Abstract

Inflammation shapes immune responses to regulate various physiological and pathological processes. Inflammation serves as an underlying cause for a host of diseases including Alzheimer's, arthritis, diabetes, cardiovascular disease, depression and even cancers. Several exogenous and endogenous inducers of inflammation are being sensed by nucleotide-binding domain, leucine-rich repeat containing receptors (NLRs), specific class of pattern recognition receptors. The NLR family members have strong implications in a wide array of inflammation and autoimmune associated diseases including cancers, due to their close association with major inflammation and immune signaling pathways. NLRs act as critical modulator of inflammation-induced tumorigenesis and the surrounding tumor microenvironment. The cell specific tumor promoting and –inhibitory roles of NLRs, present them as a promising option for advances in early cancer diagnosis, treatment and prognosis. A more in-depth analysis of NLR gene profiling across cancer stages and NLR-mediated cellular and molecular signaling cascade under pathological conditions is of paramount therapeutic importance. The aim of this dissertation was to firstly; elucidate if amorphous nanosilica particles act as DAMPs, to induce inflammation and NLR-mediated innate immune signaling, leading to cytotoxic responses. Secondly, we wanted to characterize NLR gene expression and finally understand if, NLR regulated inflammation pathways could play important role in molecular stratification of low grade glioma and glioblastoma. We have identified novel role of NLRs in glioma, by performing NLR gene expression profiling in different grades of glioma, a highly aggressive malignant brain tumor type. Both *in silico* and *in vitro* complementary approaches have been utilized to evaluate our hypotheses.