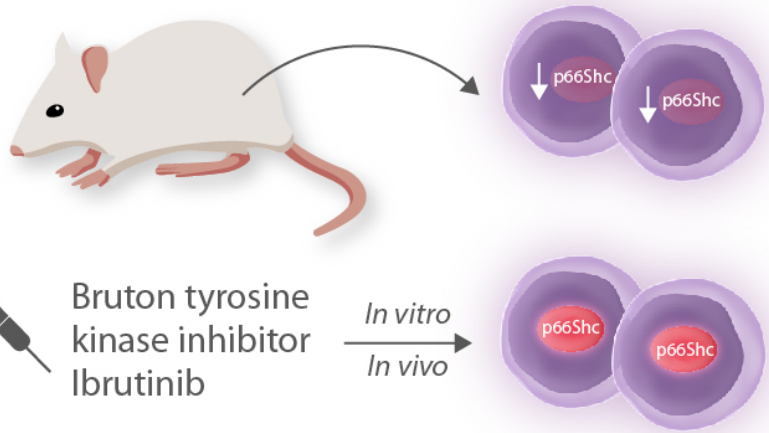
Comparison of p66Shc defect and p66Shc deficiency in two mouse models of chronic lymphocytic leukemia

Mouse models of chronic lymphocytic leukemia (CLL)

Eμ-TCL1

Leukemic cells

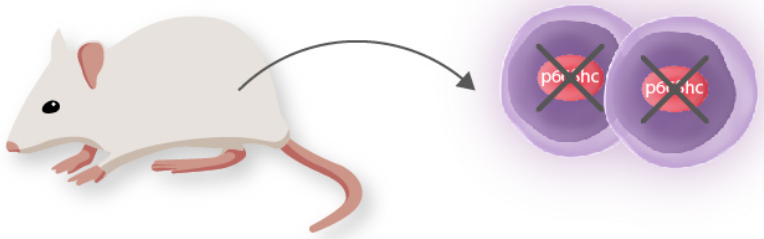


p66Shc defect

- Downregualtion of p66Shc in tumoral B cells compared to normal B cells
- p66Shc expression declines during disease progression

- Restoration of p66Shc expression

Eμ-TCL1/p66Shc$^{-/-}$



p66Shc deficiency

- Significantly higher disease incidence compared to Eμ-TCL1 mice
- Earlier disease onset (~2 months earlier) and shorter lifespan
- Reduced sensitivity to fludarabine treatment
- Increased nodal and extranodal leukemic cell accumulation

p66Shc deletion in Eμ-TCL1 mice results in accelerated leukemogenesis and enhanced disease aggressiveness, with massive nodal and extranodal infiltrations

Patrussi *et al.*, Haematologica, 2019