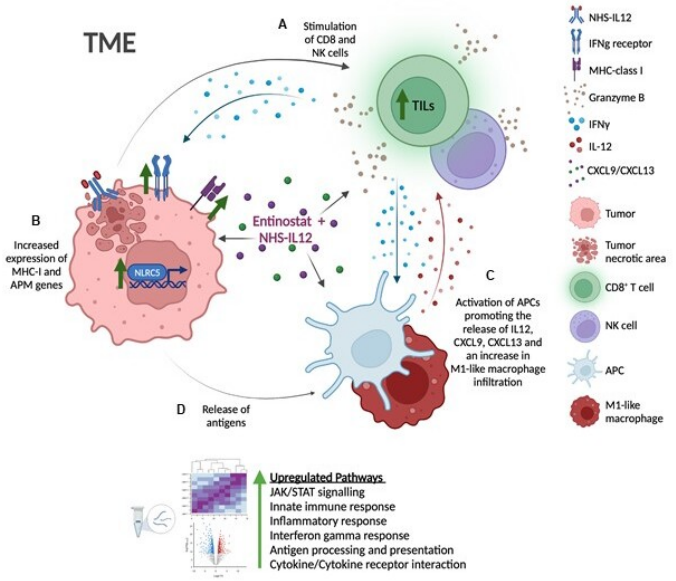
Tumor-targeted interleukin-12 synergizes with entinostat to overcome PD-1/PD-L1 blockade-resistant tumors harboring MHC-I and APM deficiencies



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In Brief
Entinostat and NHS-IL12 combination elicits potent anti-tumor activity against tumors with MHC-I, antigen processing machinery (APM), and IFNγ response deficiencies, and differing tumor mutational burden by activating an IFNγ and IL-12 feedback loop thereby increasing cytotoxic $CD8^+$ T cell, M1 macrophage and antigen presenting cell (APC) infiltration into the tumor microenvironment (TME).