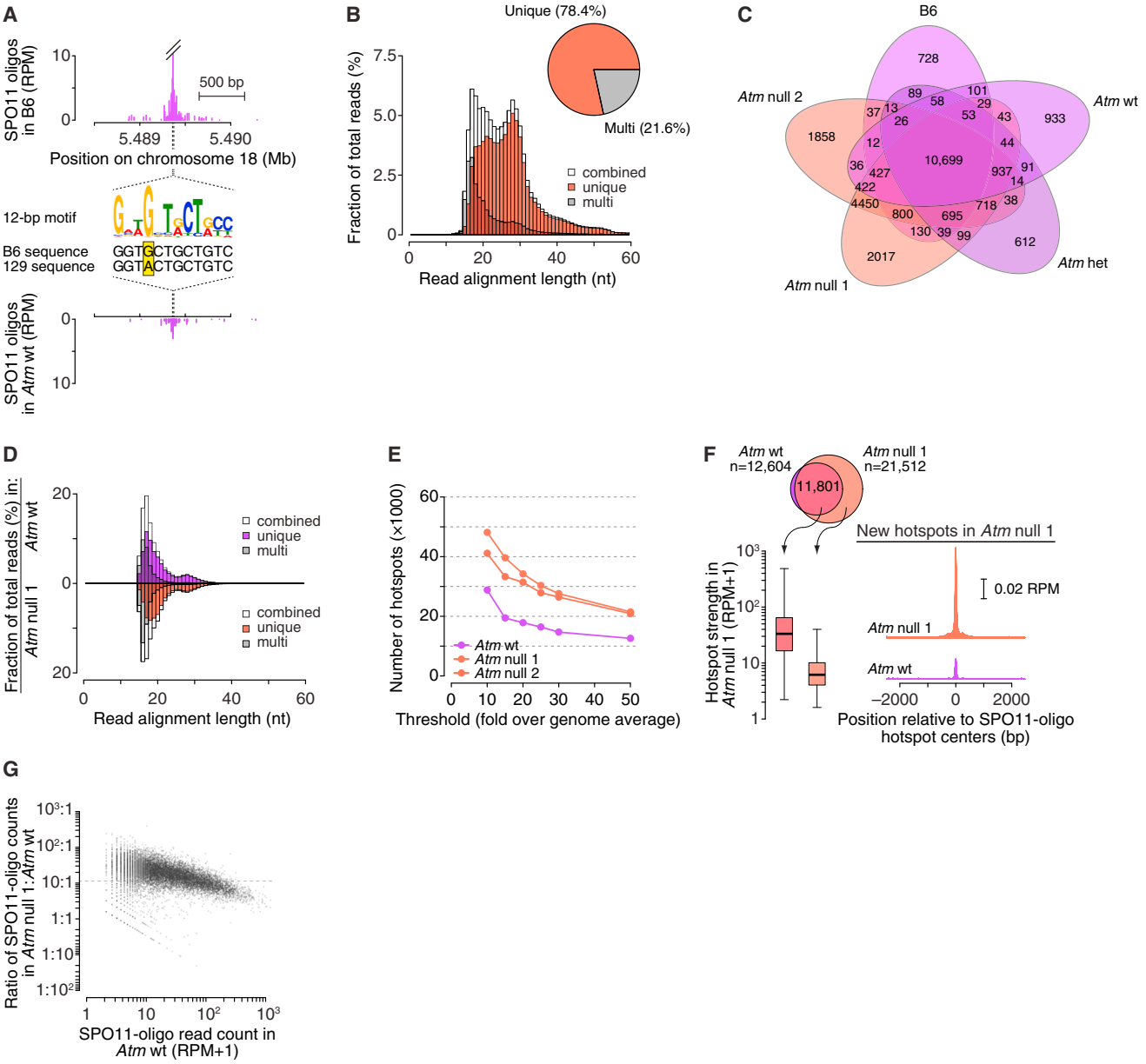

Figure S7. ATM Controls the Distribution of Meiotic DSBs, Related to Figure 7

(A) Example of a SPO11-oligo hotspot where disparate strengths in B6 and *Atm* wt may be due to a sequence polymorphism in the 12-bp PRDM9 motif. (Hotspot is on chromosome 18, with center at position 5,489,367 in B6 and 5,489,365 in *Atm* wt.) The *Atm* wt map is from mice with a mixed background of 129 and B6, strains that share the same *Prdm9* allele. At this hotspot, PRDM9 may not bind as efficiently to the 129 chromosome.

(B) Length distribution of SPO11 oligos from *Atm* null (sample 1) that mapped uniquely or to multiple sites in the genome.

(C) Overlap of hotspot calls from the five SPO11-oligo maps generated for this study.

(D) Length distribution of SPO11 oligos after trimming in silico. Sequence reads were randomly sampled from *Atm* wt and *Atm* null (5% of the reads from each), then the *Atm* null reads were trimmed from their 3′ ends to match the length distribution from *Atm* wt. Both samples were then re-mapped to the mouse genome, and the resulting maps were evaluated for the spatial patterns discussed in this study. Distributions of the lengths of aligned reads are shown for unique mappers and multi-mappers for *Atm* wt and *Atm* null sample 1.

(E) Datasets from *Atm* null animals still yielded more hotspots than from *Atm* wt after read length trimming. Compare with Figure 7B.

(F) After read length trimming, new hotspots in *Atm* null still correspond to weak hotspots that also yield small numbers of DSBs in wild-type. Boxplot is as defined in Figure S2A legend; SPO11-oligo profiles were smoothed with a 51-bp Hann filter. Compare with Figure 7C.

(G) After read length trimming, weaker hotspots are still disproportionately increased in ATM-deficient spermatocytes. Compare with Figure 7D.