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Nanomedicine in cancer: Nanotherapy and nanodevices

Drug resistance is a common drawback for most chemotherapeutic drugs and it promotes cancer survival and recurrence. The major protein that help a cell acquire MDR is P-glycoprotein (P-gp) and studies have showed that P-gp expression has been directly related to the degree of drug resistance in cells. P-gp also known as ABCB1 acts as a membranous molecular pump that effectively effluxes the chemotherapeutic drugs from within the cells. Overexpression of survivin in cancer cells has also been related to cause resistance to various chemotherapeutic compounds and therapies inhibiting survivin expression have shown sensitization of human cancer cells to various chemotherapeutic drugs such as docetaxel, paclitaxel and bortezomib. Studies have also shown that CD133 positive cancer stem cells resist chemotherapy which is mainly due higher expression of inhibitors of apoptosis protein (IAP) families and it has been also observed that the colony formation of CD133 positive cells is quite higher when compared to CD133 negative cells mainly due to overexpression of survivin. A major area of interest has come up using biomolecules which focusses on specifically target the diseased tissues. In our previous studies we have shown chimeric form of Fe-bLf (LNA-Nucleolin+EpCAM aptamer)-spions showed high specificity towards the tumour both in vitro and in vivo. In another study using ceramic polymer nanocarriers (ACSC NCs) we have shown that Fe-bLF(LNA-EpCAM aptamer+LNAsiRNA(survivin))-ACSC NCs were highly specific to tumour when compared to any other parts of the mice and the nanocarriers led to significant cytotoxicity specifically in tumour cells without harming the primary cells. We have also used novel oligo LNA siRNA (survivin) to target the nanocarriers and inhibit survivin expression in drug resistant cancer stem cells. Nanoparticles loaded with SR9 and LNAsiRNA-5-FU have been used in this study to target cellular survivin in colon cancer cells. Our results show that inhibition of survivin has a direct inhibitory effect on p-gp and CD133. The mechanism for explaining this phenomenon has also been investigated and a pathway has been proposed through which inhibition of survivin significantly lowers both p-gp and CD133 expression in colon cancer cells.

Biography

Jagat Kanwar is group leader and head of the Nanomedicine and Laboratory of Immunology and Molecular Biomedical Research has an international reputation in investigating fundamental and applied molecular aspects of cancer and chronic inflammation. Our nanomedicine laboratory of immunology and molecular biomedical research (NLIMBR) is discovering the novel and safe targeted nanomedicine based nano-nutraceuticals for cancers, autoimmune disorders and inflammatory diseases. We also vested the molecular diagnosis including role of a non-invasive exosomes in blood, inflammatory sites and cancer tissues. Our research focused on cancer and inflammatory autoimmune diseases aims to investigate the underlying mechanisms involved in apoptosis, autophagy and inflammation by targeting the production of cytokines, chemokines, oxygen radicals and matrix metalloproteinase. Our research also aims to investigate the nanotherapeutics encapsulating peptides, LNA modified aptamers/miRNAs/siRNA in vivo models. We have made significant progress in field of ocular drug delivery and microfluidic and Lab-on-a-Chip devices techniques for cancer cells as well as stem cell capture, disease specific biomarkers and exosomes. His publications more than 150 research papers and have added to the body of knowledge in the fields of nanobiotechnology, cancer gene therapy, cell biology and immunology. Kanwar's research work has generated a total of 12 patent/PCTs. He is the member of various scientific committees and societies.

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