

Extended Data Figure 6 | Cell-intrinsic activation of Wnt signalling cascade in TIS. a, b, Expression of indicated stem-cell-related transcripts by qPCR (a) and colony formation (b) in control; Bcl2 lymphomas infected with a constitutively active Ctnnb1 mutant ($^{\Delta N}$ Ctnnb1) or a mock retrovirus. Data represent mean expression fold change normalized to mock-infected cells and mean colony numbers, respectively \pm s.d. (n=3 biologically independent samples). Two-tailed, unpaired t-test with Welch's correction. *P < 0.05. c, Immunoblot analysis of Ser9-phosphorylated (that is, inactivated) or total GSK3 β , active or total β -catenin (as in Extended Data Fig. 5c), Thr202- and Tyr204-phosphorylated or total Erk1/2, and Ser473-phosphorylated or total Akt in control; Bcl2 lymphoma cells treated with ADR for five days, together with pharmacological inhibitors targeting MAPK and PI3K kinase pathways.

 α -Tubulin was used as a loading control. One out of two independent experiments shown. For gel source data, see Supplementary Fig. 1. \mathbf{d} , Expression of the indicated stem-cell-related transcripts by qPCR in never senescent and previously senescent Suv39h1-; Bcl2;Suv39h1-ER T2 cells (passage 2) exposed to Wnt signalling agonists (Wnt3a, Rspo2, or GSK3 β inhibitor) for two days. Colour scale represents mean fold change normalized to never senescent cells not exposed to Wnt agonists \pm s.d. (n=3 individual lymphomas). \mathbf{e} , Colony formation of never senescent and previously senescent cells (as in \mathbf{d}), after seven days in methylcellulose medium supplemented with the indicated Wnt agonists (mean colony numbers \pm s.d., n=3 individual lymphomas). Two-tailed, unpaired t-test with Welch's correction. *P < 0.05.