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Use of zebrafish for studying chemical induced acute kidney injury and chronic kidney disease

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Objectives: Prevalence of kidney diseases such as acute kidney injury (AKI) and chronic kidney diseases (CKD) has been increasing. While several chronic diseases, e.g., diabetes and hypertension, may explain some of these kidney diseases, evidences are accumulating that support importance of chemical exposure in etiology of AKI and CKD. Experimental animal models can be applied for developing sensitive and fast screening tools for renal toxicants, or for understanding their underlying mechanisms. Zebrafish (*Danio rerio*) is one of the most widely used experimental models for toxicological studies. This literature review was conducted to assess utility of zebrafish in investigating chemical-induced renal toxicity. The observation endpoints identified in this study may be used for fast and sensitive investigation for renal toxicity of chemicals.

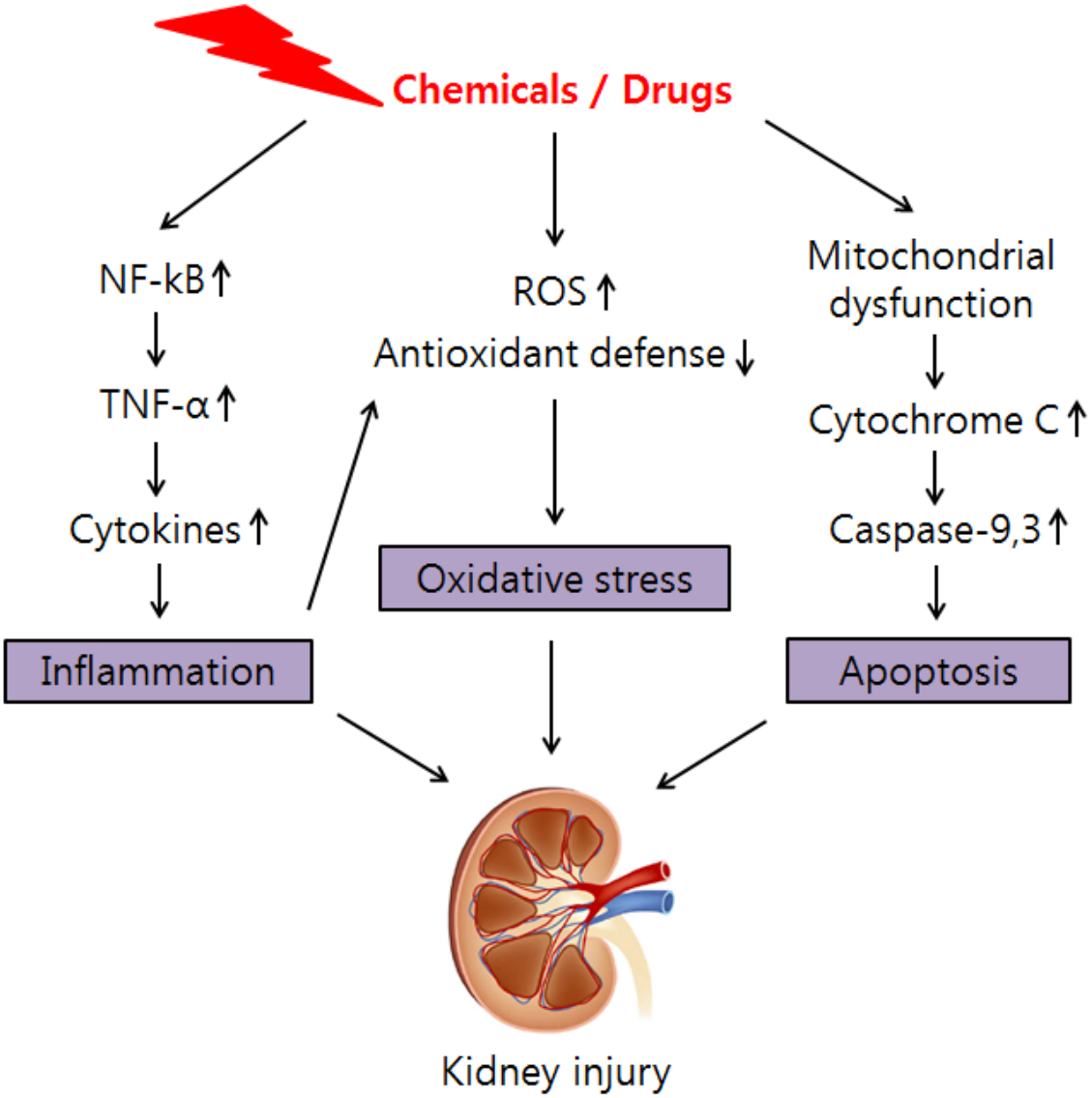
Methods: Available literatures that employed zebrafish for chemical-induced kidney toxicity were obtained from PubMed. Keywords such as 'zebrafish', 'nephrotoxicity', 'mechanism', 'chemical' and 'induced' were used. Observation endpoints considered included biochemical, molecular, histopathological, and functional level markers that related to chemical induced kidney toxicity.

Results: A total of 26 papers were identified. Most widely studied chemicals in zebrafish were gentamycin and aristolochic acid. Most studied mechanisms of chemical-induced kidney toxicity include apoptosis, oxidative stress, and inflammation, along with those related to kidney injury. For functional changes of kidney, clearance assay and protein urea were often employed. Changes of kidney histology can be used, along with markers of kidney toxicity at the cellular, protein, and gene levels. These observation endpoints are summarized.

Conclusions:

Zebrafish is a promising model for studying chemical-induced kidney diseases. This model can be employed for fast and sensitive screening for consumer chemicals with renal toxic potential.

Figure 1. Major mechanisms of kidney injury


Chemicals / Drugs
NF-kB↑
TNF-α↑
Cytokines↑
Inflammation
ROS↑
Antioxidant defense↓
Oxidative stress
Mitochondrial dysfunction
Cytochrome C↑
Caspase-9,3↑
Apoptosis
Kidney injury