UA Department of Emergency Medicine’s **Jarrod M. Mosier, MD**, also associate program director, DOM Critical Care Fellowship, spoke on “Translating Research to a Life Saved” and **Stephen M. Black, PhD**, with the Division of Translational & Regenerative Medicine, discussed “Metabolic Reprogramming in Pulmonary Hypertension.”
Glucose Metabolism

Metabolic Hypothesis of PAH

Over the last decade a number of studies have shown that during the development of PAH there is a switch from oxidative phosphorylation to the Warburg effect.

Otto Heinrich Warburg

Glucose metabolism is disrupted in PAH

PKC is up-regulated in human PAH lungs
Conclusions

- Current therapies for PH are focused on increasing PV relaxation and do not reverse disease progression.
- In vivo, PH appears to be a disease of metabolism and is associated with changes in glycolysis and NO production.
- Increasing the glucose-organic acid (OGA) ratio reduces PH development in certain PH conditions.
- Our approach is to reduce the activation of the nrf2 pathway signaling by reducing the nrf2 pathway in PH.
- The nrf2 pathway in PH is critical in its development.
- Reducing nrf2 pathway activation reduces NO production.
- Coenzyme supplementation preserves NO signaling and endothelial function in early forms of PH.
- Coenzyme supplementation prevents the development of more advanced PH.
Archived video of the Feb. 8, 2018, DOM Research Seminar can be viewed here.
Photos courtesy of David Mogollón, Communications Coordinator, UA Department of Medicine, (520) 626-1137 or dmogollon@deptofmed.arizona.edu