Available Immediately:

Postdoctoral position to study the role of TGF beta and inflammation in aneurysm production

We have funds to hire an ambitious and motivated Ph.D. to investigate the role of TGF beta and inflammation in the pathogenesis of thoracic aneurysms. In animals and humans, a number of mutations of proteins of the extracellular matrix (ECM) result in dilation and eventual rupture of the thoracic aorta. The most common of these conditions is Marfan syndrome, an autosomal dominant condition caused by mutations in the gene for fibrillin-1, a large multi-domain, ubiquitous ECM protein. The one other known protein group in the fibrillin family are the latent transforming growth factor beta (TGFβ) binding proteins (LTBP). As the name implies, the LTBPs bind TGFβ. The fact that fibrillin-1 binds to LTBP-1 led to the suggestion that abnormalities in fibrillin-1 might affect TGFβ function. Indeed, lowering TGFβ levels in Marfan syndrome mice decreases the severity of aortic aneurysms. We have found that loss of LTBP-3 in Marfan mice also prevents dilation and protects against rupture. Additionally, the loss of Rag2 in Marfan mice blocks rupture but does not inhibit dilation.

Our current research is focused on the molecular role of LTBP-3 in aneurysm production, the role of the immune system in vessel rupture, and early and late contributions of LTBPs and TGFβ to blood vessel physiology. We are currently using mouse genetics, as well as cell, molecular, and genomic approaches to identify unique molecular targets of TGFβ, as well as immune-dependent lytic reactions. We also use cell culture methods to examine the effect of permuting specific TGFβ-related proteins of the physiology on vascular smooth muscle cells derived from our mouse models.

In addition, we are interested in the role of LTBPs in tumor metastasis. We use mouse and chick models for metastasis and we have found that decreasing LTBP-3 levels severely diminishes the number of metastatic cells, presumably by modifying the angiogenic response. Work on this project will go forward with analysis of the biochemical mechanism affecting angiogenesis both in vitro and in vivo.

Rifkin Lab Publications
http://www.ncbi.nlm.nih.gov/sites/myncbi/daniel.rifkin.1/bibliography/46398929/public/?sort=date&direction=descending

Our laboratory is located on the main NYU School of Medicine campus at 31st Street and First Avenue in mid-town Manhattan. We collaborate with several groups within NYU School of Medicine as well as groups at Mt. Sinai School of Medicine and Yale University Medical School. We provide an exciting and stimulating scientific atmosphere with emphasis on the mechanistic and translational aspects of growth factor, matrix, and vascular biology. We will provide a competitive salary and excellent benefits.

To apply, please send your CV, a letter describing your research experience and career goals, the names and contact information for three individuals who can serve as references, and expected availability date to Dr. Daniel Rifkin at Daniel.Rifkin@nyumc.org