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Protein coated gold nanoparticle synthesis: A mathematical model approach for proteasome inhibition

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Cancer is characterized by the uncontrolled cell division and proliferation. As compared to normal cells, the NF- κ B signaling pathway is highly active in multiple myeloma as well as in many other cancers. Inhibition of this pathway has been shown to undermine the survival of myeloma cells, making NF- κ B an attractive therapeutic target. NF- κ B is activated by ubiquitin proteasome complex which ubiquitinates and degrades its inhibitor, I- κ B. Inhibiting I- κ B degradation leaves NF- κ B in an inactive state and hence proteasomal inhibitors have great potential in cancer therapy. The reduction in NF- κ B activity by proteasome inhibition reduces the cell proliferation and induces the apoptosis of multiple myeloma cells. The only proteasome inhibitor, bortezomib had been approved by US – FDA and few drugs under clinical trials to treat multiple myeloma. Here we propose ubiquitin tagged monoclonal antibody to Gold nanoparticles (Ub-AuNPs) with core diameter of <2 nm has comparable dimensions of proteasome subunits, can evolve as potential novel proteasomal inhibitors (PIs). The gold acts as a theranostic agent and it is having excellent biocompatibility where it directly attached to the highly oxidative stress of cancerous cells. In this model as the monoclonal antibody is already ubiquitinated it easily finds its way to proteasome of myeloma cells. Further we use a mathematical model to analyze how these monoclonal antibody associated with gold nanoparticles enter to the target site for proteasome inhibition. And also gives an idea about proposed mechanism of inhibition by invading the proteasome and block the lumen of proteasomes, further inhibiting the degradation of I- κ B. Thus, PIs has emerged as a powerful strategy for treatment of multiple myeloma (MM).

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