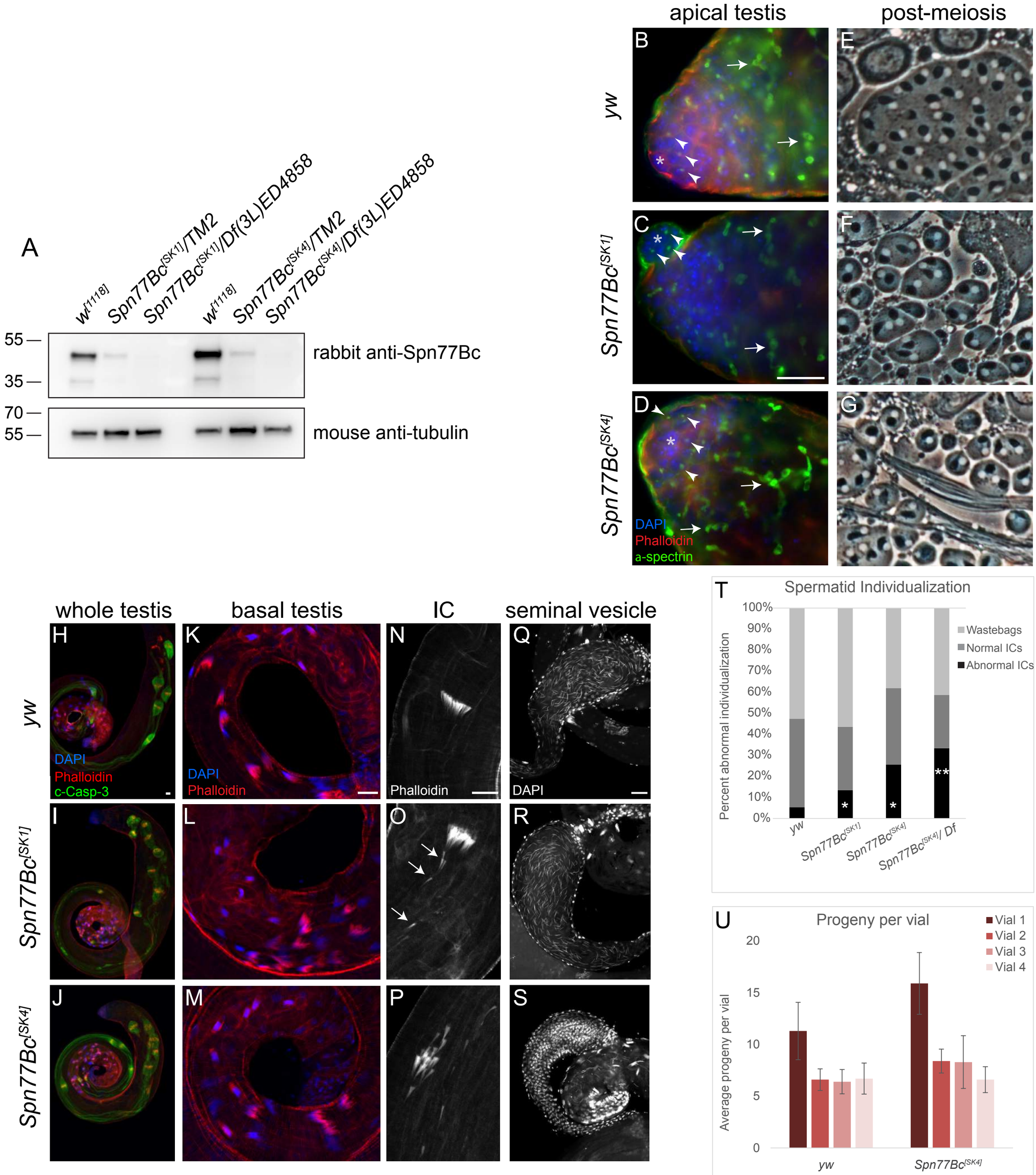

Supplementary Figure 15. Mutations in *Spn77Bc* display spermatid individualization defects. (A) Western Blot showing that two different alleles of CG6289/Spn77Bc are strong loss-of-protein alleles. (B-D) At the apical hub of the testis (asterisk), stem cells and gonialblasts, marked by round spectrosomes (anti-spectrin, green, arrowheads), are normal in the two mutant alleles of *Spn77Bc* (SK1 and SK4). Mitotic spermatogonia, marked by branched fusomes (a-spectrin, green,arrows), are also normal. (E-G) Meiotic cytokineses are normal, as evidenced by the appearance of one phase dark mitochondrial aggregate for each phase light nucleus in squashed testis preparations. (H-M) In *Spn77Bc* mutants, Caspase-3 staining is normal (H-J, cleaved Caspase-3, green), as are nuclear condensation and bundling (K-M, DAPI, blue) and individualization complex formation (K-M, phalloidin, red). (N-P) In *Spn77Bc* mutants, individualization complexes (phalloidin) disperse abnormally as they progress away from the nuclei towards the apical region of the testis. (Q-S) In the weaker mutant allele, SK1, mature needle shaped sperm nuclei (DAPI) are visible in the seminal vesicle (R), while in the stronger allele, SK4, fewer mature sperm are seen (S). Scale bars are 20 μm. (T) *Spn77Bc* mutants have individualization defects, showing significantly more abnormal individualization complexes (ICs) than controls. *, $p < 0.005$. **, $p < 0.0001$. (U) Despite testis cytology defects, *Spn77Bc* mutants do not have detectable fertility defects.

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Supplementary Figure 15