GLOSSARY

DAMPs	Danger/damage-associated molecular patterns are molecules with certain functions during homeostasis that turn into an immunostimulatory or cytotoxic alarm signal once injury misplaces them out of their natural compartment, i.e. extracellular histones or Tamm-Horsfall protein leaking from injured tubules.
Danger control	Response programs such as clotting, inflammation, regeneration, and scarring that were positively selected by evolution because they limit potentially fatal dangers such as bleeding, infection, barrier dysfunction, and tissue instability, respectively.
Inflammation	Local or systemic expression of cytokines that activate endothelial cells. This implies vascular plasma leakage and recruitment of activated leukocytes to enforce killing of pathogens, if present. Collateral tissue injury is particularly obvious in the absence of pathogens, i.e. sterile inflammation. Systemic inflammation may present with fever or as shock.
Adaptive immunity	Antigen-induced and -specific Immune response based on antigen-presentation and antigen-specific B and T cells.
Innate immunity	Genetically determined, intrinsic mechanisms of host defence.
Necrosis	Mode of cell death characterized by the rupture of the plasma membrane.
Necroinflammation	An auto-amplification loop of cell necrosis and inflammation that is driven by DAMP-release of necrotic cells and inflammation-related regulated necrosis.
PAMPs	Pathogen-associated molecular patterns activate immune and parenchymal cells via specific pattern recognition receptors, i.e. bacterial endotoxin/LPS activating Toll-like receptor-4.
Regulated necrosis	Active forms of necrosis induced via specific signalling pathways. Such pathways can be activated from outside the cell (outside-in) via distinct surface receptors or from inside the cell during cell stress. Blocking these pathways prevents necrosis.