

# Comprehensive multi-omics analysis categorises distinct pathogenic processes in CLL

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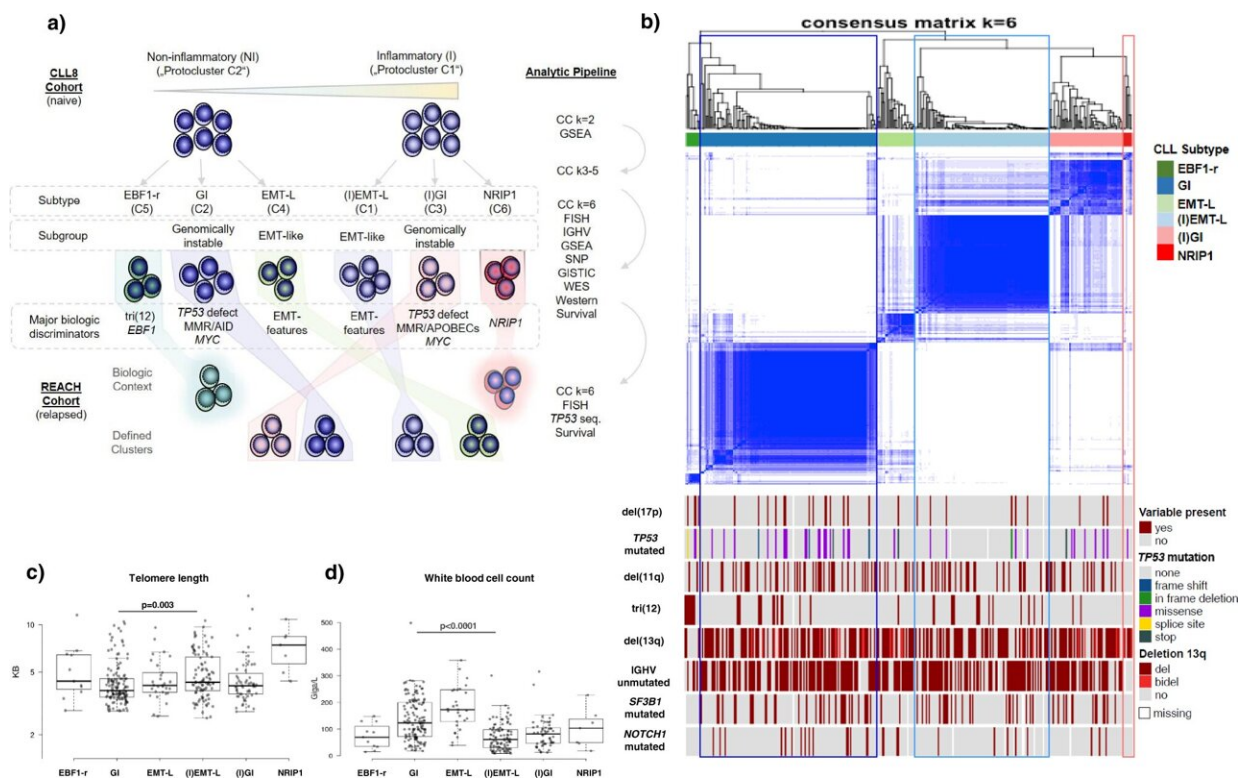


Fig. 1: Composition and relationship of CLL subtypes in clustered data. a Schematic representation for analysis, identification of CLL subtypes in the CLL8, and confirmation in the REACH cohort. The four largest clusters (GI, (I)GI, EMT-L, (I)EMT-L), and associations of NRIP1 with the inflammatory or tri(12) with the EBF1-r signature were also identified in the independent validation cohort of the REACH trial. Co-clustering of GI/(I)GI and EMT-L/(I)EMT-L cases in the REACH cohort supports the selection of subgroup-specific characteristics during treatment. b Heatmap showing the consensus

clustering for  $k = 6$  used for defining CLL subtypes ( $n = 337$ ). The distribution of genetic characteristics is shown below the heatmap. Significant enrichment of variables in clusters is observed for  $\text{del}(17p)$  ( $p = 0.05$ ), TP53 mutation ( $p = 0.01$ ),  $\text{tri}(12)$  ( $p = 7e-06$ ),  $\text{del}(13q)$  ( $p = 0.03$ ), and IGHV mutation status ( $p = 0.008$ ) (all Fisher's exact test (two-sided)). TP53 frameshift mutations occur exclusively in GI and splice site mutations in EBF1-r cases. Tri(12) is strongly overrepresented in EBF1-r (72.7%). c Telomere length is significantly different across CLL subtypes ( $p$

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