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Enhancement of the Proapoptotic Properties of Newcastle Disease Virus Promotes Tumor Remission in Syngeneic Murine Cancer Models

Sara Cuadrado-Castano, Juan Ayllon, Mena Mansour, Janis de la Iglesia-Vicente, Stefan Jordan, Shashank Tripathi, Adolfo Garcia-Sastre, and Enrique Villar

RNA Interference Using c-Myc–Conjugated Nanoparticles Suppresses Breast and Colorectal Cancer Models


The newly generated Newcastle disease virus rNDV-B1/Fas encodes the human TNF receptor Fas. Human and murine cancer cells infected by rNDV-B1/Fas displayed a modified cell death response compared with the wild-type rNDV-B1, characterized by an earlier and enhanced apoptosis response due to the overexpression of Fas following the coactivation of both extrinsic and intrinsic apoptosis pathways. The enhanced cytotoxicity shown in vitro correlated with an improved oncolytic activity and therapeutic effect of rNDV-B1/Fas virus in intratumoral-treated melanoma-bearing mice. The cover image shows murine B16-F10 melanoma cells infected by rNDV-B1/Fas virus. The human Fas receptor (red) is expressed on the surface of infected cells (green) but also internalized following induction of apoptosis. DNA was counterstained with Hoechst. For details, see the article by Cuadrado-Castano and colleagues on page 1247.

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