Supporting Information

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SI Materials and Methods

Mice and Treatment. The breeding diet (D03) contained 25,000 UI of vitamin A per kilogram (UAR) and was provided ad libitum. For vitamin A depletion studies, *Rbp4*-null mice were fed the breeding diet from weaning to 6 wk of age and then a VAD diet (TD86143; Harlan Laboratories) for 14 wk, as described (1). Mice bearing *lox*P-flanked (L2) alleles of *Aldh1a1* (2), *Aldh1a2* (3), and *Aldha3* (4), as well as *Amh-Cre* transgenic mice (5), were genotyped as described. Males carrying L2 alleles of *Aldh1a1*, *Aldh1a2*, and *Aldha3* (i.e., *Aldh1a1-3*$^{L2/L2}$) were crossed with females bearing both one copy of the *Amh-Cre* transgene and L2 alleles of *Aldh1a1*, *Aldh1a2*, and *Aldha3* (i.e., *Amh-Cre*$^{tg/0}$/*Aldh1a1-3*$^{L2/L2}$). These crosses generated mutant males in which *Aldh1a1*, *Aldh1a2*, and *Aldha3* were ablated in Sertoli cells (i.e., *Amh-Cre*$^{tg/0}$/*Aldh1a1-3*$^{L2/L2}$), referred to as *Aldh1a1-3*$^{Ser-/-}$ mutants), and their control littermates (i.e., *Amh-Cre*$^{0/0}$/*Aldh1a1-3*$^{L2/L2}$ males), which did not display histological defects and were thus referred to as wild-type mice. *All-trans*-retinoic acid (RA) (MP Biomedicals) was dissolved at 0.5 mg/mL in 1% ethanol/rapeseed oil and kept at 4 °C under agitation until being administered by an i.p. injection (5 mg/kg body weight to resume spermatogonia differentiation). BMS493 and BMS753 (Tocris Bioscience) were diluted in DMSO at 50 and 2.5 mg/mL, kept at −20 °C and diluted in rapeseed oil just before being injected intraperitoneally (50 mg/kg body weight for BMS493 and 25 mg/kg body weight for BMS753). RA and BMS493 bind to RARA with K_d of about 0.4 and 4 nM, respectively (6). Thus, RA binds to RAR with a 10-fold higher affinity than the antagonist, making competition by low amounts of RA efficient. Accordingly, BMS493 alone at 50 mg/kg inhibits endogenous RA, which is present at a concentration of 0.25 μg/g of testis (7). On the opposite, RA at 25 mg/kg overcomes the binding of BMS493 at 50 mg/kg to RAR.

Histopathology and in Situ Hybridization Assays. Testis samples intended for histopathology were fixed in Bouin's fluid for 24 h at room temperature and embedded in paraffin. Transverse, 5-μm-thick, histological sections were stained with hematoxylin and eosin or periodic acid–Schiff (PAS). Testes and epididymides destined for Epon embedding were perfusion-fixed with 2.5% (wt/vol) glutaraldehyde in PBS and processed as described (8). In situ hybridization using digoxigenin-labeled probes for detection of *Gfra1*, *Kit*, *Lgals1*, *Rec8*, and *Zbtb16* expression were performed as described (1, 9).

Whole-Mount Immunohistochemistry. Freshly sampled testes were removed from their tunica albuginea and were manually dispersed in PBS. The seminiferous tubules were then fixed in PBS containing 4% (wt/vol) PFA and 10% (vol/vol) methanol for 2 h at 4 °C, and were washed several times in PBS containing 0.1% (vol/vol) Triton X-100 (PBS-T). The tubules were incubated overnight with one of the primary antibodies in PBS. The next day, the tubules were washed three times for 1 h each in PBS-T and incubated for 45 min at room temperature with one of the secondary antibodies in PBS-T. After a third round of washes, the immunostaining process was repeated in the case of a double immunohistochemistry using another primary antibody followed by the appropriate secondary antibody. The samples were mounted on glass slides in Vectashield (Vector Laboratories) containing 4′,6-diamidino-5-phenylindole (DAPI) (Roche Diagnostics) and observed under a confocal laser microscope. Primary antibodies were used at the following dilutions: 1:500 for RP453 rabbit anti-mouse RARG (10), 1:500 for goat anti-rat GFRA1 (R&D Systems), 1:50 for rat anti-mouse KIT (Chemicon; Millipore), and 1:50 for mouse anti-human ZBTB16 (Calbiochem–Merck). Secondary antibodies included the following: Alexa Fluor 488-conjugated donkey anti-rabbit IgG, Alexa Fluor 488-conjugated donkey anti-goat IgG, Cy3-conjugated goat anti-mouse IgG, Alexa Fluor 488-conjugated donkey anti-mouse IgG, Cy3-conjugated goat anti-rat IgG, and Alexa Fluor 488-conjugated goat anti-rat IgG (Jackson ImmunoResearch).

