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## Dysregulation of mitochondrial quality control and diabetic cardiomyopathy

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**Background:** Mitochondrial dysfunction and reactive oxygen species (ROS) are critical to diabetic heart damage. However, antioxidant therapies have failed to reduce heart failure in clinical trials, underscoring the need to develop new therapeutic strategies. A healthy pool of mitochondria is maintained through a number of quality control mechanisms including mitochondrial autophagy known as mitophagy which degrades dysfunctional mitochondrial fragments that are segregated by the fission process.

**Objectives:** This study investigates the functional status and roles of mitophagy and mitochondrial fission in type 1 diabetic heart.

**Methods:** A novel dual fluorescent mitophagy reporter was used to label and quantify mitochondrial fragments that are being degraded within the lysosome in isolated cardiomyocytes and in type 1 diabetic hearts. The functional roles of mitophagy and mitochondrial fissionwere determined in cardiomyocytes by using genetic gain- and loss-of-function approaches.

**Results:** Using this mitophagy reporter, we found that mitophagic flux is inhibited in cardiomyocytes cultured in media that mimic hyperglycemic conditions. Parkin overexpression diminishes, while parkin knockdown exaggerates high glucose toxicity, as measured by the levels of ROS generation, oxidative injury and cardiomyocyte death. Further, high glucose increases mitochondrial fragmentation and Drp1 knockdown inhibits this effect, predisposing cells to high glucose toxicity. Similarly, mitophagy is inhibited in type 1 diabetic heart as assessed by mitophagy reporter mice, which is associated with increased small-sized mitochondria.

**Conclusion:** These findings not only demonstrate a mismatch between mitochondrial fission and mitophagy in high glucose-treated cardiomyocytes and in the diabetic heart but also suggest that improving mitochondrial quality control processes may protect the heart against hyperglycemic toxicity in type 1 diabetes.

### Biography

Qiangrong Liang was MD, in 1986, Xian Medical University, Xian, China. He has completed his PhD, 1999, from University of North Dakota, Grand Forks, ND. also did the Postdoctoral Training, in 1999-2003, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio. He was the Assistant and Associate Professor, 2003-2012, Sanford Research-University of South Dakota, Sioux Falls, SD. Currently he is the Associate Professor, 2013, Department of Biomedical Sciences, NYIT College of Osteopathic Medicine, Old Westbury, NY. Peer-reviewed publications: 35. Dr. Liang's research interest is in the cellular and molecular mechanisms that underlie acquired heart disease, cardiac hypertrophy and heart failure. His laboratory is conducting research in three areas: 1) Investigate why diabetes exacerbates progression to heart failure and explore mechanism-based approaches to reduce this susceptibility. 2) Explore mechanisms of myocardial protection by caloric restriction (CR) and develop drugs that mimic the beneficial effects of CR. 3) Investigate why the anti-cancer drug doxorubicin (DOX) can cause heart failure and how myocardial homeostasis can be restored by coordinately promoting survival mechanisms and blocking cell death pathways.

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