NLRP3 Inflammasome in Cancer Immunotherapy

Sheeja K
Regional Cancer Centre, India

Abstract:
Modulation of body’s immune responses has emerged as one of the major approaches for treating different types of cancers. Cytokines, the messengers of the immune system and their interaction within the tumor microenvironment determine the fate of tumor cells. Nod-like receptor, one of the pattern recognition receptors forms inflammasome, which is a part of innate immune responses. NLRP3 inflammasome, the most studied inflammasome, is composed of NLRP3; ASC (apoptosis-associated speck-like protein containing a caspase-recruitment domain) which is an adaptor protein encoded by PYCARD; and the precursor procaspase1. On activation, NLRP3 inflammasome convert the inactive IL-β AND IL-18 into their respective active forms. Studies on NLRP3 inflammasome documented that it is a key regulator that determines the entry and exit of immune cells in the tumor microenvironment, which in turn regulate the tumor progression. The interplay between malignant cells and inflammasome complex has a very contrasting role. It exerts procarcinogenic effect by suppressing NK and T cell–mediated anticancer activities. In addition, it stimulates the production of trophic factors that favours the development of secondary tumors. Moreover, this protein complex produces anticancer effects through inflammatory cell death. Hence it is assumed that regulation of NLRP3 inflammasome will help to generate a stable anti and proinflammatory cytokine profile and to regulate the anti-tumor immunity. Thus NLRP3 inflammasome offer novel emerging immunotherapeutic target in cancer therapy.

Biography:
Dr. Sheeja K is currently associated with Regional Cancer Centre, India

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