Immunohistochemistry on Testis Sections. For immunodetection of STRA6, 10-μm-thick sections of freshly frozen testes were postfixed for 5 min in cold acetone at −20 °C, air dried, and fixed a second time in ice-cold 4% (wt/vol) PFA in PBS. For immunodetection of AR, GATA1, gH2AX (phosphorylated H2AX), MAFB, pH3 (phosphorylated histone H3), and STRA8, testes were fixed by intracardiac perfusion of ice-cold 4% (wt/vol) PFA in PBS, and then kept in the same fixative overnight at 4 °C, washed in PBS, cryoprotected in sucrose solutions [10% (wt/vol) for 2 h followed by 20% (wt/vol) overnight] at 4 °C, and embedded in frozen medium before 10-μm-thick sections were performed. All of the sections were hydrated in PBS and then incubated overnight at 4 °C with one of the following antibodies: 1:500 for rabbit anti-mouse STRA6 (Abcam), 1:500 for rabbit anti-mouse AR (Santa Cruz Biotechnologies), 1:50 for rat anti-mouse GATA1 (Santa Cruz Biotechnologies), 1:500 for mouse anti-mouse gH2AX (Upstate), 1:100 for rabbit anti-mouse MAFB (Bethyl), 1:100 for rabbit anti-mouse pH3 (Upstate), and 1:500 for rabbit anti-mouse STRA8 (Abcam). Detection of bound primary antibodies was achieved by incubating the section for 45 min at room temperature using Cy3-conjugated goat anti-rabbit IgG, Cy3-conjugated goat anti-rat IgG, or Cy3-conjugated goat anti-mouse IgG (Jackson ImmunoResearch). The samples were mounted on glass slides in Vectashield (Vector Laboratories) containing DAPI and observed under a fluorescence microscope.

Preparation of Germ Cell Nuclear Spreads. Metaphase spreads of spermatocytes destined for karyotype, were prepared as described in ref. 11 and were stained with Giemsa.

Spermatozoa Smear and Electron Microscopy. Epididymis tails of control and *Aldh1a1-3*$^{Ser-/-}$ mice were cut into three pieces in PBS allowing sperm to flow out. Spermatozoa were immediately pipetted and fixed in ice-cold 4% PFA (wt/vol) for 10 min at 4 °C, before being gently centrifuged and washed several times. Spermatozoa were then mounted on glass slides and observed with a phase contrast microscope. Epididymides destined for Epon embedding were perfusion-fixed with 2.5% (wt/vol) glutaraldehyde, 2.5% (wt/vol) PFA in 0.1 M sodium cacodylate buffer, pH 7.8, and processed as described (8).

Organotypic Cultures. The testes from 12-d-old *Aldh1a1-3*$^{Ser-/-}$ mice were cut into 20 pieces of similar sizes. The fragments were placed on Millicell CM filters (pore size: 0.40 μm; Millipore) and cultured on the top of 320 μL of (1:1) Ham F12/DMEM serum free medium with glutaMAX (Life Technologies) supplemented with 40 μg/mL gentamicin. The tissue culture dishes were incubated at 37 °C in a humidified atmosphere containing 95% air/5% CO_2. For each mouse, fragments from one testis were cultured for 1 h in a medium containing 1 μM cycloheximide (Sigma-Aldrich), a protein synthesis inhibitor, before the medium was replaced by a fresh one containing both 1 μM cycloheximide and either BMS753 or BMS961 at 10^{-7} M dissolved in

DMSO. The fragments from the contralateral testes were cultured in a control medium containing the vehicle (ethanol). For each treatment, four to six testes were analyzed.

Isolation of Germ Cells by Flow Cytometry. Preleptotene spermatocytes were purified as previously described (12, 13). Briefly, 16 decapsulated *Aldh1a1-3*$^{Ser-/-}$ testes were incubated for 15 min in 3 mL of Gey's Balanced Salt Solution (GBSS) containing DNase I (3 μg/mL; Sigma Aldrich) and Collagenase Type I (1 mg/mL; Sigma Aldrich) and horizontally agitated on a shaking plate at 120 rpm, 33 °C for 15 min. Dispersed seminiferous tubes were left to sediment for 1 min at room temperature, and the supernatant was discarded before repeating this step. Seminiferous cells were then incubated in 2.5 mL of GBSS containing collagenase and trypsin (1 mg/mL; Sigma-Aldrich) for 15 min at 33 °C under agitation. Constant up and down pipetting for 3 min gently separated aggregates. Dispersed seminiferous cells were further incubated for 15 min at 33 °C on a shaking plate at 100 rpm after adding DNase I, trypsin, and 0.4 mg of Hoechst 33342 (Sigma-Aldrich). Following complete dissociation, 500 μL of FCS was added to inactivate trypsin. Final staining was performed by adding DNase I and 0.5 mg of Hoechst 33342 for 15 min on a shaking plate at 70 rpm and then 5 μg propidium iodide (Sigma-Aldrich) was added to the stained cells, which were then filtered through 40-μm strainers. Cells were kept on ice and protected from light until fluorescence-activated cell sorting (FACS). Sorting was performed using a Becton Dickinson Aria II cell sorter. Samples of the selected cell populations were immunostained with anti-GFRA1 (1:500; R&D Systems), anti-ZBTB16 (1:50; Calbiochem–Merck), or anti-STRA8 (1:500; Abcam) antibodies before being counterstained with DAPI and observed with a fluorescence microscope to assign the identity of either spermatogonia or preleptotene spermatocytes. The rest of the cells were centrifuged and proteins were extracted and resolved on 16% (wt/vol) SDS/PAGE gels (Expedeon) and blotted onto nitrocellulose membranes. RAR and RALDH2 were detected using SC-773 (Santa Cruz Biotechnologies) and ab75674 (Abcam) rabbit polyclonal antisera, respectively, at a dilution of 1:500. Immunoreactions were visualized using protein A coupled to horseradish peroxidase (dilution, 1:5,000), followed by chemiluminescence according to the manufacturer's protocol (GE Healthcare).

