Background

Trilaciclib is a highly potent, selective, and reversible cyclin-dependent kinase 4 and 6 (CDK4/6) inhibitor in development to preserve hematopoietic stem and progenitor cells (HSPC) and immune system function during chemotherapy (myelopreservation).

To prevent myelosuppression, administration of trilaciclib prior to chemotherapy has been shown to induce transient cell cycle arrest of hematopoietic progenitors through unknown mechanisms, such as S/G2/M arrest. Because of this potential mechanism of action, trilaciclib was studied in preclinical models with CDK4/6-dependent tumors in xenografts and PDX models to determine the potential for myelopreservation.

Results

To determine whether transient CDK4/6 inhibition with trilaciclib prior to chemotherapy administration antagonizes the intended anti-tumor effects of the chemotherapy in CDK4/6-dependent tumors.

Conclusions

Trilaciclib, a highly potent, selective, and reversible CDK4/6 inhibitor, does not impair the efficacy of chemotherapy in CDK4/6-dependent tumor models.