Supplemental Figure Legends

Figure S1. Features and Validation of the SPO11-Oligo Map, Related to Figure 1
(A) Specificity of PCR amplification of SPO11 oligos ligated to HiSeq adaptors, shown for sample *Atm* null 1. PCR was templated with adaptor-ligated SPO11 oligos or with samples processed from mock immunoprecipitates (IP). Amplification products were separated by native polyacrylamide gel electrophoresis. Vertical bar indicates expected range of SPO11-oligo amplicons; asterisk indicates adaptor dimers.
(B) SPO11 oligos in known crossover hotspots. From published reports we compiled 16 crossover hotspots characterized by fine-scale analysis of recombinant products in sperm isolated from F_1 progeny of crosses between B6 and either A/J, C3H/HeJ ("C3H") or DBA/2J ("DBA") (Kauppi et al., 2007; Bois, 2007; Wu et al., 2010; Getun et al., 2010; Cole et al., 2010; Getun et al., 2012; de Boer et al., 2015). All 16 hotspots displayed evidence of $PRDM9^{B6}$-dependent crossover formation. See **Table S2B** for detailed information. In 15 of these crossover hotspots (all except *HS18.2*), SPO11 oligos were enriched within the region encompassed by the coordinates of the recombination site. In *HS18.2*, most crossovers in the B6×DBA F_1 hybrids are presumably initiated from DSBs on the DBA haplotype. *HS44.2* encompasses two SPO11-oligo clusters that are merged by our algorithm into one SPO11-oligo hotspot on account of their proximity. Each plot shows a 5001-bp region centered on the midpoint between the start and end coordinates of the crossover hotspot (defined by outermost polymorphisms that encompass all crossover events in the cited references). SPO11 oligos were smoothed with a 201-bp Hann filter.
(C) Individual maps of SPO11 oligos and of SSDS coverage on the top (Watson) strand and bottom (Crick) strand (Brick et al., 2012) in a 3001-bp window around hotspot *A3*.
(D) We employed two strategies to assign positions for multi-mapped reads. In one, we reasoned that a multi-mapped SPO11 oligo is more likely to have originated from the position that also had a greater number of neighboring unique reads. Thus, we fractionally distributed each multi-mapped read in proportion to the local density of unique reads, using an iterative imputation strategy similar to ones used previously to analyze ChIP-seq data and to map *S. pombe* Rec12 oligos (STAR Methods) (Chung et al., 2011; Fowler et al., 2014). In the second strategy, we normalized by dividing each read evenly among its mapped positions. SPO11-oligo maps shown are for three 501-bp regions that share high sequence identity, centered on the genome positions indicated. Whereas the normalized maps of multi-mapped reads are very similar, the imputed maps are different because multi-mapped reads are assigned using the relative local density of unique reads. Each map is plotted as RPM of the total 69.4 million reads mapping to the nuclear genome.
(E) Fine-scale patterns of unique reads and multi-mapped reads are similar at hotspot *A3*. Each map is plotted as RPM based on the total 69.4 million reads that mapped to the nuclear genome. This is in contrast to other figures (e.g., **Figure 1E**), in which only the 46.6 million uniquely mapped reads were considered. Totals given are for the 3001-bp window around the SPO11-oligo hotspot center.
(F) Multi-mapped reads are enriched in hotspots in proportion to hotspot strength. In hotspots called using unique SPO11-oligo reads at 50× over genome average, the normalized multi-mapped read counts showed good correlation with the counts of uniquely mapped reads (Pearson's *r*). This finding supports the conclusion that the majority of multi-mappers arise in similar proportion from the same genomic regions as do unique mappers, justifying the use of the imputation strategy outlined in panel D. For each hotspot, one read was added to unique and multi counts to include on the log scale those hotspots with <1 normalized multi-mapped read.

Figure S2. Features of SPO11-Oligo Hotspots, Related to Figure 2

(A) SSDS hotspots with an overlapping SPO11-oligo hotspot are substantially stronger than those without an overlapping SPO11-oligo hotspot. In the box plots, thick horizontal lines indicate medians, box edges show the 25th and 75th percentiles, and whiskers indicate lowest and highest values within 1.5-fold of the interquartile range; outliers are not shown.

(B) Distribution of SPO11 oligos (smoothed with a 51-bp Hann filter) and SSDS coverage around centers of SSDS hotspots with or without an overlapping SPO11-oligo hotspot. Note that a small peak of SPO11 oligos was present even for the non-overlapping set of SSDS hotspots.

(C) Hotspot centers are defined more precisely by SPO11-oligo mapping. Panels show heat maps of Watson and Crick SSDS coverage around hotspot centers as defined by SSDS (left panels) or by SPO11-oligo mapping (right panels). The normalized SSDS coverage across each 5010-bp window was binned in 10-bp bins. Because locally normalized coverage was used, color-coding reflects spatial patterns within hotspots and not relative intensity between hotspots. Only the 12,694 hotspots called in both data sets are shown and are ordered in each plot by center-to-center distance between the alternative center definitions. The black line in each plot indicates the center position of the matched hotspot as defined in the alternative data set. The SPO11-oligo hotspot centers track more faithfully with the switch between Watson and Crick SSDS coverage: compare the black lines to the boundary between red and blue SSDS signal (left and right panels), and note that this boundary lines up vertically across all hotspots when aligned by the SPO11-oligo hotspot center definition (right panels).

