3. Aims and Objectives

3.1. Rationale of hypothesis

Abundant evidence supports the view that chronic inflammation contributes to cancer initiation. ER-mitochondrial interface forms a signaling platform that subsequently mounts the inflammatory response against the infection or injury. MAVS (**M**itochondrial **A**nti **V**iral **S**ignaling protein) residing on the outer membrane of mitochondria is regulator of both NF-κB and IFN pathway. ER-mitochondrial interface protein MITA (**M**ediator of **I**RF3 **A**ctivation) has been postulated to be adaptor for MAVS. These proteins are well studied in innate immunity during viral infection; however, role of these proteins in regulation of inflammation in normal patho-physiological condition is not well understood. The role of these proteins in regulation of inflammation in tumor microenvironment has not yet been investigated. Inflammation plays important role in breast cancer pathogenesis however the role of these proteins has not been investigated. IFNs as well as NF-κB aid in tumor development by modulating cell proliferation, death and tumor microenvironment. NF-κB activates a plethora of cytokines that provides suitable microenvironment for tumor development and further favors the process of migration and angiogenesis. We plan to investigate the role of MAVS and MITA in regulation of inflammation in breast cancer. The study will help us understand the linkage between mitochondria, inflammation and cancer. Modulating the inflammatory pathways, and thus NF-κB and IFN, can prove to be an essential therapeutic for breast cancer.

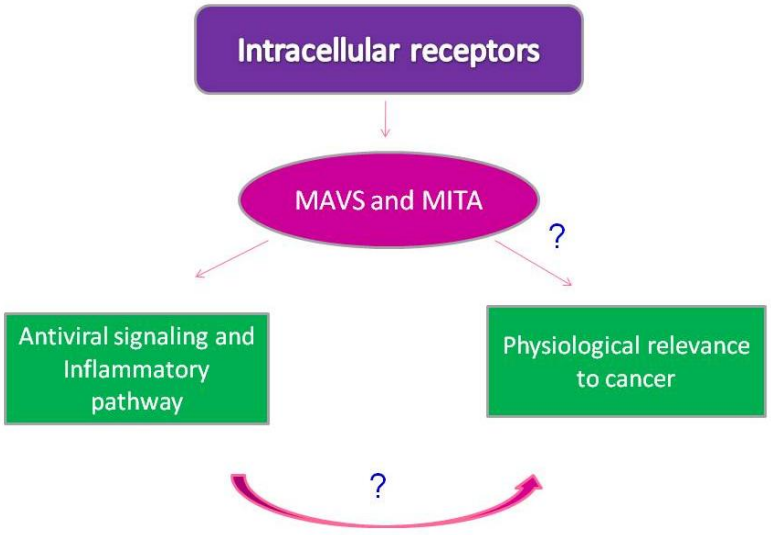

Figure-3.1: Diagrammatic representation of hypothesis of the study

3.2. Objectives

- To study the expression of MAVS and MITA in different breast cancer cell lines as well as patients tissues.
- Study the role of MAVS and MITA in modulation of inflammatory pathway (NF-κB and IFN) in breast cancer cell line.
- Study the crosstalk between MAVS and MITA and its effect on tumorigenesis of breast cancer.