
Harnessing ERV Expression to Ignite Anti-Tumor Immunity

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Abstract

Endogenous retroviruses (ERVs), integrated remnants of ancestral viral infections, are largely epigenetically silenced in the human genome to circumvent potential harm. Nonetheless, accumulating evidence now posits the de-repression of ERVs as a potential weapon against cancer. This is based on the ability of ERV-derived elements to stimulate antiviral signalling, and consequently drive immune responses. Here, I will highlight the role of TRIM28, a pivotal regulator of ERV silencing, as a potential target for cancer treatment. Our approach utilizes TRIM28 genetic silencing techniques alongside TAK-981, an innovative SUMOylation inhibitor that blocks TRIM28's SUMOylation. The SUMOylation of TRIM28 is a critical mechanism involved in the recruitment of epigenetic silencing complexes that inhibit ERVs. Overall, I will present the possibility of targeting TRIM28 to derepress ERV expression and trigger ERV mediated activation of IFN/NF- κ B signalling and necroptosis in the cancer setting.

Keywords: ERVs, immunogenic cell death, innate immune signaling, Z, RNA, cancer

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