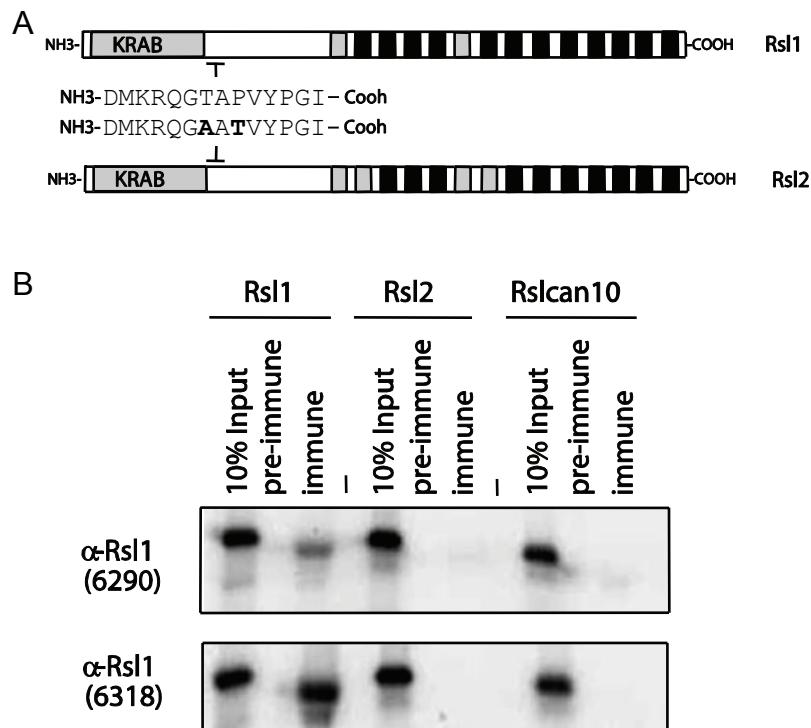
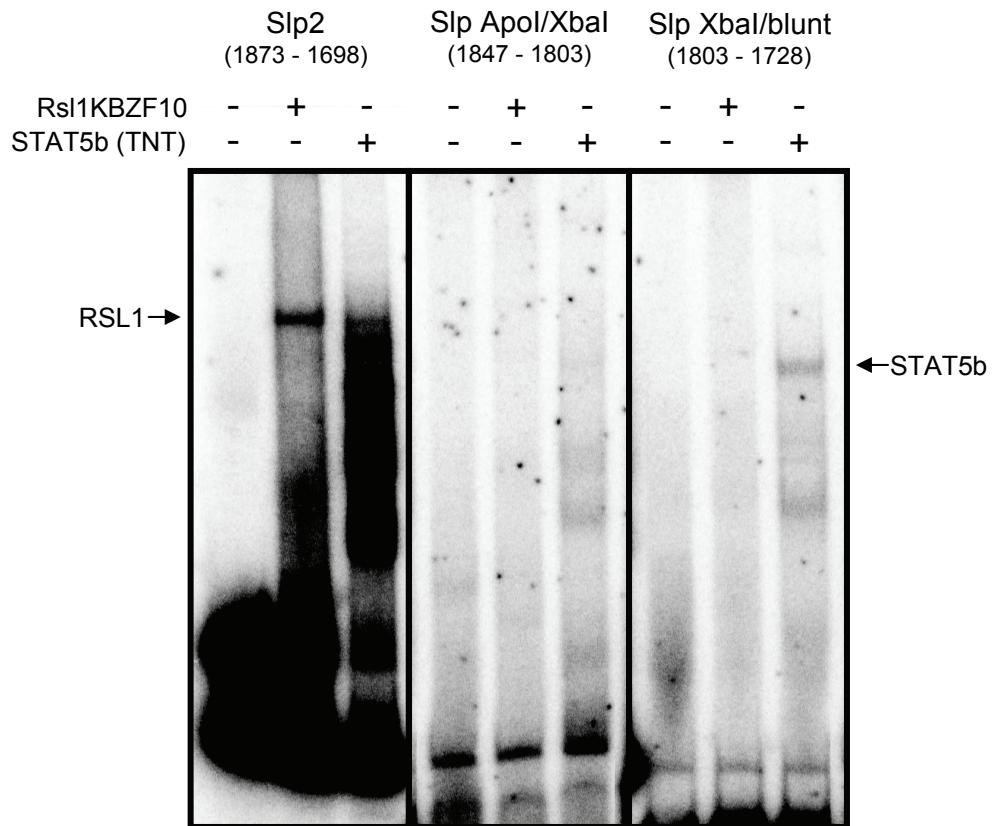


Supplemental Fig. 1 - *Rs1* is expressed in liver and kidney.
Rs1 mRNA was measured by real time qRT-PCR in liver and kidney of WT males and females. For comparison of relative expression across sexes and tissues, all data was normalized to liver expression in WT males. Bars indicate the mean \pm SEM; n \geq 3 mice per sex.



