



Supplementary Figure 1: Mutant mice ($Selm^{-/-}$) were generated from C57BL/6 embryonic stem cell clones 11537A-A8 and 11537A-B11 (referred to as AA8 and AB11) by injection into blastocysts from C57BL/6 mice with genetically deleted *Selm* obtained from the knockout mouse project (KOMP) repository (an NCCR-NIH-supported strain suppository). ES-cells were created by VelociGene from funds provided by the trans-NIH KOMP. Subsequent intercrosses of heterozygous animals generated two congenic strains with a C57BL/6 genetic background. **(A)** The *Selm* ORF was deleted and replaced by a ZEN-Ub1 cassette harbouring the reporter and neo resistance gene. Genotyping primers are indicated by arrows. lacZ: β -galactosidase coding sequence from *E. coli* lacZ gene, hUbCpro: promoter from the human Ubiquitin C gene, neo^r: coding sequence for neomycin phosphotransferase. **(B)** Southern blot analysis of genomic DNA from the kidney of wild type (+/+) and $Selm^{-/-}$ mice (AA8/AB11) using a probe against a 493 bp fragment of the LacZ gene from the knockout cassette (5'-gccaggcacagatgggtaccg-3' and 5'-taaccgaccagcgcccggtt-3'). **(C)** Genotypes were determined by multiplex PCR using different primer pairs for wild type (-/-; 583 bp) and knockout (-/-; 392 bp) mice (wild type: 5'-tcagccaaatgaccgggacg-3' and 5'-ctgccccgtctgtcaaacacc-3'; knockout: 5'-ttgggtgcagcctgcggaa-3' and 5'-ttctccgtgggaacaaacgcg-3'). +/-, heterozygous mice. **(D)** RNA *in situ* hybridization on hindlimb sections of postnatal day 4 wild type (+/+) and $Selm^{-/-}$ (-/-) mice. 5 μ m paraffin sections were hybridized with antisense riboprobes against *Selm*; scale bar: 0.5 mm. **(E)** *Selm* expression of wild type (+/+) and $Selm^{-/-}$ (-/-) P4 calvarial osteoblasts relative to *Actb* expression (mean + S.D., n = 4).