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Urine metabolic biomarkers for mouse ischemic acute kidney injury and nephrotoxic acute kidney injury

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Acute Kidney Injury (AKI) previously known as acute renal failure, refers to abrupt reduction in renal function or urine output, occurring over hours or days, caused by kidney damage due to sepsis, respiratory failure, heart failure, trauma, major surgery, burns, toxic insult caused by medications and contrast agents used for imaging. AKI accounts for 1% of all hospital admissions, complicates 7% of hospitalizations and is present in up to 20% of critically ill patients. Overall mortality associated with AKI is estimated at 45-70% mortality associated with AKI in intensive care unit patients requiring renal replacement therapy is 50-60% and more than 2 million die from AKI each year. The aim of the present study was to examine the effects of Ischemia and Nephrotoxin induced AKI on the urine metabolomic profile. Two groups of Swiss-Webster mice were injected with cisplatin or renal ischemia reperfusion injuies (IRI) were performed, respectively, on the mice to induce AKI. Serum creatinine and urine neutrophil gelatinase-associated lipocalin levels were tested to confirm AKI after the injection and the surgery. After analysis of NMR data, significant metabolic differences were found. Metabolic differences could result in the discovery of new biomarkers for AKI. Comparison of the differences between the IRI and cisplatin treated mice could give insight to a common biomarker for AKI.

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Phenotyping the virulent switch in *C. alibicans* using fingerprinting and footprinting metabolomics

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Candida albicans is a polymorphic yeast that inhabits the gastrointestinal tract of humans as part of the commensal microflora under normal conditions. But when the host defenses are compromised, *C. albicans* can become an opportunistic pathogen causing both superficial and/or severe systemic infection. *C. albicans* has the ability to grow as budding yeast (round shaped cells) that can switch to hyphae cells (presence of elongated filament). Hyphae cells are considered the most virulent form mainly because its structure facilitates adhesion and invasion into the body and is more resistant against host defenses. Suitable diagnostic targets must be identified to inhibit the yeast-to-hypae transition, which can be only accomplished by understanding the molecular changes that occur during the virulent switch. Therefore, the objective of the work was to phenotype the conformation changes of *C. albicans* during its virulent progression using fingerprinting and footprinting metabolomics, with an emphasis on Fourier Transform Infrared (FTIR) spectroscopy. Whole cell fingerprinting and footprinting of two *C. albicans* strains (A72 and SC5314) indicated different conformations and metabolic during the shift, but for each the most significant occurred at the 2-3 hour transition point. FTIR spectroscopy of isolated DNA further showed that genetic conformation differed between the two strains during the switch, but their DNA phenotype was similar after transition to the most virulent phenotype, the hyphae stage. Such conformation changes, as evidenced by shifts in peak positions, bandwidths, and band intensities, provide valuable structural and functional information to develop appropriate agents for preventing infections.

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