DPY30 loss leads to DNA re-replication and immunoediting in pancreatic ductal adenocarcinoma

1. DPY30 expression associates with poor prognosis

2. In mouse model of PDAC, DPY30 expression associates with tumor grade
3. DPY30 loss favorites uncoordinated DNA replication

4. DPY30 loss induces DNA damage and chromosomal instability
5. DPY30 loss impairs tumor growth only in immune-competent mice

6. DPY30 knockout tumors display higher CD8+ T cell infiltration and respond better to anti-PD-1

**Conclusions:** our findings indicate that, in PDAC, DPY30 promotes genome stability, thus providing a rationale for targeting DPY30 or its effector proteins in combination with immune-checkpoint inhibitors.

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