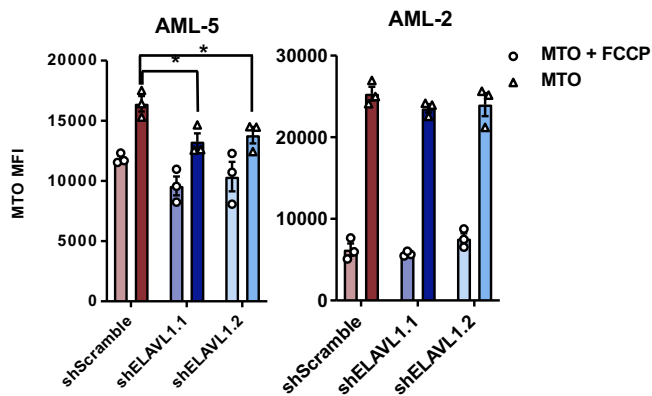
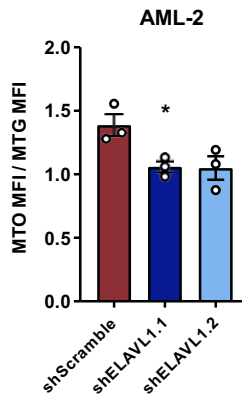


Supplemental Figure S7

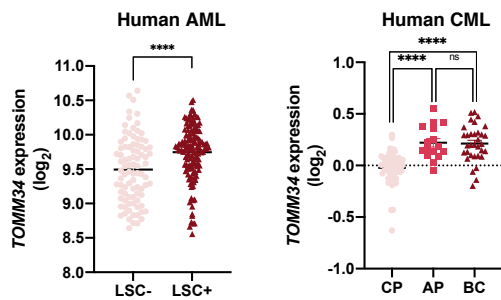
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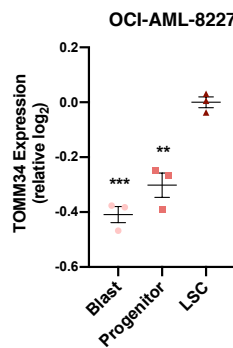
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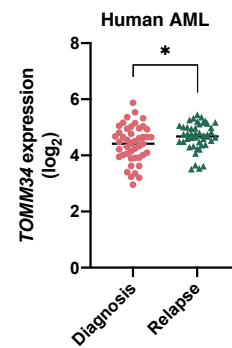
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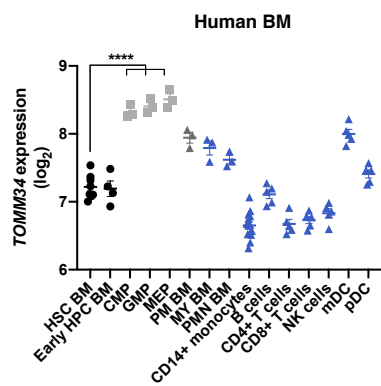
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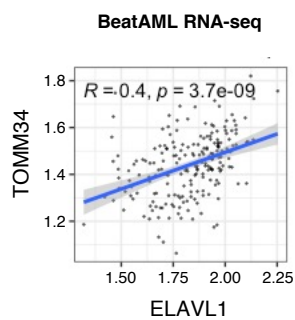
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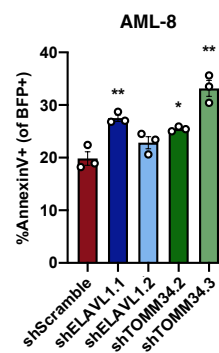
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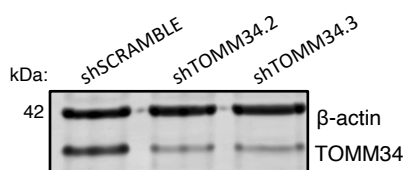
G



H



I



J



Supplemental Figure S7: TOMM34 depletion in human primary AML phenocopies ELAVL1 and impairs mitochondrial function. (A) Quantification of MTO median fluorescence intensity in the presence and absence of FCCP in shScramble- or shELAVL1-infected human AML. (B) Membrane potential as measured by the fraction of active mitochondria (MTO MFI) from total mitochondrial mass (MitoTracker Green (MTG)) in human AML upon ELAVL1 loss. (C-F) Expression of *TOMM34* in human AML LSC⁺/LSC⁻ subfractions (C, left panel)²⁷, throughout human CML disease stages (adapted from Radich *et al.* 2006 and www.oncomine.org) (C, right panel), in subpopulations of the OCI-AML- 8227 cell line (D), in paired diagnosis-relapse human AML (E), and in sorted subfractions of the human BM hematopoietic hierarchy (F), adapted from BloodSpot43. Mature hematopoietic lineages, progenitor populations and primitive populations are shown in blue, grey and black, respectively. (G) Correlation of TOMM34 and ELAVL1 expression in the BeatAML dataset. (H) Apoptotic analysis of ELAVL1- and TOMM34-depleted human primary AML cultures. (I,J) Western blot validation of TOMM34 knockdown in K562 (I) and overexpression in THP-1 (J) 4 days post-infection.