**Necroptosis and its role in the pathogenesis of human diseases**

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**ABSTRACT**

Necroptosis is distinguished from apoptosis in that it does not require caspases, and unlike apoptosis, necroptosis directly results in plasma membrane rupture. Repression of necroptosis by apoptotic proteins is essential for proper mammalian development and prevents spontaneous cell death and inflammation, underscoring the physiological relevance of necroptosis. 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 RIP3 kinase activity in necroptosis. The kinase activity of RIP3 is required for downstream signaling events in necrotic cell death. While signal-inducible RIP3 activation mechanisms have been extensively characterized, the regulation process of RIP3 level remain poorly understood. In this seminar, I will discuss recent advances in necroptosis and the mechanisms regulating necroptosis and its potential role in necroptosis-mediated human diseases.

**Keywords: Necroptosis, RIP3, Cell death**