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Oral presentation

Core stemness mechanisms revealed through homology

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The "stemness" hypothesis states that different types of stem cells share a core set of mechanisms that regulate the shared stem cell properties of self-renewal and multi-lineage potential. Previous attempts to identify genes required for core stem cell function across stem cell types using transcriptional profiling have identified few such genes. We hypothesized that functional redundancy and tissue-specific expression of functionally redundant homologs mask common stem cell mechanisms. Using an unbiased, genome-wide computational screen of many publicly available mouse stem cell profiling experiments, we tested for shared differential expression across different stem cell types accounting for the possible tissue-specific expression of gene homologs. We found 103 evolutionarily related groups of homologous genes with reproducible, statistically significant, cell type diverse and stem cell-specific upregulation in multiple stem cell types. Shared homolog groups include previously identified self-renewal genes in the Myc, Myb, Chd and Cip/KIP families, as well as genes newly implicated in stem cell function. Our results suggest that different stem cells express distinct repertoires of genes that are functionally synonymous and point to specific examples of functional redundancy in pathways controlling cell adhesion, quiescence, and gene silencing. Genes within these homolog families are prime candidate regulators of conserved stemness mechanisms and may play critical roles as stem cell markers.