세미나 초록

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발표 주제	**Necroptosis: Molecular Mechanisms and Disease Implications**
발표 내용	Necroptosis is distinguished from apoptosis in that it does not require caspases, and unlike apoptosis, necroptosis directly results in plasma membrane rupture. Necroptotic cells may play multiple roles in innate immunity and shape subsequent adaptive immunity through the release of endogenous danger signals known as damage-associated molecular patterns (DAMPs), which interact with pattern recognition receptors (PRRs) of innate immune cells to prime immune cells to respond to pathogens and potentially harmful cells, such as those that are infected or tumorigenic. Receptor-interacting protein kinase-3 (RIP3, or RIPK3) is an essential protein for necroptosis, along with its upstream sister kinase RIPK1, which it interacts with via a homotypic interaction motif (RHIM). Mixed Lineage Kinase Domain-like protein (MLKL) is an essential target of RIPK3 kinase activity in necroptosis. The kinase activity of RIPK3 is required for downstream signaling events in necrotic cell death which is canonical function. Over the years, our understanding of a core necroptotic pathway consisting of RIPK3 activation increased substantially, but the recent discovery indicates that RIPK3 kinase may functions through non-canonical pathway, and also suggests tissue-specific roles of RIPK3. In this seminal, I will discuss about the functions of RIPK3 in various human diseases.