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Cell Death in Kidney Disease

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In the last decade, the role of apoptosis in the pathophysiology of acute kidney injury (AKI) and AKI to chronic kidney disease (CKD) progression has been revisited as our understanding of ferroptosis and necroptosis has emerged. A growing body of evidence ascribes a central pathophysiological role for ferroptosis and necroptosis to AKI, nephron loss, and acute tubular necrosis. I will introduce concepts to the non-cell-autonomous manner of kidney tubular injury during ferroptosis, a phenomenon that we refer to as a "wave of death" of a kidney tubule.
The remaining necrotic debris requires effective removal processes to prevent a secondary inflammatory response, referred to as necroinflammation. Open questions include the differences in the immunogenicity of ferroptosis and necroptosis, and the specificity of necrostatins and ferrostatins to therapeutically target these processes to prevent AKI-to-CKD progression and end-stage renal disease.