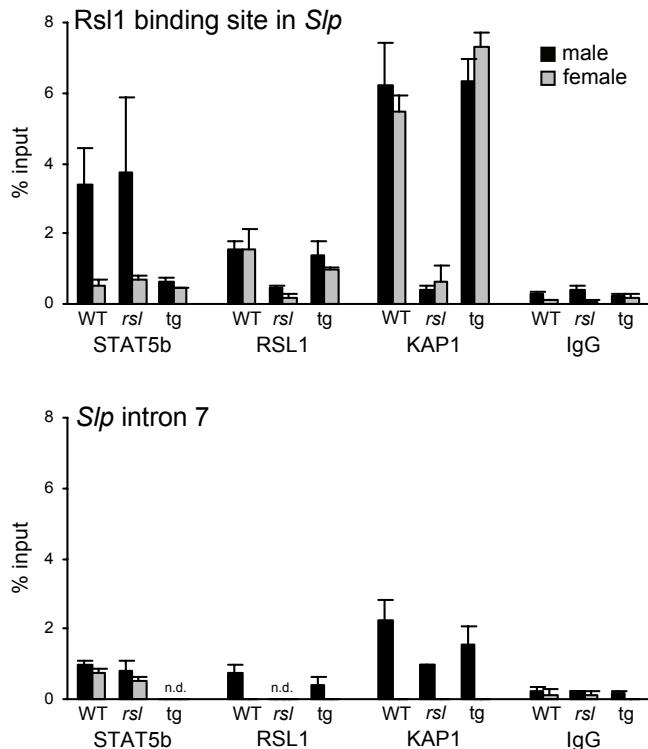
Supplemental Fig. 2 - RSL1 antibody specificity. A) Schematic diagram illustrating domain structure of RSL1, including position and sequence of peptide antigen. Amino acid differences between RSL1 and RSL2 are indicated in bold. Black boxes: zinc finger modules; Grey boxes: degenerative zinc fingers. B) Autoradiograph of immunoprecipitated ³⁵S-labeled *in vitro* translated RSL1, RSL2, and Rslcan10 with anti-Rsl1 serum.



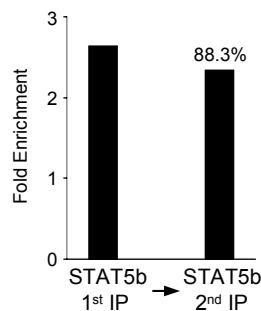
The diagram illustrates the STAT5 promoter region with the following features:

- Apal:** A site at position -1905 is marked with a black dot.
- XbaI:** A site at position -1905 is marked with a black dot.
- SmaI:** A site at position -1905 is marked with a black dot.
- Transcription Start Sites:**
 - ab:** Located between the Apal and XbaI sites.
 - bc:** Located upstream of the Apal site.
 - cd:** Located downstream of the XbaI site.
 - 1905:** Located upstream of the Apal site.
- STAT5 consensus:** The sequence is shown as **CCAGAA**CCCC CAAAATGGCG CTCAAATAGTT TATAACTCCA GTTTCAGGGG ACCCAGTACC.
- Start Site -1726:** Located downstream of the SmaI site.

Supplemental Fig. 3 - RSL1 and STAT5b bind different regions of Slp2. To narrow the area required for RSL1 binding within Slp2, the Slp2 probe DNA was cleaved into restriction fragments (Apol/XbaI and XbaI/blunt) that were then used individually as EMSA probes. Despite binding to the full-length Slp2 probe (*left panel*), RSL1 was unable to bind the Apol/XbaI or the XbaI/blunt fragments. STAT5b binding was evident with the XbaI/blunt fragment (*right panel*), which contains the canonical STAT5 response element. Below is the sequence flanking the RSL1 binding site in the *Slp* enhancer. Restriction sites and the canonical STAT5 response element are in bold. Numbers indicate distance in base pairs from the *Slp* transcription start site. Lines below the sequence represent overlapping EMSA probes ab, bc and cd.



Supplemental Fig. 4 - STAT5b, RSL1 and KAP1 bind upstream of *S/p* in male and female mouse liver. Chromatin immunoprecipitation (ChIP) analysis is plotted to compare males with females. *Top* - ChIP assays were performed for STAT5b, RSL1 and KAP1 on liver from adult male and female WT, *rs/l* and *Rs/l^{tg}* mice ($n \geq 3$) using primers flanking the RSL1 binding site as defined in Fig. 4. *Bottom* - Quantitative PCR of ChIP DNA from mice shown above amplified with primers located within intron 7 of *S/p* (~ 10 kb downstream from the RSL1 binding site). n.d. = not determined. IgG was used as a negative control. Bars are the mean of the % input \pm SEM.



Supplemental Fig. 5 - STAT5b is re-precipitable in sequential ChIP (Re-ChIP) using primers flanking the RSL1 binding site. Re-ChIP was performed on WT male liver chromatin with the STAT5b antibody in both the first (*left*) and second (*right*) immunoprecipitations (IP). Graphed is the fold enrichment relative to the IgG negative control. Number indicates the percent of STAT5b-containing complexes from the first reaction that were recovered in the second.