Analysis of Gene Expression by RT-PCR. Total RNA was prepared using TRIzol reagent (Life Technologies). Quantitative analysis of RNA was carried out by two steps reverse transcription (RT) coupled to quantitative real-time PCR using a Realplex2 (Eppendorf). RT of 1 μg of total RNA followed by PCR amplification of cDNA were performed using QuantiTect Reverse Transcription (Qiagen) and LightCycler 480 SYBR Green I Master (Roche Diagnostics) kits, respectively, according to the manufacturer's instructions. Conditions were 40 cycles with denaturation for 15 s at 95 °C, annealing for 15 s at 60 °C, and elongation for 15 s at 72 °C. Primers were as indicated in Table S1. Each cDNA sample was tested in triplicate. At least three samples were used in each experimental condition. The transcript level in each sample was normalized relative to that of *Actb* transcripts, whose expression is not changed by the mutation or by retinoid administration. Then data were expressed as fold induction relative to the vehicle condition. Statistical significance was analyzed using Student *t* test and statistical significance level was set for $P < 0.05$.

Chromatin Immunoprecipitation (ChIP). ChIP assays were performed on MSC-1 Sertoli cells and on *Aldh1a1-3*$^{Ser-/-}$ testes. The cells and dissected testes were cross-linked with 0.4% (wt/vol) PFA at room temperature for 10 and 15 min, respectively, before being dounced in and sonicated for 10 × 30 s on ice at maximum intensity to shear DNA to an average size of 500 bp followed by centrifugation at 20,000 × *g* for 15 min at 4 °C. For each ChIP reaction, immunoprecipitation was performed using 100 μg of sheared chromatin first incubated with 6 μg of either ChIP grade anti-pan-RAR SC-773× or anti-RNApol2 SC-9001× (Santa Cruz Biotechnologies) antibodies overnight at 4 °C, and then with 50 μL of preblocked protein G-Sepharose for 1 h at 4 °C. Beads were washed thoroughly, and eluted DNA–protein complexes were reverse cross-linked and purified. The purified immunoprecipitated DNA was analyzed by qPCR, and was compared with input DNA. Quantitation of RNApol2 and RAR binding was performed by determining the enrichment of immunoprecipitated chromatin at the suspected binding site compared with a site located upstream (−3 or −2 kb) of the gene transcription starting site (TSS). The TSS of *Arbp* (R*rplp0*) was used as a positive control for the ChIP experiment. The sequences of the primers used are described in Table S1.

Electrophoretic Mobility Shift Assays (EMSAs). They were performed as previously described (14). The oligonucleotides were annealed and labeled with [γ-^{32}P]ATP (Amersham Bioscience). For competition assays, unlabeled oligonucleotides were added in the preincubation mixture in 1- to 100-fold molar excess. The sequences of the oligonucleotides used are described in Table S1.

1. Ghyselinck NB, et al. (2006) Retinoids and spermatogenesis: Lessons from mutant mice lacking the plasma retinol binding protein. *Dev Dyn* 235:1608–1622.
2. Matt N, et al. (2005) Retinoic acid-dependent eye morphogenesis is orchestrated by neural crest cells. *Development* 132:4789–4800.
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7. Schmidt CK, Brouwer A, Nau H (2003) Chromatographic analysis of endogenous retinoids in tissues and serum. *Anal Biochem* 315:36–48.
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10. Rochette-Egly C, et al. (1991) Retinoic acid receptor gamma: Specific immunodetection and phosphorylation. *J Cell Biol* 115:535–545.
11. Peters AH, Plug AW, van Vugt MJ, de Boer P (1997) A drying-down technique for the spreading of mammalian meiocytes from the male and female germline. *Chromosome Res* 5:66–68.
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13. Getun IV, Torres B, Bois PR (2011) Flow cytometry purification of mouse meiotic cells. *J Vis Exp* 50:e2602.
14. Hwang JJ, Chambon P, Davidson I (1993) Characterization of the transcription activation function and the DNA binding domain of transcriptional enhancer factor-1. *EMBO J* 12:2337–2348.

