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New Strategy for Overcoming ATO-resistant APL

Chimeric fusion proteins are common oncogenic drivers in multiple cancers. The PML/RARα oncofusion dysregulates differentiation and self-renewal of myeloid progenitors, resulting in the onset and progression of acute promyelocytic leukemia (APL). Arsenic trioxide (ATO), which could target and destabilize PML/RARα oncofusion protein, is one of the most effective drugs for APL therapy. Nevertheless, the relapse and arsenic-resistance APL remain a challenging problem in clinic treatment. Here, we for the first time reported that our new strategy could destabilize PML/RARα oncofusion protein as well as its Arsenic-resistant mutants (A216, P218L) in vitro and in vivo. Collectively, we provided a new strategy that may improve therapeutic efficacy in arsenic-resistant or refractory APL patients by taking advantage of a biophysical vulnerability of PML/RARα protein.

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