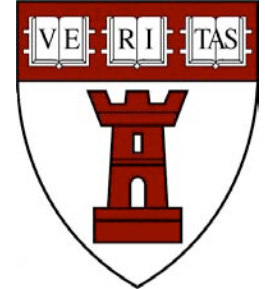


HSDM OFFICE OF RESEARCH BULLETIN



September 1, 2004

Who's Who In Research? The following research investigators will be moving their laboratories into the new building this October.



SHIGEMI NAGAI, PHD, DDS

Instructor of Restorative Dentistry and Biomaterials Sciences

Currently at 188 Longwood Avenue Laboratory

Dr. Nagai is an Instructor in Restorative Dentistry and Biomaterials Sciences as well as a certified Prosthodontic Specialist. Dr. Nagai is active in several research projects focusing on the color science in dentistry related to dental biomaterials and tooth color esthetics and will have bench space on the 3rd floor in the new building.



ARKHAT ABZHANOV, PHD

Instructor of Oral and Developmental Biology

Currently at the HMS NRB, Tabin Laboratory

Dr. Abzhanov is a 2004 Dean's Scholar recipient and will be moving to our new building from the Tabin Lab this October. Dr. Abzhanov's research interest focuses on craniofacial development and evolution. He also studies the differentiation of the cranial neural crest cells into chondrocytes and osteocytes on the cellular and tissue level.



MALCOLM WHITMAN, PHD

Associate Professor of Oral and Developmental Biology

Currently at LHRRB Cell Biology Laboratory

Dr. Whitman's research interest focuses on polypeptide growth factors and how they act to specify cell fate during the earliest stages of vertebrate embryogenesis. Dr. Whitman's lab uses the frog embryo as a model system to characterize both the roles of known signaling pathways in the specification of early embryonic mesoderm and to identify novel signal transduction pathways.



RECENT PUBLICATIONS & AWARDS:

If we have not captured your recent publications, please email Dawn DeCosta at dawn_decosta@hsdm.harvard.edu. You can be assured they will be listed in the next Bulletin.

Ahmed AR. Treatment of autoimmune mucocutaneous blistering diseases with intravenous immunoglobulin therapy. *Expert Opinions Investigational Drugs* 2004 Aug;13(8):1019-32.

Chen T, Hosogi Y, Nishikawa K, Abbey K, Fleischmann RD, Walling J, Duncan MJ. Comparative Whole-Genome Analysis of Virulent and Avirulent Strains of *Porphyromonas gingivalis*. *Journal of Bacteriology* 2004 Aug;186(16):5473-9.

Howell TH, Karimbux NY. Academy: strengthening the educational mission in academic health centers. *Journal of Dental Education* 2004 Aug;68(8):845-50.

Li Q, Olsen BR. Increased Angiogenic Response in Aortic Explants of Collagen XVIII/Endostatin-Null Mice. *American Journal of Pathology* 2004 Aug;165(2):415-24.

Merchant AT, Pitiphat W, Parker J, Joshipura K, Kellerman M, Douglass CW. Can nonstandardized bitewing radiographs be used to assess the presence of alveolar bone loss in epidemiologic studies? *Community Dentistry and Oral Epidemiology* 2004 Aug;32(4):271-6.

Moulton K, Olsen BR, Sonn S, Fukai N, Zurakowski D, Zeng X. Loss of Collagen XVIII Enhances Neovascularization and Vascular Permeability in Atherosclerosis. *Circulation* 2004 Aug.

Okamatsu Y, Kim D, Battaglino R, Sasaki H, Spate U, Stashenko P. MIP-1 gamma promotes receptor activator of NF-kappa B ligand-induced osteoclast formation and survival. *Journal of Immunology* 2004 Aug 1;173(3):2084-90.

Congratulations to **NACHUM SAMET, DMD**, Instructor in Restorative Dentistry and Biomaterials Sciences for his recent grant from Medical Implant System, Inc. This project, **Immediate Loading of Implant Supported Overdentures Using Magnet Attachments** is a randomized, controlled clinical trial comparing outcomes of immediate vs. delayed loading of implants supporting an existing overdenture.