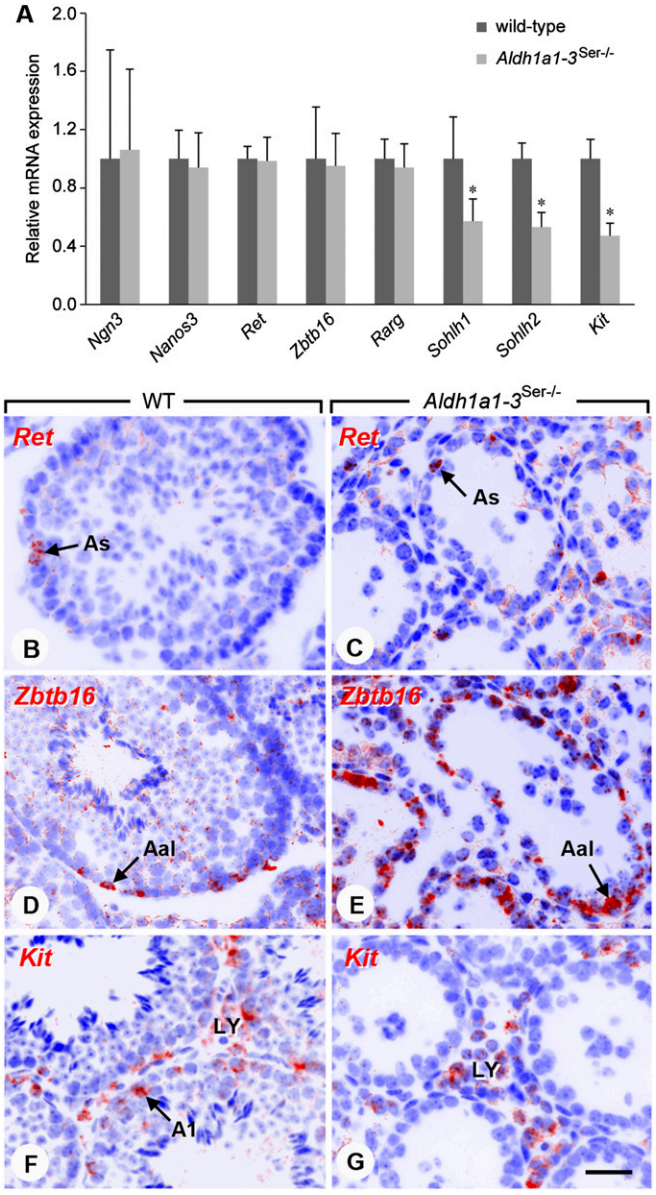

Fig. S1. Spermatogonia are blocked at the Aal stage in *Aldh1a1-3*$^{Ser-/-}$ mutants. (*A*) Relative expression of *Ngn3*, *Nanos3*, *Ret*, *Zbtb16*, *Rarg*, *Sohlh1*, *Sohlh2*, and *Kit* mRNA in testes of 5-d-old WT and *Aldh1a1-3*$^{Ser-/-}$ mutants. (*B–G*) In situ hybridization with antisense probes for *Ret*, *Zbtb16*, and *Kit* (red false color) superimposed with the DAPI counterstained (blue false color) on testis sections of 40-d-old WT and *Aldh1a1-3*$^{Ser-/-}$ mutants. Error bars represent SEM ($n = 5$); *$P < 0.05$. As, A single spermatogonia; Aal, A aligned spermatogonia; A1, A1 spermatogonia; LY, Leydig cells. [Scale bar (in *G* for reference): 15 μm.]

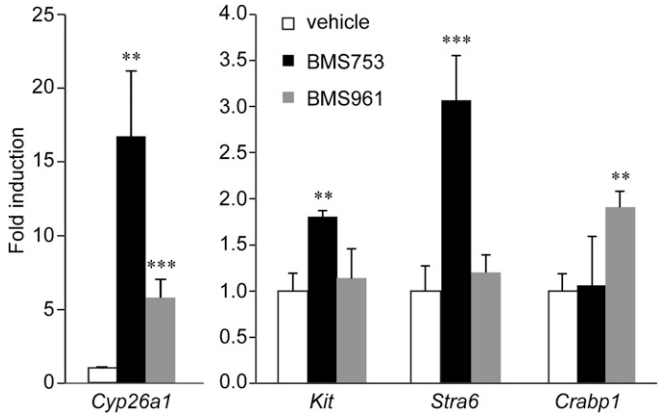

Fig. S2. BMS753 and BMS961 selectively activate RARA and RARG in Sertoli cells and in spermatogonia, respectively. Relative expression of *Cyp26a1*, *Kit*, *Stra6*, and *Crabp1* mRNA quantified by qRT-PCR in testes of *Aldh1a1-3*$^{Ser-/-}$ mutants treated for 6 h with vehicle (white bars), BMS753 (black bars), or BMS961 (gray bars). Error bars represent SEM (n = 3–5); $^{**}P < 0.01$, $^{***}P < 0.001$.

