Investigator:	**Brian Pollack** Phone: (404) 321-6111 ext. 207573 Email: bppolla@emory.edu
Primary Research Interest:	Dermatology
Description of Research:	The overarching hypothesis of our research is that the immunologic effects of EGFRIs and MAPKIs on tumor cells and non-tumor cells impact anti-tumor immunity. Our research goals are to define the mechanisms through which EGFR/MAPK signal transduction modulates the expression of major histocompatibility complex (MHC) genes (focusing mainly on MHC class I (MHCI)) and to define how EGFRIs/MAPKIs influence anti-tumor immune responses in vivo. Ongoing experiments are predicated upon the literature documenting the ability of EGFRIs and MAPKIs to modulate immune responses in vivo including those against tumors, and our published and unpublished findings that EGFRIs and/or MAPKIs augment MHC expression and that locally delivered EGFRIs can enhance immune cell activation and antigen-specific immune responses in vivo. The rationale for our work is that defining how these medications influence anti-tumor immunity will ultimately add critical insight to optimize their use in Veterans with cancer.
Relevance to VA:	Aberrant expression and/or activation of the epidermal growth factor receptor (EGFR) or components of the mitogen-activated protein kinase (MAPK) pathway such as RAS family members and BRAF occur in many cancers affecting veterans. Because of this, large efforts to develop medications that can inhibit the enzymatic activity of the EGFR and enzymes within the MAPK pathway have taken place recently. These efforts have led to the approval (by the FDA) of several EGFR inhibitors (EGFRIs) and MAPK inhibitors (MAPKIs) also known as ‘targeted therapies’ for the treatment of advanced cancer. However, these medications are rarely curative and the overall prognosis for patients with advanced cancer is still poor. Thus, there is a critical need to potentiate the response to targeted therapies and broaden existing paradigms to treat Veterans with advanced cancer.