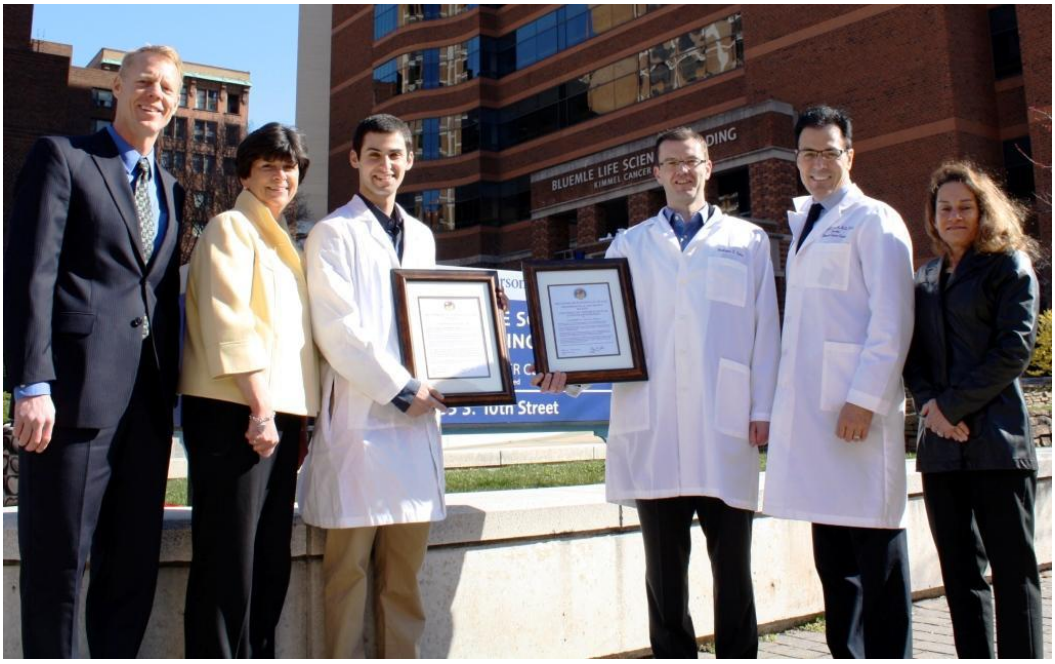
2013 JMNMF RSA Summary & Photo
Curtis H. Kugel, III, Thomas Jefferson University, Kimmel Cancer Center



Pictured Left to Right: Greg Safko, JMNMF President; Regina S. Bodnar, RN, JMNMF Chair; Curtis Kugel, III, JMNMF RSA Recipient; Andrew Aplin, PhD, Professor, Department of Cancer Biology; Richard Pestell, MD, PhD, Director, Kimmel Cancer Center, Professor and Chairman, Department of Cancer Biology, Thomas Jefferson University; and, Esther Hoffberg, JMNMF Board Member.

Targeting ERBB3 in Combination with RAF Inhibitors in Murant BRAF Melanoma

Skin cancer is the most common cancer in the United States. Melanoma type skin cancer represents less than 5% of all diagnosed skin cancers however, is responsible for over 80% of skin cancer related deaths each year. This makes melanoma the most deadly form of skin cancer. In 2011 the FDA approved the use of a new anti-cancer drug called Vemurafenib designed specifically to block a mutated protein proven to be the root cause of approximately 50% of all melanomas. Patients treated with Vemurafenib saw a remarkable decrease in the size of their tumors. Some tumors even seemed to disappear altogether. However, most of the tumors including those that seemingly disappeared eventually grew back while still be treating with the drug. It is our belief that a very small portion of the melanoma cells have the inherent ability to survive the initial treatment with Vemurafenib. These surviving cells persist in a somewhat dormant state until additional changes occur that allow the cells to re-grow in the presence of the drug as Vemurafenib resistant tumors. A number of these additional changes have already been identified by our lab and others. Consequently, the focus of my research is to target and destroy the original surviving cells before they can acquire any changes that allow them to become resistant to treatment. Our lab has identified a protein found at the surface of the melanoma cells that we believe to be responsible for the ability of the melanoma cells to survive the initial Vemurafenib treatment. My research aims to identify how this protein allows melanoma cells to survive drug treatment, and it is our hope that by targeting this surface protein in combination with Vemurafenib we can destroy all of the melanoma cells before any have a chance to develop resistance.