Chidamide re-sensitized chemo-resistant B cell lymphoma to chemotherapy agents

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Purpose
Chemo-resistant B cell lymphoma patients have a poor clinical outcome with current available treatments. HDAC inhibition is an attractive strategy to evaluate its anti-proliferation activity in cancer cells. Since chidamide is a novel histone deacetylase (HDAC) inhibitor of the benzamide class, we studied its anti-proliferation activity and mechanisms in chemotherapy resistant patients.

Methods
MTT assay, cell cycle arrest and apoptosis assay by flow cytometry were performed to study cell proliferation, cell cycle arrest and cell apoptosis. RNA-sequence were used to analysis RNA expression.

Results
Chidamide induced dose-dependent cell proliferation inhibition in both chemo-resistance (Raji-4RH) and their parental cells (Raji). In addition, in vitro exposure to chidamide induced G1 cell cycle arrest in both Raji and Raji-4RH cell lines, while apoptosis induced by chidamide was tested in Raji-4RH but not Raji cells.

In the future, we aimed to study chidamide and chemotherapy agents synergistic antitumor effect in vivo. Additionally, the role of E2F pathway involved in chidamide drug efficacy was also of interest.