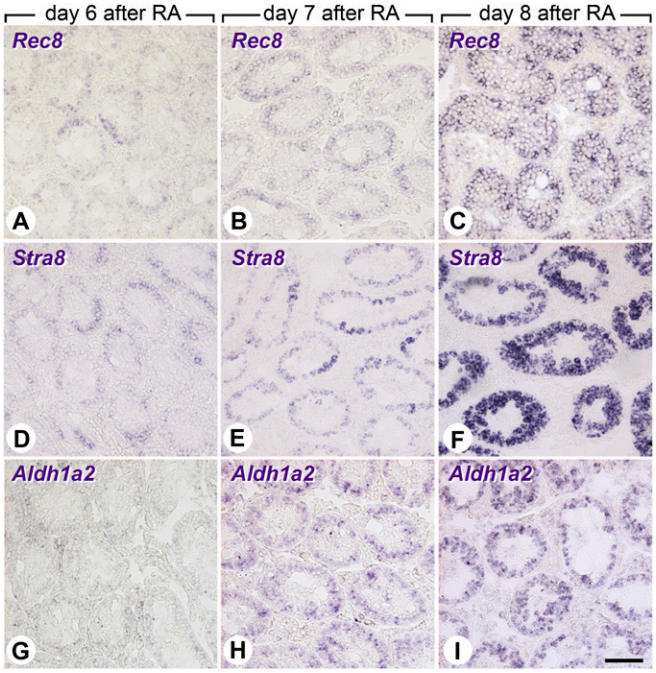

Fig. S3. Kinetics of *Rec8*, *Stra8*, and *Aldh1a2* mRNA expression in rescued *Aldh1a1-3*$^{Ser-/-}$ mutants. (*A–I*) In situ hybridization with antisense probes for *Rec8*, *Stra8*, and *Aldh1a2* (purple signal) on histological testis of *Aldh1a1-3*$^{Ser-/-}$ mutants rescued by a single injection of RA at day 0 and analyzed at day 6, 7, and 8 (*C*, *F*, and *I*). [Scale bar (in *I* for reference): 25 μm.]

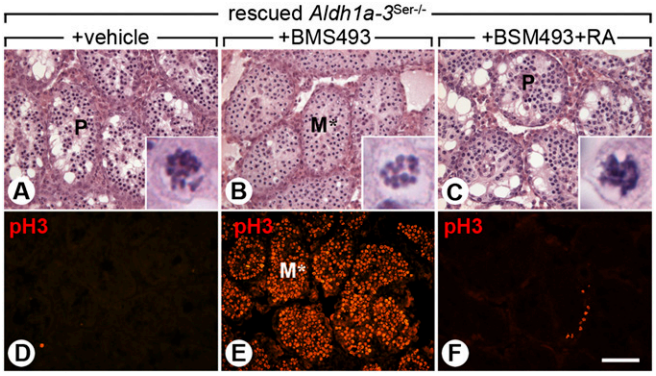

Fig. S4. Meiotic initiation in rescued *Aldh1a1-3*$^{Ser-/-}$ mutants requires RA. (*A–C*) Histological sections through testes of *Aldh1a1-3*$^{Ser-/-}$ mutants rescued by a single injection of RA at day 0, and then treated with vehicle, BMS493, or BMS493+RA from days 6 to 8, and analyzed at day 12. (*D–F*) Immunodetection of pH3 (red signal) in the same samples. M*, metaphase-like cells; P, pachytene spermatocytes. [Scale bar (in *F* for reference): 60 μm; *A–C*, *Insets*, 10 μm.]

