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Genome-wide CRISPR screening identifies critical role of phosphatase and tensin homologous (*PTEN*) in sensitivity of acute myeloid leukemia to chemotherapy

Key words: **Genome-wide CRISPR library, *PTEN*, Rapamycin, AML, Chemotherapy**

Research Summary

This article aims to identify the genes playing a crucial role in AML sensitivity to NL101 by utilizing genome-wide CRISPR library screening in AML cells:

- **Antileukemia activity of NL101 is conducted in vitro and in vivo.**
- **CRISPR library screening identifies genes critical for drug resistance to NL101.**
- **Gene knockout confers resistance to NL101.**

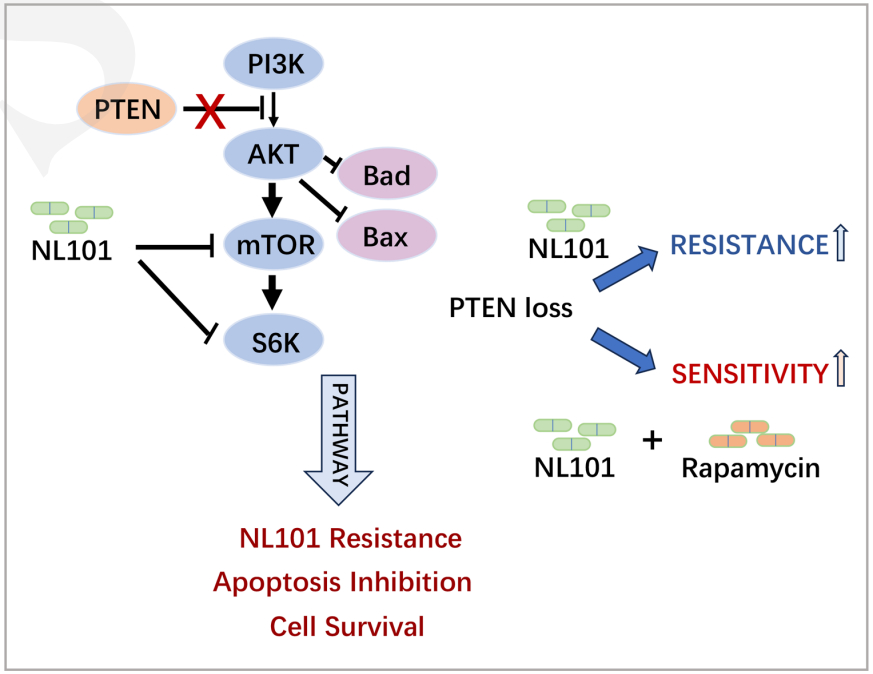

Fig. 5

Innovation points

- **Illustrating a novel small compound NL101 inhibits the proliferation of myeloid cancer cells in vitro and in vivo.**

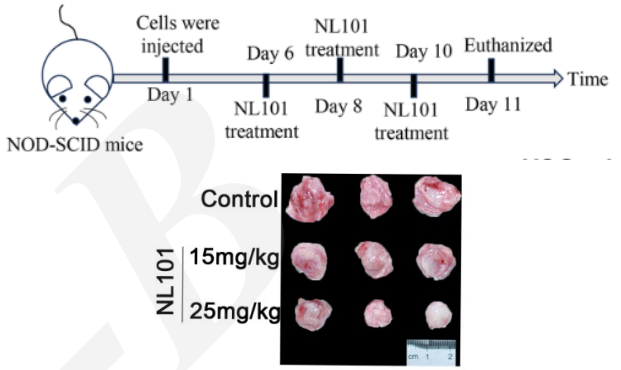

Fig. 1

- **Demonstrating PTEN expression as a major determinant of chemosensitivity to NL101.**

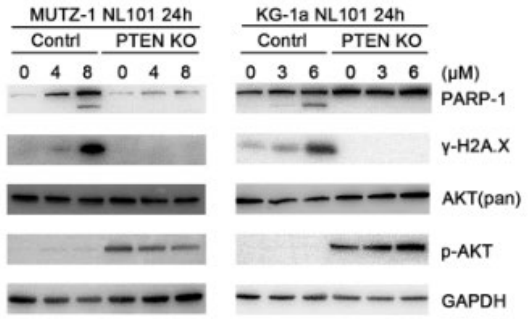

Fig. 3

- **Demonstrating that rapamycin can sensitize myeloid cancer cells to NL101-induced cytotoxicity.**

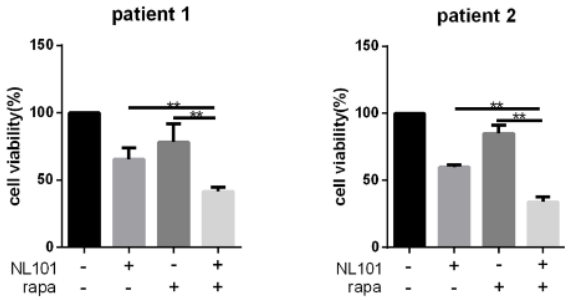

Fig. 4