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Resumo	Exportin 1 (XPO1) is the main nuclear export receptor that controls the subcellular trafficking and the functions of major regulatory proteins. XPO1 is overexpressed in various cancers and small inhibitors of nuclear export (SINEs) have been developed to inhibit XPO1. In primary mediastinal B-cell lymphoma (PMBL) and classical Hodgkin's lymphoma (cHL), the XPO1 gene may be mutated on one nucleotide and encodes the mutant XPO1E571K . To understand the impact of mutation on protein function, we studied the response of PMBL and cHL cells to selinexor, a SINE, and ibrutinib, an inhibitor of Bruton tyrosine kinase. XPO1 mutation renders lymphoma cells more sensitive to selinexor due to a faster degradation of mutant XPO1 compared to the wild-type. We further showed that a mistrafficking of p65 (RELA) and p52 (NFκB2) transcription factors between the nuclear and cytoplasmic compartments accounts for the response toward ibrutinib. XPO1 mutation may be envisaged as a biomarker of the response of PMBL and cHL cells and other B-cell hemopathies to SINEs and drugs that target even indirectly the NFκB signaling pathway.
Fomento	