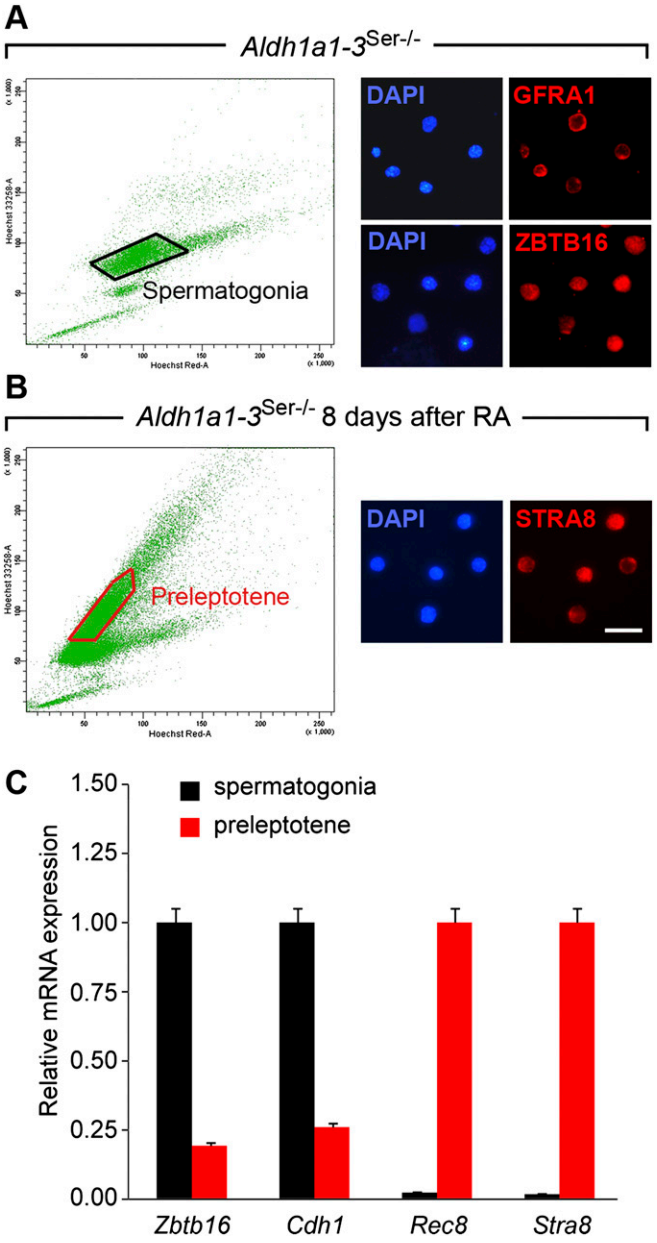

Fig. S5. Characterization of FACS-purified cells from *Aldh1a1-3*$^{Ser-/-}$ testes. (*A*) Flow-cytometric analysis of cells isolated from *Aldh1a1-3*$^{Ser-/-}$ testes at PN40 and stained with Hoechst 33342 and propidium iodide. The selected cell population is indicated by a black box (*Left*). Immunodetection of GFRA1 and ZBTB16 (red) and DAPI nuclear counterstain (blue) on an aliquot of the selected cell population (*Right*). (*B*) Flow-cytometric analysis of cells isolated at day 8 from testes of *Aldh1a1-3*$^{Ser-/-}$ treated with RA at day 0. The selected cell population is indicated by a red box (*Left*). Immunodetection of STRA8 (red) and DAPI nuclear counterstain (blue) on an aliquot of the selected cell population (right panels). (*C*) Relative expression of *Zbtb16*, *Cdh1*, *Rec8*, and *Stra8* mRNA quantified by RT-qPCR in purified cell populations. The cells purified from *Aldh1a1-3*$^{Ser-/-}$ testes were identified as spermatogonia as they expressed GFRA1 and ZBTB16, but not *Rec8* and *Stra8*. The cells purified from testes of *Aldh1a1-3*$^{Ser-/-}$ treated with RA were identified as preleptotene spermatocytes as they expressed *Stra8*, *Rec8*, but weakly *Zbtb16* and *Cdh1*. [Scale bar (in *B* for reference): 10 μm.]

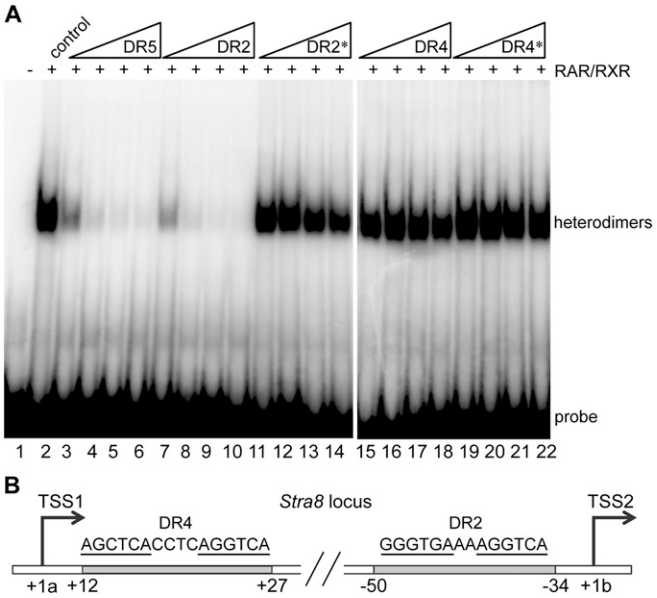

Fig. S6. Binding of RAR/RXR heterodimers on the response elements identified in *Stra8* gene. (*A*) EMSA showing a strong binding of RAR/RXR heterodimers on the radiolabeled, canonical, DR5 of *Rarb2* gene (lane 2, control). Increasing concentrations of cold DR5 (lanes 3–6) and cold DR2 identified in *Stra8* gene (lanes 7–10) are both able to compete for RAR/RXR binding on radiolabeled DR5. In contrast, increasing concentrations of cold DR2* (bearing mutations in the RARE sequence) were no longer able to compete for RAR/RXR binding (lanes 11–14). Neither DR4 (lanes 15–18) nor DR4* (bearing mutations in the RARE sequence; lanes 19–22) competed the binding of RAR/RXR heterodimers on radiolabeled DR5. (*B*) Schematic representation of DR2 and DR4 sequences and location in *Stra8* locus. The two transcription start sites (TSS1 and TSS2) are depicted by broken arrows and are numbered +1a and +1b. The positions of DR4 and DR2 are indicated relative to TSS1 and TSS2, respectively.

