Research points to a new way to protect kidneys threatened by insufficient blood or toxins (w/Video)

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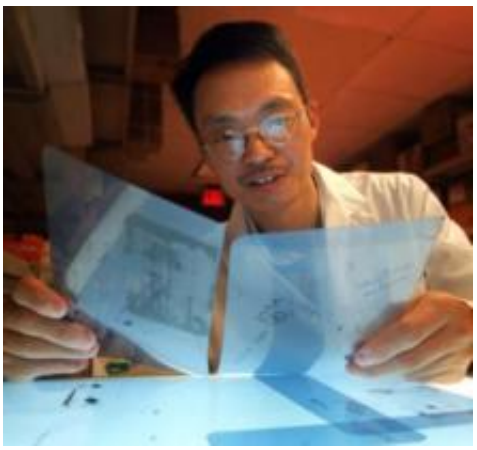

Dr. Zheng Dong, cell biologist in the MCG Schools of Medicine and Graduate Studies and at the Charlie Norwood Veterans Affairs Medical Center. Credit: Medical College of Georgia

Better treatments for acute renal failure may be possible by blocking the mitochondrial fragmentation that occurs when kidneys don't get enough blood or are exposed to toxins, researchers at the Medical College of Georgia report in the may issue of the *Journal of Clinical Investigation*.

Stress on kidney cells caused by vascular obstruction, trauma, chemotherapy, even antibiotics cause mitochondria - the cell's powerhouse - to "go to pieces," says Dr. Zheng Dong, cell biologist in

the MCG Schools of Medicine and Graduate Studies and at the Charlie Norwood Veterans Affairs Medical Center.

Fragmentation sets in motion a chain of events that prompts kidney cells to commit suicide and leads to acute renal failure.

"When mitochondrial fragmentation is blocked, it can save the cells and the kidneys," he says.

Dr. Dong and his colleagues used an inhibitor of the natural fission process mitochondria use to multiply. They will further examine the cell death pathway - and try blocking it - in human kidney biopsies and eventually in whole organs. One of his goals is to develop safer, more efficacious drugs that can be given to patients in acute renal failure.

Acute renal failure, a disease with high mortality rates, is on the rise because of increasing cases of cardiovascular disease, diabetes and other health-related problems that prevent the kidneys from getting sufficient blood.

In addition to acute renal failure, Dr. Dong believes fragmentation inhibitors can improve preservation of kidneys extracted from a donor for transplant.

What these drugs may block is a rapid death march that only begins with the breakup. Next, two proteins, Bak and Bax, typically found at divergent places in the cell, work together to make pores in the mitochondrial outer membrane. Cytochrome c, a protein that normally helps mitchondrion breathe and produce fuel, escapes through the pores. Out of place, it becomes a deadly accomplice that activates suicide pathways. Interestingly, in a study published in 2007 in the *Proceedings of National Academy of Sciences* (USA), Dr. Dong's laboratory showed that mitochondrial fragmentation involves Bak but not Bax.

Source: Medical College of Georgia