RLS/WED Research Grant Recipients

The RLS Foundation Board of Directors met in April 2012 to consider the research grant applications reviewed by the Scientific Advisory Board. The quality of the grant submissions was so high that the Board voted to fund two research grants in 2012.

This is an interview with Stephanie Patton, PhD of Pennsylvania State University. Dr. Patton is an Assistant Professor of Neurosurgery with a PhD in Pharmaceutical Sciences with an emphasis in Biomedical Chemistry. She has also received research grants from the Restless Legs Syndrome Foundation in 2005, 2006 and 2009. The title of this project is: The role that the nitric oxide pathway plays in regulating vasodilation of the legs in restless legs syndrome.

RLSF Staff: Can you describe your research project?

Dr. Patton: My research project, which was funded in October 2012 by the RLS Foundation, involves an examination of the role of the nitric oxide (NO) pathway plays in regulating vasodilation in RLS subjects. We examined the role of the NO pathway in two ways: 1) by examining brain microvessels from RLS autopsy subjects for changes in NO protein and 2) by examining the blood flow in the femoral artery of RLS and control subjects under normal and low-oxygen conditions.

RLSF Staff: What is the primary goal of your research?

Dr. Patton: The primary goal of my research is to determine whether changes in blood flow occur in the femoral artery of RLS subjects and how these differ from control subjects.

RLSF Staff: Why is this important to the RLS research community?

Dr. Patton: The information generated by this study will permit us to identify additional mechanistic pathways for RLS and potentially develop and identify diagnostic and treatment strategies.

RLSF Staff: You have been awarded four grants from our Foundation since 2005. How has this support shaped your research career?

Dr. Patton: The generous funding provided by the RLS Foundation has permitted me to establish novel in-roads into the pathophysiology of RLS. The research I have established focuses on the etiology of RLS and its potential mediation by two pathways: the hypoxia inducible factor (HIF) pathway and the nitric oxide synthase pathway. In the early grants (2005-2007), I recognized that iron homeostasis proteins were changed in the lymphoblasts of RLS patients. Because lymphoblasts reside outside the brain, these findings suggested that cellular changes involved in RLS are global in nature. In that early work, it was further evident that there were significant changes in the hypoxia pathway and its activation. In the 2009-2010 grant, I showed that there were changes in the hypoxia-induced proteins found in the brains of RLS subjects at autopsy. Further, a protein in a pathway downstream of HIF activation neuronal nitric oxide synthase (nNOS) — was also found to change in a manner parallel to hypoxia-induced proteins.

In the end, understanding the interrelatedness of these pathways may provide the Foundation for both a more basic understanding of RLS and perhaps specific cellular targets for affecting cellular processes in RLS. Building on this work, the 2012 grant, expands my research into the exploration of physiological differences (blood flow changes) in the legs of RLS subjects. By examining blood flow changes when patients are experiencing induced, temporary hypoxia, we hope to tie the changes in hypoxia-induced proteins to the changes in microvasculature flow.

Overall, it almost goes without saying that RLS Foundation funding has provided a deeper understanding of the processes involved in RLS. Further, the work performed under these grants has provided significant preliminary data for a grant proposal to the National Institutes of Health. Lastly, this work is allowing me to establish my career as a Primary Investigator in the molecular and cellular process involved in a disease.