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Macrophage activation by immune complex in pigeon fanciers lung

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Pigeon fanciers lung (PFL) is a form of extrinsic allergic alveolis (hypersensitivity pneumonitis) the pathogenesis of the disease is through to be cased by the deposition of immune complexes in the alveoli. Although this is generally agreed in the bionic pathogenesis of disease it is difficult to understand why a large proportion of people with high titres of antibody do not get disease. In this study I firstly showed the optimal concentration of LPS and the optimum time for activation was 10 μ/ml for 24 hours after stimulated the cells by PMA for 72 hours. Immune complexes were generated with mucin, fresh pigeon droppings (PDF), old pigeon droppings (PDO), and patents sera. Immune complexes with PDF and PDO activated macrophages to produce TNFα. However immune complex with mucin did not active macrophages. There was different in the ability of immune complexes from symptomatic and asympomatic individual to activation macrophage.

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Tumor immunotherapeutic potential of *Mycobacteriuam Indicus Pranii* and its underlying mechanisms

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Owing to their immunostimulatory properties, mycobacteria can counter the negative regulatory mechanisms employed by growing tumors, while inducing a potent antitumor immune response. *Mycobacterium indicus pranii* (MIP) is an atypical mycobacterial species possessing strong immunomodulatory properties. Therefore, we evaluated the immunotherapeutic potential of MIP in a mouse tumor model and examined the underlying mechanisms. It was observed that MIP therapy led to a significant tumor regression and enhanced the survival of tumor bearing mice. MIP promoted a tumor specific, T_H1 type of immune response in tumor bearing mice. Higher NK cell and CTL cytotoxicity were observed in MIP-treated tumor bearing mice compared with PBS-treated control mice. MIP therapy also resulted in decreased levels of immunosuppressive CD4+FoxP3+ T regulatory cells in tumor mass and draining lymph node. Next, the role of dendritic cells and macrophages in mediating the MIP-induced antitumor immune response was analyzed. It was observed that MIP led to a significant production of proinflammatory cytokines and upregulation of co-stimulatory molecules by these cells. MIP-activated macrophages lysed the tumor cells in peroxynitrite-dependent manner. MIP promoted dendritic cell survival by inhibiting their apoptosis. MIP-stimulated DCs promoted T_H1 and T_H17 polarization in naïve allogeneic T cells. TLR2 and/or TLR9 were found to play critical role in MIP-induced DC and macrophage activation. Comparable levels of tumor volumes in controls and MIP-treated MyD88 knockout mice demonstrated that the antitumor effects of MIP are mediated by TLRs. Further studies with TLR-knockout mouse strains showed that TLR2 played a key role in MIP-induced tumor regression.

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