**Recent Grant Awards**

Jacek Hawiger, MD, PhD  
VA Merit Award  
“Regulation of Innate Immunity and Inflammation Through Nuclear Reprogramming”

Jon Kropski, MD  
1. Pulmonary Fibrosis Foundation  
“Enhancing ER chaperone function to prevent herpesvirus-induced lung fibrosis.”

2. Vanderbilt Faculty Research Scholars Award

Pierre Massion, MD  
VA Merit Award  
“Phenotypic Heterogeneity in Small Cell Lung Cancer”

Ciara Shaver, MD, PhD  
Vanderbilt Faculty Research Scholars Award

**Vanderbilt 5 Star Excellence Award Winners**

Congratulations to Drs. Lisa Lancaster, MD, Ivan Robbins, and Elisabeth Willers for each being awarded 5 Star Excellence Awards for Provider Medical Specialty Services, Overall Quality of Doctor Care. Additionally, the MICU received an award for inpatient services for overall quality of care, and Pulmonary Function Testing received one for outpatient services overall quality of care.

**Misc Notes from Faculty meeting**

- Fellowship interviews for both programs begin in August
- The case conferences start next Thursday, August 6th
- MCN renovations are underway

**Vanderbilt UAB Research Summit**

The First Vanderbilt / UAB Pulmonary Research Summit was a great success. The focus of this years summit was Repair and Remodeling. Below are a few pictures from the summit, which was held on June 26 - 27.
Marginal zone (MZ) B cells have immunologic properties that are not shared by other mature B cells and have been implicated in type 1 Diabetes. To test disease contributions of MZ B cells in NOD mice, Notch2 haploinsufficiency (Notch2+/−) was introduced but failed to eliminate the MZ, as it does in C57BL/6 mice. Notch2+/− mice have MZ B cells numbers similar to those of wild-type C57BL/6, yet still develop diabetes. To test whether SCF also supports MZ B cells, Cre-driver of transgenic Notch2lox/lox B cells was utilized. Surprisingly, MZ B cells failed to develop in B6-ΔF5/6Notch2−/− NOD mice. Expression of Notch2 isoforms and its transcriptional target, IRF7, was increased in MZ B cells compared with C57BL/6 B cells. B6ΔF5/6Notch2−/− delay difference reduced Notch2−/− signaling specifically in NOD B cells, suggesting that BCR signaling enhances Notch2 signaling in this autoinflammatory model. The role of BCR signaling was further investigated using an anti-immunoglobulin transgene (3y BCR, B2G). Antiimmunoglobulin in ΔF5/6Notch2−/− mice populated an enlarged MZ, suggesting that low-level BCR signaling overcomes reliance on Notch2. Teaching definitions of anti-mutants B cell in MZ in the C57BL/6NOD mice showed that IRF-dependent apoptosis in the MZ depends on strength of antigen binding, whereas Notch-mediated selection does not. Importantly, anti-immunoglobulin B cells numbers were reduced by ΔF5/6, but Notch2 haploinsufficiency limits MZ B cell expansion without provoking type 1 diabetes. Notch2−/− mice, and MZ B cell survival occurs on BTK more than Notch2−/−, regardless of MZ location, which may have important implications for therapeutic intervention strategies.


Notch2 is critical for MZ B cell development (8–12). Notch2 on the MZ surface transmits signals from the BCR, is known for its role in FO B cell maturation, and permits FO B cell expansion. MZ B cells expressing Notch2 also allow increased numbers of autoreactive B cells to reach maturity (8, 12). As Notch2 haploinsufficiency limits MZ B cell expansion without provoking type 1 diabetes in C57BL/6 mice, and MZ B cell survival occurs on BTK more than Notch2−/−, regardless of MZ location, which may have important implications for therapeutic intervention strategies.