(D) Reducing the precision of SPO11-oligo hotspot definitions recapitulates genome averages derived from SSDS hotspot center definitions. Resection uncovers tracts of ssDNA that are much longer than the width of most DSB hotspots, and sequencing of DMC1-bound ssDNA by SSDS displays marked local biases. (Note the strong peaks-and-valleys appearance of the SSDS maps around *A3* as an example; **Figure 1E**). These two features would be predicted to negatively affect the precision in estimating DSB hotspot centers. In contrast, the 5′ ends SPO11 oligos are more directly and precisely tied to DSB positions per se, and SPO11 oligos derive from narrow zones. Thus, centers of SPO11-oligo hotspots are predicted to provide a more precise estimate of true DSB hotspot centers. To test this conclusion, we artificially blurred the precision of SPO11-oligo hotspot centers and examined resulting average patterns in the SPO11-oligo and SSDS datasets. We first determined, for overlapping SPO11-oligo and SSDS hotspots, the distribution of distances between the hotspot centers defined by the two methods (range: -1676 to +1634 bp). We then sampled with replacement from that distribution to shift the location of each SPO11-oligo hotspot center in the 5′ or 3′ direction. The simulation was run 1000 times. As expected, when we plotted SPO11 oligos (smoothed with a 51-bp Hann filter) and SSDS coverage around these blurred centers, we reconstituted the average patterns observed around SSDS hotspot centers (compare with **Figure 1G**). We conclude that the SPO11-oligo map more accurately reflects the fine-scale distribution of recombination initiation in mice.

(E) Distribution of SPO11 oligos (51-bp Hann filter) and SSDS coverage around SPO11-oligo hotspot centers. Data are the same as in **Figure 2D**, but zoomed into the lower range of SPO11-oligo values to focus on secondary peaks.

(F) The average patterns of unique reads, imputed multi-mapped reads, and normalized multi-mapped reads around the centers of SPO11-oligo hotspots are similar. Each map was plotted as RPM of the total 69.4 million reads mapped to the nuclear genome and was smoothed with a 51-bp Hann filter.

(G) Cumulative fraction, by distance from hotspot centers, of SPO11 oligos around all hotspots. Of the 29.4 million SPO11-oligo reads that mapped within 2500 bp of hotspot centers, 76.9% were within 100 bp of hotspot centers and 93.9% within 500 bp.

(H) Comparison of average SPO11-oligo profiles around mouse and *S. cerevisiae* hotspots. The profile from each species is scaled to yield matched areas under the curves for the regions

shown. Note that the average for budding yeast hotspots is less compact than the central peak for mouse, as indicated by the lower height and greater width for yeast. In both species, hotspot substructure appears to be strongly shaped by the underlying chromatin structure. This substructure shows more variation between hotspots in budding yeast because the NDRs in gene promoters that are targeted by Spo11 show more variation from one to another.

Figure S3. H3K4me3 and PRDM9 Binding, Related to Figure 3

(A) Mean locally normalized SPO11-oligo and H3K4me3 profiles. Data are the same as in **Figure 3A**, but zoomed to focus on the lower range of values for SPO11 oligos.

(B) Mean locally normalized SPO11-oligo and H3K4me3 profiles for subsets of hotspots. Top: 20% highest left-over-right (L>R) asymmetry; Bottom: 20% highest right-over-left (R>L) asymmetry (n=2,792 for each group). Data are the same as in **Figure 3C**, but zoomed to focus on the lower range of values for SPO11 oligos. On average, the distribution of SPO11 oligos in secondary peaks to the left versus the right displays weak asymmetry in the same direction as H3K4me3 asymmetry. However, even at hotspots with strongly asymmetric H3K4me3, significant secondary peaks of SPO11 oligos can occur on the side that has low or no H3K4me3 signal.

(C) Enrichment of the 12-bp motif (see **Figure 3D**) coincides precisely with the central peak of SPO11 oligos. SPO11 oligos and motif density were each smoothed with a 51-bp Hann filter.

(D) The 12-bp motif enriched in SPO11-oligo hotspots is similar to motifs enriched in SSDS hotspots or in $PRDM9^{B6}$-dependent H3K4me3 peaks and matches part of a larger 36-bp sequence to which $PRDM9^{B6}$ is predicted to bind (Brick et al., 2012; Baker et al., 2014; Baker et al., 2015; Davies et al., 2016). In the subset of hotspots lacking this “primary” 12-bp motif, a “secondary” 15-bp motif that largely overlaps the primary motif was enriched. A 15-bp motif largely overlapping the primary motif was also enriched in new hotspots arising in *Atm* null 1. See STAR Methods.

