10th ANNUAL DEPARTMENT OF PATHOLOGY YOUNG INVESTIGATORS’ DAY
POSTER SESSION
Thursday, April 17th, 2008
TURNER CONCOURSE
REGISTRATION FORM

Applicant’s Name:  _Brian Simons__________________  Degree:  _D.V.M._

Applicant’s Division:  _Urologic Pathology______________________________

Faculty Preceptor:  _David Berman_____________________________________
(Must hold a primary appointment in Pathology)

Appointment Category:   _____House Staff   _____Clin Fellow   ___Research Fellow

        _____Medical Student   _____Graduate Student (Program:____________)

Register for:    _____ Clinical Research   _____Translational Research   ___Basic Research

Full Poster Title *  _A Functional Requirement for the Oncogene Beta-catenin in
         _Prostate Development___________________________

Where has the work been presented?
Meeting Name   _JHMI Dept. of Urology Prostate Cancer Research Day
Meeting Date   _February 9 2008____________________________________

Not Previously Presented  _______________________________________

Where is this work being published?  ___________________________________
Journal Name, Volume, Page, Date _______________________________________

In Preparation (Y/N) - Where?   _No_____________________________________

Author(s) (First & Last)  Brian Simons, Edward Schaeffer, Luigi Marchionni and David Berman

In-House Address: _CRB2 Room 532____________________
(Room # and Building Name, Lab, etc.)

Telephone:   _4432870877__________   Beeper: _3-9921________________

Fax: _______________ ________   E-mail: _bsimons3@jhmi.edu____

*INCLUDE A ONE-PAGE ABSTRACT (including title and all authors) OF THE WORK
YOU WILL BE PRESENTING

***************************************************************************
E-mail COMPLETED Registration form and abstract to:  
Stacey Morgan (smorgan9@jhmi.edu) on or before 
  Friday, March 14th, 2008

If you have questions or problems regarding your submission, please 
contact Stacey Morgan via e-mail (smorgan9@jhmi.edu)
A Functional Requirement for the Oncogene β-catenin in Prostate Development

Brian Simons\textsuperscript{1,4}, Edward Schaeffer\textsuperscript{3}, Luigi Marchionni\textsuperscript{2} and David Berman\textsuperscript{1,2,3}

Departments of Pathology\textsuperscript{1}, Oncology\textsuperscript{2}, Urology\textsuperscript{3} and Molecular & Comparative Pathobiology\textsuperscript{4}, Johns Hopkins University School of Medicine, Baltimore, MD

Developmental programs coordinate a variety of cellular processes including proliferation, invasion, and differentiation. During carcinogenesis, rather than randomly acquiring oncogenic mutations, uncontrolled reactivation of developmental pathways may provide a streamlined route to cancer. Abnormal expression or localization of Wnt pathway members are found in a significant minority of advanced prostate cancer. In rodent models, transgenic mice with activated Wnt signaling develop prostate cancer. The oncogene β-catenin is a transcriptional activator and the central mediator of Wnt signaling, but also plays key roles in E-cadherin mediated cell adhesion and androgen receptor function. Furthermore, microarray analysis identified multiple components of the Wnt pathway as components of the prostate development program in mouse embryos. To further address the role of β-catenin in prostate development, we used a novel organ culture system to efficiently delete β-catenin in the embryonic prostate. In vitro and in vivo development after β-catenin deletion yielded dramatically decreased organ size and strikingly abnormal morphology. Deletion of β-catenin in the adult prostate was less efficient and did not significantly affect organ homeostasis. These findings suggest that β-catenin and Wnt signaling play a key role in prostate development, and further study of this developmental program may give insight into its abnormal function in cancer. Our ability to efficiently target genes for deletion in developing prostate epithelium and mesenchyme should greatly accelerate our efforts to elucidate the mechanisms governing prostate growth and differentiation.