iNUP98-KMT2A has in vivo transforming activity and interferes with cell cycle progression rather than primarily blocking differentiation

1. Expression of iNUP98-KMT2A leads to expansion and competitive advantage of hematopoietic stem and progenitor cells

2. Some iNUP98-KMT2A mice develop transplantable AML

3. Expression of iNUP98-KMT2A results in aberrant cell cycle progression and escape from senescence

4. AML cells do not express the HoxA-B-C gene cluster and are resistant to compounds targeting the KMT2A-menin interaction

Fisher et al., Haematologica, 2020