**Weekly Colloquium**

Tuesday, 03/01/2016, 12:30pm, Billings Building – Rosedale Conference Room

“Surviving Under (ER) Stress: p21 as a Modifier of the Unfolded Protein Response”

Hippokratis Kiaris, Ph.D.  
Associate Professor  
Director, Peromyscus Genetic Stock Center  
Department of Drug Discovery and Biomedical Sciences  
University of South Carolina

---

**Research Abstract**

At conditions during which protein production exceeds the cells’ folding capacity endoplasmic reticulum (ER) stress is induced that in turn initiates the unfolded protein response (UPR). Initially UPR is adaptive while subsequently, during prolonged or intense ER stress becomes proapoptotic. This pro-apoptotic action of UPR has been associated causatively with the onset of various pathologies such as neurodegeneration and diabetes. We have identified the anti-apoptotic cell cycle regulator p21 as a target of the UPR and an important modifier of its outcome, promoting the pro-survival mode of action. Modulation of p21 activity alters the sensitivity of pancreatic islets to glucotoxicity and changes the susceptibility of mice to diabetes. Experiments in aged mice as well as in pancreatic islets from older animals suggest that this role of p21 as a modifier of the outcome of the UPR is particularly relevant during aging. Pharmacological activation of p21 may be a strategy of choice to manage diabetes and other aging-associated pathologies.

---

**Related References**


---

For more information contact: dwhite@burke.org

THE BURKE MEDICAL RESEARCH INSTITUTE IS AN ACADEMIC AFFILIATE OF WEILL CORNELL MEDICINE

---