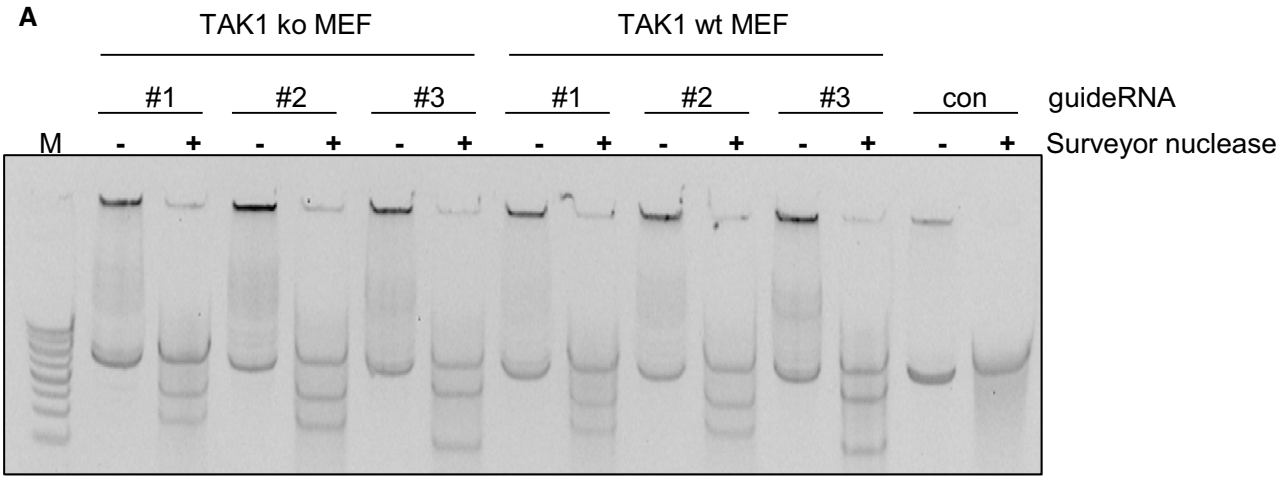
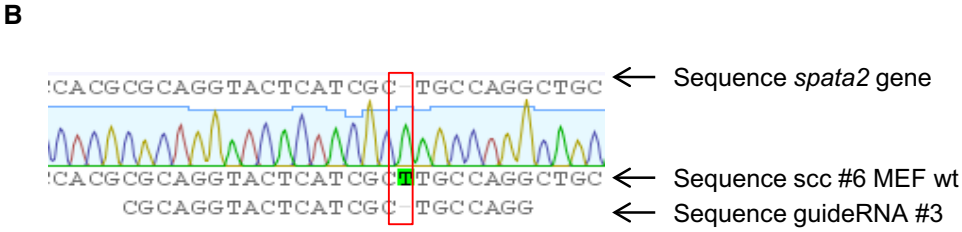
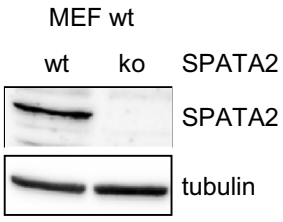
Expanded View Figures







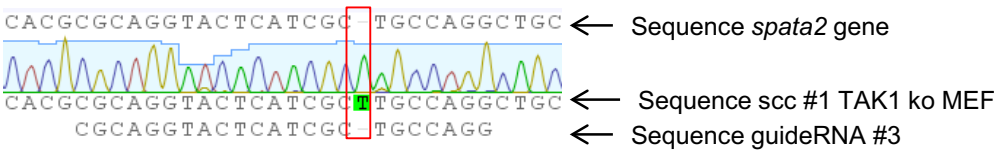


Figure EV1. CRISPR/Cas9-mediated knockout of SPATA2 in wild-type and TAK1$^{-/-}$ MEFs.

A Surveyor assay shown for mixed cell cultures generated with different guide RNAs.

B Sequencing of a single cell clone of wild-type MEFs, generated by CRISPR/Cas9 targeting the *Spata2* gene, with an identical deletion on both alleles, creating a premature STOP codon.

C Western blotting of control cells generated by CRISPR/Cas9, targeting the luciferase gene or a *Spata2* knockout clone of wild-type MEFs, as described in (A).

D Sequencing of a single cell clone of TAK1$^{-/-}$ MEFs, generated by CRISPR/Cas9, targeting the *Spata2* gene, with an identical deletion on both alleles, creating a premature STOP codon.

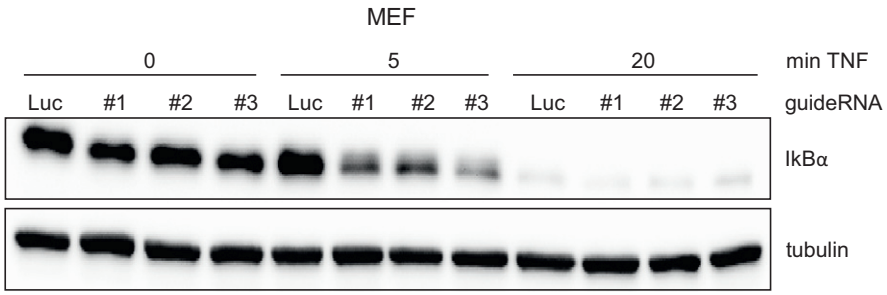
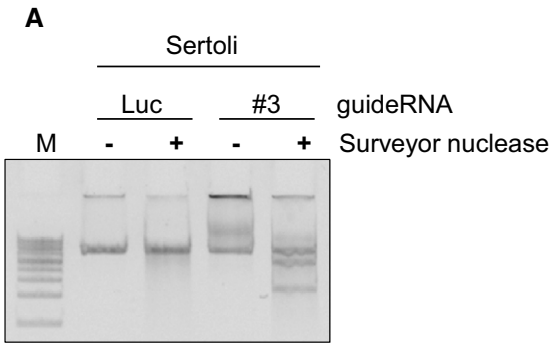


Figure EV2. Increased IκBα degradation in various SPATA2 knockout clones generated with different guide RNAs.
Control cells generated by CRISPR/Cas9 targeting the luciferase gene (Luc) or various SPATA2 knockout clones generated with different guide RNAs (#1–3) were treated with TNF (10 ng/ml) for the indicated time and IκBα degradation was assessed by Western blotting, followed by normalization with anti-tubulin.



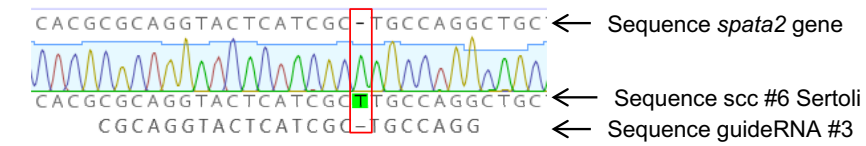


Figure EV3. CRISPR/Cas9-mediated knockout of *Spata2* in 15P-1 Sertoli cells.

A Surveyor assay shown for a mixed cell culture generated with guide RNA #3.

B Sequencing of a single cell clone, generated by CRISPR/Cas9, targeting the *Spata2* gene, with an identical deletion in both alleles, creating a premature STOP codon.