17th Annual Research Day Symposium
March 28, 2012

ABSTRACT FORM INSTRUCTIONS

Eligibility:

1. Students: Any student within the invited GWU departments may submit one abstract as the first author.
2. Alumni: If you graduated after April 2011 you may submit one abstract.
   For Students & Alumni: Your faculty advisor MUST BE A CO-AUTHOR AND MUST REVIEW AND APPROVE YOUR ABSTRACT PRIOR TO SUBMISSION
3. Faculty: Any faculty member within the invited GWU department may submit one abstract as a first author.

Guidelines:

1. Only one abstract per presenter as first author will be accepted.
   • Each student/alum may submit one abstract only.
   • Although a faculty member may be the sponsor of more than one student abstract, faculty may submit only one abstract as a presenting author.
2. All abstracts should include the following elements:
   ✓ Title
   ✓ Author(s) [including faculty advisor for students and eligible graduates]
   ✓ Current Affiliation of each author
   ✓ Objectives/Background
   ✓ Methods
   ✓ Results to Date
   ✓ Conclusions

Instructions:

1. All of the required elements (stated above) must be included for printing in the Research Day abstract book.
2. For all submissions SEE SAMPLE FORMAT BELOW:
   • THE TEXT OF THE ABSTRACT SHOULD BE NO LONGER THAN 400 WORDS. Please note: the word count pertains to the abstract text ONLY. The abstract title and authors are NOT counted toward the word count total.
   • Student/Alumni submissions: Faculty advisor name should be followed by (faculty advisor). Please be sure your faculty advisor has approved your abstract PRIOR to submission.
3. The abstract form can be filled out online at the following website http://research.gwu.edu/newsevents/events/researchdays2012 (Abstract Submission form can be found on the right side of the page).
4. Click on the submit button and your abstract will be emailed to Joyce Javois at jjavois@email.gwu.edu
5. Abstracts must be received by 5 P.M., WEDNESDAY, FEBRUARY 1, 2012. NO EXCEPTIONS.

PLEASE DO NOT INCLUDE THIS INSTRUCTION SHEET WITH YOUR ABSTRACT SUBMISSION

SAMPLE ABSTRACT
Inhibition of TGF-β1 Leads to Improved Lymphatic Regeneration Following Surgical Wounding
Daluvoy, Sanjay: George Washington University; Avraham, Tomer: Memorial Sloan Kettering Cancer Center; Clavin, Nicholas: Memorial Sloan Kettering Cancer Center; Mehrara, Babak: Memorial Sloan Kettering Cancer Center

Background: TGF-β1, a known regulator of abnormal wound repair, has recently been shown to have a direct inhibitory effect on lymphatic endothelial cells (LEC) within tumors. Therefore the purpose of these studies was to further evaluate the impact and role of TGF-β1 on wound repair and lymphatic regeneration following surgical lymphatic ablation.

Methods: Circumferential full thickness skin excisions and microsurgical ligation of the deep lymphatics were performed on the tails of mice. In order to prevent wound desiccation all wounds were filled with collagen type I gel and an occlusive dressing. Animals were divided into various groups and treated with either collagen gel, collagen gel impregnated with 50ng rhTGF-β1, collagen gel with a TGF-β1 dominant negative virus, or collagen gel with LacZ adenovirus. Lymphatic regeneration and function were evaluated using tail volume measurements, lymphoscintigraphy, and immunohistochemistry after 2 and 6 weeks.

Results: When compared to collagen controls alone, the addition of TGF-β1 resulted in nearly 40% greater increase in tail volumes from baseline at 6 weeks following surgery (p<0.03), impaired lymphatic transport (p<0.001), decreased LEC proliferation (p<0.007), impaired lymphatic capillary regeneration, and in lymphatic fibrosis at all time points following surgery. Conversely, blockade of TGF-β1 by dominant-negative adenoviral transfection resulted in approximately 40% less increase in tail volumes from baseline (p<0.03), improved lymphatic transport (p=0.01), and increased LEC proliferation and recruitment. The addition of vehicle control LacZ adenovirus demonstrated effective transfection, but no significant differences from collagen gel alone in all other assays.

Conclusions: We show that TGF-β1 plays a central role in inhibiting lymphatic regeneration following surgical wounding. Administration of exogenous TGF-β1 impaired LEC proliferation and caused lymphatic capillary fibrosis. In contrast, blockade of TGF-β1 activity markedly accelerated and improved lymphatic regeneration. These findings suggest that TGF-β1 inhibition may be a means for improving lymphatic regeneration clinically, thereby reducing the risk of lymphedema.