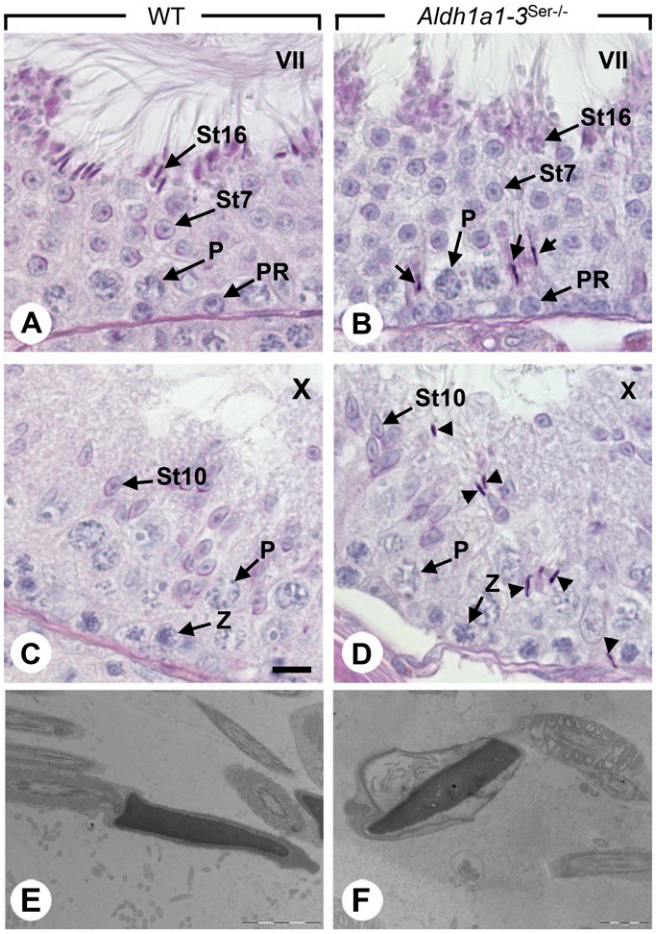

Fig. S7. Failure of spermatid release in RA-rescued *Aldh1a1-3*$^{Ser-/-}$ mutants. (*A–D*) Histological sections through testes of 24-wk-old WT and *Aldh1a1-3*$^{Ser-/-}$ mutants rescued by a single injection of RA at 4 wk of age. Normal germ cell associations or stages of the seminiferous epithelium cycle were observed in the mutant testis. However, numerous mature (i.e., step 16) spermatids did not align at the luminal side of the seminiferous epithelium at stage VII (arrows in *B*; compare with *A*) and remained trapped within the epithelium at stage X (arrowheads in *D*; compare with *C*), indicating a failure of spermiation. (*E* and *F*) Transmission electron microscope micrographs of representative epididymal spermatozoa isolated from WT and RA-rescued *Aldh1a1-3*$^{Ser-/-}$ mutants. P, pachytene spermatocytes; PR, preleptotene spermatocytes; St7, St10, and St17, step 7, 10, and 16 spermatids, respectively; Z, zygotene spermatocytes. Roman numerals indicate the stage of the seminiferous epithelium. PAS stain. [Scale bar (in *C* for reference): *A–D*, 20 μm; *E* and *F*, 1 μm.]

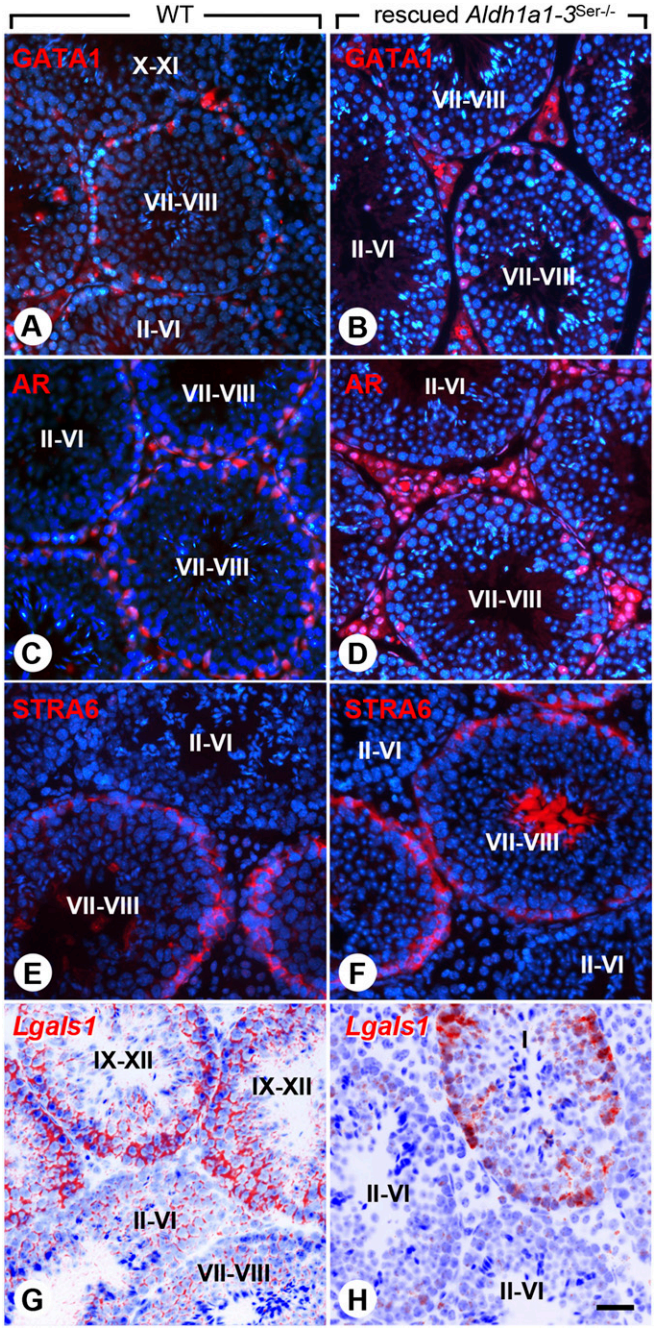

Fig. S8. The oscillating activity of Sertoli cell is normal in RA-rescued *Aldh1a1-3*$^{Ser-/-}$ mutants. (*A–F*) Immunodetection of GATA1, AR, and STRA6 on testes sections from 24-wk-old WT and *Aldh1a1-3*$^{Ser-/-}$ mutants rescued by a single injection of RA at 4 wk of age. The signals (red) are superimposed with the DAPI nuclear counterstain (blue). (*G* and *H*) In situ hybridization with antisense probe for *Lgals1* on testis sections. The signals (red false color) are superimposed to the DAPI nuclear counterstain (blue false color). Roman numerals designate the stages of the seminiferous epithelium cycle: II–VI, stages II, III, IV, V, or VI; VII–VIII, stages VII or VIII; X–XI, stage X or XI. [Scale bar: (in *H*) 60 μm.]

