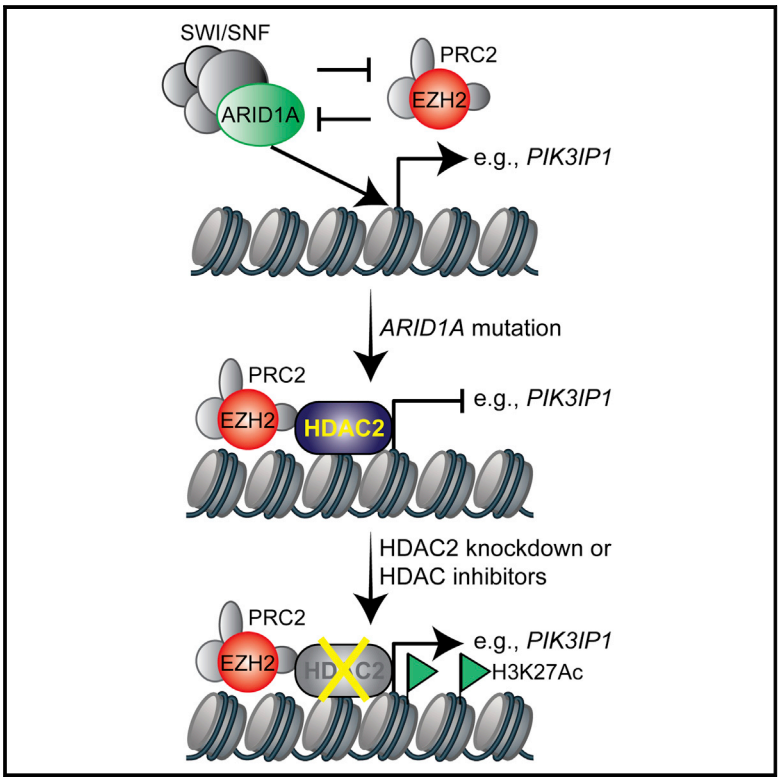
Cell Reports

Report

Repurposing Pan-HDAC Inhibitors for *ARID1A*-Mutated Ovarian Cancer

Graphical Abstract



Highlights

- ARID1A inactivation enhanced growth suppression induced by HDAC2 inhibition
- HDAC2 interacts with EZH2 in an ARID1A status-dependent manner
- HDAC2 functions as a co-repressor of EZH2 to promote apoptosis
- SAHA improved the survival of mice bearing *ARID1A*-mutated cancer

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In Brief

Fukumoto et al. show that *ARID1A* mutation confers sensitivity to pan-HDAC inhibitors such as SAHA in ovarian cancers. This correlated with enhanced growth suppression induced by the inhibition of HDAC2 activity in *ARID1A*-mutated cells. These findings provided preclinical rationales for repurposing FDA-approved pan-HDAC inhibitors for treating *ARID1A*-mutated cancers.

Data and Software Availability

GSE107201

Fukumoto et al., 2018, Cell Reports *22*, 3393–3400
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