Dr. Samet is presently recruiting patients for his study on the prevention of aphthous ulcers (canker sores). If you, or someone you know is interested, please call 617-432-1474.

DR. CORTINO (TINO) SUKOTJO

Gene Profiling Analysis of SLA Implant, an In Vivo study in Rats

Congratulations to Tino, an HSDM advanced graduate prosthodontics student and candidate for an MMSc degree (June 2005). Tino received funding from the ITI Foundation for this project, and most recently, a grant from The American Academy of Fixed Prosthodontics.



RESEARCH LAB FOCUS: THE LI LAB

YEFU LI, PHD, MD

*Assistant Professor
Oral and Developmental Biology*

KENPAN HU, DDS

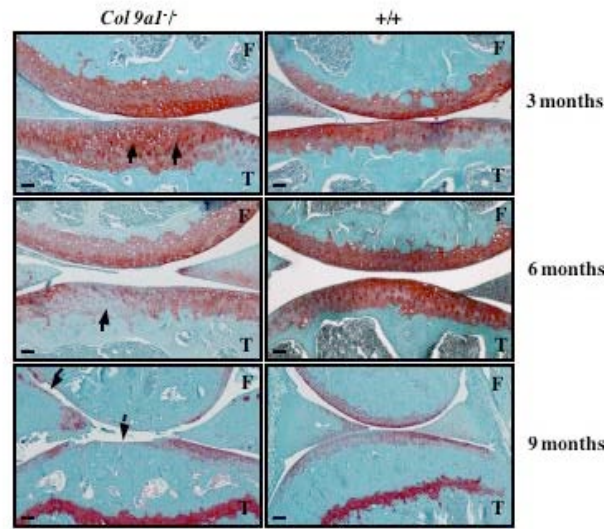
Research Associate

KERWIN KAMWING HO, DMD

Post-Doctoral Fellow

NATALIE LAM, DMD

Post-Doctoral Fellow



Dr. Li's research interest focuses on the unique areas listed below. The image above represents collagen fibers on the articular cartilage of mice knee joints in Dr. Li's laboratory.

(1) Genetic regulation of skeletogenesis

The long-term goal of this project is to elucidate a novel genetic regulatory mechanism of skeletogenesis. For this purpose, Dr. Li studies a spontaneous mouse mutation, osteochondrodystrophy (*Ocd*). *Ocd* is an autosomal recessive disorder associated with cartilage and bone defects. Preliminary data from Dr. Li's laboratory demonstrate that the mutation in *Ocd* results in decreased chondrocyte proliferation and bone formation during skeletal growth. On the basis of high-resolution genetic linkage and physical maps of the *Ocd* locus, Dr. Li has positioned *Ocd* in a defined chromosome region (about 1 million base-pairs containing 37 known genes and open reading frames). To identify the *Ocd* gene, these genes are being examined by direct DNA sequence analysis.

(2) Cartilage mechanics in small animal models of osteoarthritis (OA)

This is a collaborative project with Dr. Olsen and with Dr. Setton at Duke University. The study is aimed at understanding pathogenetic mechanisms of OA by using a naturally occurring mouse model, chondrodysplasia (*cho*). A previous study by Dr. Li and Dr. Olsen demonstrated that a cytosine deletion in the *Col11a1* (the gene for type XI collagen) is responsible for the *cho* phenotype. The deletion causes a reading-frame shift resulting in a premature stop codon. No functional collagen type XI can be detected in the homozygous *cho/cho* mice and cartilage from these mice contains thick collagen fibers and is mechanically extremely fragile. Of particular relevance to human OA is that the presence of thicker collagen fibers in articular cartilage is one of the early pathological changes of OA in humans. This suggests that a reduction of collagen type XI may be a factor in the pathogenesis of OA. Preliminary data support this possibility. Knee joints from heterozygous *cho/+* mice demonstrate progressive and age-dependent degenerative changes in articular cartilage. At the age of 15 months, *cho/+* mice have human OA-like joints. The study is expected to provide novel insights into the role of cartilage matrix genes in the pathogenesis of OA.

Questions? Comments? Suggestions?

<http://www.hsdm.harvard.edu/asp-tml/research.html>

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