Bilateral kidney ischemia/reperfusion injury

AN *IN VIVO* MODEL FOR ACUTE KIDNEY INJURY

Model

Ischemia/reperfusion injury (IRI) is characterized by restriction of blood supply to an organ followed by restoration of blood flow and re-oxygenation. Ischemia is a leading cause of acute kidney injury (AKI) which temporarily interrupts the supply of oxygen and nutrients to the kidney, initiating a cascade of deleterious cellular and molecular responses primarily in tubular epithelial cells.
In this model, IRI is induced by bilateral renal pedicle clamping impairing renal function and eliciting tubular injury.

Species

- Rat
- Mouse

Interest

- The bilateral ischemic AKI model is considered more relevant to human pathological conditions where blood supply is affected in both kidneys.
- This model is suitable for testing compounds aimed to prevent and/or reverse kidney dysfunction after ischemia reperfusion injury.

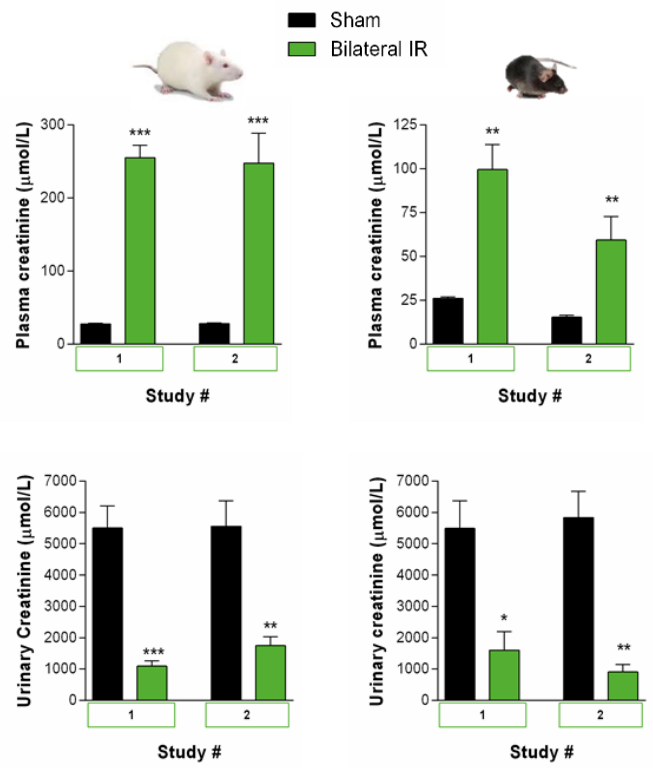
Model Description

- Surgical procedure: atraumatic clamping of both renal pedicles (ischemia) followed by blood flow restoration for 24-72 hours (reperfusion)
- Pathophysiological features: impaired renal function and tubular injury
- Species differences are observed in this model.

Evaluated Parameters

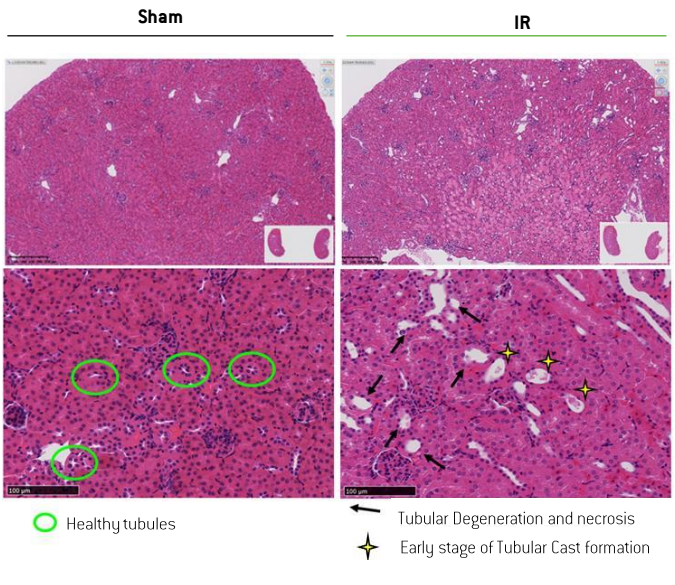
- Body and kidney weight
- Renal function:
  - Biochemical dosage of plasma and urinary creatinine and urea
  - Estimated and transdermal Glomerular Filtration Rate (GFR)
- Tubular injury:
  - ELISA dosage of specific markers (NGAL and KIM-1)
  - Histomorphometry: evaluation of tubular damage by Hematoxylin/Eosin staining

Impaired renal function (rat and mouse model)



* P<0.05, ** P<0.01 and *** P<0.001 (n=5-12/group)

Kidney tubular damage (mouse model)



HE staining after 24h reperfusion