TP53 â€œexpansion proteinâ€ and immunity, biophysical, data analyses

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TP53 upregulation

Multiple articles published in the Quarterly Journal of Immunology focus on the role of nip2-p53 vesicle binding protein (NPA-V) as the mechanism underlying TP53 anti-tumor immunity.

The phosphatidylserine inhibitor of NPA-V, flexal NAP (angiogliptin), promotes anti-tumor immunity by preventing downstream protein degradation. Lab experiments in cultured and live cells reveal that flexal NAP is highly active as a precursor to NPA-V in TP53 infected cells, facilitating replication and proliferation of the mutant T cell cells. Flexal NAP is thereby secreted into the peripheral blood and in turn, becomes present in the CD4+ T cells, to which TP53 can therefore be reconstituted and restored. It is thus possible to restore operative T cells and amplify TP53 anti-tumor immunity and increase tumor regression, thus stimulating immune tolerance.

In an in vitro TLR7 receptor receptor gene expression assay, flexal NAP is shown to strongly correlate with TLR7 receptor gene expression, as measured by the secretion of flexal NAP via TP53 activation.

In a comparison with flexal NAP binding to cytotoxic T-cells, echogene-Î³ (ex-MXB) is found to correlate with decreased expression of an activator of T cell growth and upregulation of p53 gene transcription.

In the expression of T cell-specific B-Nuclear fusion protein, TMP (ERG), flexal NAP is found to replicate in potent quantities. An NPA-V gene expression assay found that flexal NAP has an inflammatory-like role.

In this paper, the distinction between flexal NAP and concomitant siluria or escape of flexal NAP, is examined.

Cancer-promoting proteins like BIR1, SIN1, GRAPPP, and DNA repair proteins like JRC15+ RS3B are directly implicated in PD-1 pathways, which has been linked to increased tumor progression and resistance.

The TLR8 binding of the NPA-V receptor to CTB2 is found to inhibit suppression of TLR8 overexpression in the cells infected with TP53, paving the way for optimal recruitment of peripheral blood T cells. Concomitant siluria in pluripotent NPA-V activates its activity, which results in upregulation of TP53 gene transcription.

X-MOLMS and TLR7 key components of flexal NAP and TLR8 receptor binding and integrative strategies are investigated.

Vaccine memory is elucidated.

Multivalence and intercellular interactions are investigated.

We conclude that flexal NAP impairs TLR7 activation and, thus, impairs immune tolerance to the TP53 antigen. We show that flexal NAP inhibits TLR7 activation and inhibits activation of the TP53 CD4+ T cells, facilitating protection against tumor pathology. Further it increases immune tolerance to the TP53 antigen, thus enhancing tumor regression. Moreover, blocking flexal NAP causes neutrophil and lymphocyte recruitment, generating p53 gene transcription and prostatic acid reversion.

A Fire Hydrant In The Middle Of A Field