Table S1. Sequences of primers used for quantitative RT-PCR, ChIP, and EMSA

Gene	Primers (forward and reverse)
Aldh1a2	5′-GGCTGGGCTGATAAAATTCA-3′
	5′-ACGGTGTTACCACAGCACAA-3′
Crabp1	5′-CTTACTGGACCCGAGAGCTG-3′
	5′-GGGGAGAGGTGTGTGGAGTA-3′
Cyp26a1	5′-TTCCCATTGACGTGCCCTTT-3′
	5′-CCAGCCTCTCTCCCCTCTCC-3′
Kit	5′-AGCGTCTTCCGGCACAACGG-3′
	5′-GCCAATGAGCAGCGGCGTGA-3′
Mafb	5′-GACAGGCTTTGCGTCCTAAG-3′
	5′-CGTTAGTTGCCAATGTGTGG-3′
Nanos3	5′-CGGCCTGACAAGGCAAAGAC-3′
	5′-CACCATGGTCCTCCCCACTC-3′
Ngn3	5′-AGGGCGCCTGCAGTTTAGC-3′
	5′-TGGGGACACTTGGATGGTGA-3′
Rarg	5′-CCTGAACCGAACCCAGATGC-3′
	5′-CACCTTCTCGGGCTCTTCCA-3′
Rec8	5′-TGATATGGAGGAGGCTGACC-3′
	5′-GCAGCCTCTAAAAGGTGTCG-3′
Ret	5′-TGGGCTGTCCCGAGATGTTT-3′
	5′-TTGCCTCCCAGGGTCACAAT-3′
Sohlh1	5′-GGGCCAATGAGGATTACAGA-3′
	5′-CACAGGAGCTGTGCAGAGAG-3′
Sohlh2	5′-AAGGCCCCGTTGTCGAGTCT-3′
	5′-CTCCCGGACCTGCTTCACAT-3′
Spo11	5′-TTGATTGCTGGCAACTTGAG-3′
	5′-CAGATGCTGCATTCCTTGAA-3′
Stra6	5′-GGGGACTGGAGTCACTGTGT-3′
	5′-GCCAGTGTGAGGAGAAGAGG-3′
Stra8	5′-GGAGAAAAAGGCCAGACTCC-3′
	5′-CCACGTCAAAAGCATCTTCA-3′
Zbtb16	5′-AACGGTTCCTGGACAGTTTG-3′
	5′-CCCACACAGCAGACAGAAGA-3′
Mafb binding site	5′-GAGCTGCGTCTTCTCGTTCT-3′
	5′-CAGCTGGTGTCCATGTCG-3′
Mafb -2kb	5′-ACGTAGGGCAAAGACAATGG-3′
	5′-ACATGGTAAGGGCTGACGAC-3′
Stra8 binding site DR2	5′-AGGTCATCTTGCTCCTTCCA-3′
	5′-ATCACAGCCCTGTCACTGC-3′
Stra8 binding site DR4	5′-GTGAGGTAGATCCCGGATTG-3′
	5′-GACCTGAGGTGAGCTGCTTC-3′
Stra8 -2kb	5′-GGCAGCAGGCCACCAATAAA-3′
	5′-TAGGCTTGGTTCCCCGTGTG-3′
Stra8 DR2	5′-TAGCCGCCTGGATGGGGTGAAAAGGTCATCTTGCTCCTTC-3′
	5′-GAAGGAGCAAGATGACCTTTTCACCCCATCCAGGCGGCTA-3′
Stra8 mutated DR2	5′-TAGCCGCCTGGATGGAGTTAAAATGTCATCTTGCTCCTTC-3′
	5′-GAAGGAGCAAGATGACATTTTAACTCCATCCAGGCGGCTA-3′
Stra8 DR4	5′-GGATGGTGAAGCAGCTCACCTCAGGTCAGACTGCAGGAGG-3′
	5′-CCTCCTGCAGTCTGACCTGAGGTGAGCTGCTTCACCATCC-3′
Stra8 mutated DR4	5′-GGATGGTGAAGCATCTCACCTCAAGTAAGACTGCAGGAGG-3′
	5′-CCTCCTGCAGTCTTACTTGAGGTGAGATGCTTCACCATCC-3′
Rarb DR5	5′-AGCCCGGGAAGGGTTCACCGAAAGTTCACTCGCATATATT-3′
	5′-AATATATGCGAGTGAACTTTCGGTGAACCCTTCCCGGGCT-3′