Clec12a regulates inflammation in response to cell death

The innate immune system has the ability to sense and respond to dead cells. Allowing the immune system to control non-infectious inflammatory responses. To better understand the mechanics of this process researchers from the University of Munich identified an inhibitory C-type lectin receptor, Clec12a, as a specific immune receptor for dead cells. Both human and mouse Clec12a was shown to act as a direct sensor for uric acid crystals. Uric acid crystals are key signals that alert the immune system to cell death. Dead cells or uric acid crystals both triggered Clec12a signaling. Furthermore Clec12a-deficient mice exhibited hyperinflammatory responses to uric acid crystals or sterile dead cell challenges, thereby indicating that Clec12a represents a regulatory immune receptor capable of dampening the inflammatory immune response to dead cells. Indicating an important mechanism with the ability to minimize the potential damage during non-infectious inflammation.

Neumann, K. 2014. Clec12a Is an Inhibitory Receptor for Uric Acid Crystals that Regulates Inflammation in Response to Cell Death. *Immunity*.