(E) Asymmetry in H3K4me3 signal is present in all three classes of PRDM9 binding sites. Shown for each of the three classes of PRDM9 motifs (from **Figure 3F**) are heat maps of H3K4me3 (left) and SPO11-oligo (right) signal around motif centers, ordered within each class according to asymmetry in local H3K4me3 pattern. Normalized SPO11-oligo values across each 1005-bp window were binned in 5-bp bins. k-means clustering of SPO11-oligo spatial patterns was carried out on SPO11-oligo profiles in 501-bp windows centered on motif occurrences and smoothed with a 15-bp Hann filter.

(F) The three PRDM9 motif classes have indistinguishable sequences. Sequence logos for 101-bp windows around motif midpoints are shown.

Figure S4. Effects of Differences Between Sex Chromosomes and Autosomes With Respect to Timing of DSB Formation and the Lifespan of Resected DSBs, Related to Figure 4

(A) The ratio of SSDS tag counts to SPO11-oligo counts is substantially higher for SSDS hotspots on sex chromosomes than on autosomes. One SSDS tag count and one SPO11-oligo read count were added to each hotspot. Boxplots are as defined in **Figure S2A**. The p-value is from a Wilcoxon rank-sum test.

(B) Model for the relative timing of DSB formation and the persistence of SPO11-oligo complexes and DMC1-bound ssDNA. The lifespan of SPO11-oligo complexes is known to be long relative to the length of meiotic prophase (Lange et al., 2011), so differences in the timing of DSB formation cause little net difference in lifespan of SPO11-oligo complexes. Whole-testis SPO11-oligo levels are thus expected to represent total DSB numbers well. In contrast, the average lifespan of DMC1-bound ssDNA is much shorter relative to prophase I, because it disappears as recombination progresses. However, this lifespan can differ markedly depending on the chromosomal context. On autosomes, RAD51 and DMC1 foci first begin to appear in

leptonema and disappear progressively over the course of zygonema and early pachynema, but on the non-PAR portions of the sex chromosomes RAD51 and DMC1 foci are present at later stages of prophase I (Moens et al., 1997). The late-persisting foci eventually disappear in mid-to-late prophase, presumably via sister-chromatid or intra-chromatid recombination. Note also that DSBs form over longer periods on unsynapsed chromosome segments (including on the X chromosome) because a regulatory circuit inhibits DSB formation where synaptonemal complex has formed (Kauppi et al., 2013). As depicted schematically in the figure, these temporal considerations can explain why SSDS signal relative to SPO11-oligo density is higher for hotspots on the sex chromosomes than on autosomes, and why this discrepancy is more extreme for stronger hotspots if these tend to be the ones that experience the earliest DSBs.

Figure S5. Estimating Resection Tract Lengths Using SPO11 Oligos and SSDS Coverage, Related to Figure 5

We compared the SPO11-oligo map with the SSDS map to estimate the distribution of resection tract lengths. All plots in this figure display data from a subset of 11,804 SPO11-oligo hotspots chosen on the basis of overlap with an SSDS hotspot and sufficient distance (>5 kb) from neighboring hotspots. See STAR Methods for further details on hotspot selection, modeling, and validation.

(A) Example of a DSB hotspot and the spatial relationship between resected DSBs, SPO11 oligos, and strand-specific SSDS coverage. This panel shows the same hotspot depicted in **Figure 5B**, but in greater detail. (Hotspot is on chromosome 3, with center at position 107,616,828.) Note that SSDS coverage on the Crick strand arises from 5′→3′ exonucleolytic resection moving to the right away from the DSB, whereas coverage on the Watson strand arises from resection moving leftward. Notice also the marked spikiness in the SSDS coverage, which is not predicted for random sampling of ssDNA from a population of resected DSBs. This spikiness likely reflects biases in SSDS library preparation from the sonication end effect and from the need for microcomplementarity for foldback annealing of ssDNA (see panels D, E, and F).

(B) Approach for modeling ssDNA coverage as a function of the distribution of DSBs and the resection profile *R*. At a given genomic position *i*, ssDNA on the Crick strand can arise from a DSB at that position that was resected for a distance of at least 1 nt, or from a DSB 1 bp to the left that was resected at least 2 nt, and so on. We can therefore model the population-average ssDNA distribution at position *i* as the sum of DSBs located *x* bp to the left times the probability of each DSB being resected more than *x* nt, for values of *x* from 0 to the end of the chromosome. For practical purposes, however, we only consider plausible resection distances, i.e., ≤2.5 kb from the DSB. We also include a term to account for all sources of error in measuring the DSB distribution (empirically, the SPO11-oligo distribution) and ssDNA amount (empirically, SSDS coverage). The same logic applies to modeling ssDNA on the Watson strand, except that resection comes from DSBs located to the right of the genomic position in question.

(C) Deriving an estimate of resection tract lengths. The graph on the left shows the best-fit solution to the series of >1.2×10^7 multinomial equations of the form shown in panel B (see STAR Methods). This is an initial estimate (*R′*) of the resection profile *R*. This distribution has somewhat arbitrary units because of difference in measurement types for SPO11 oligos and SSDS. More importantly, *R′* contains a pronounced peak at short distances that is not expected for the true ssDNA coverage profile around DSBs. This peak can be accounted for as a sequencing bias in favor of recovery of DSB-proximal ssDNA fragments (see panels E and F). To correct for this bias, we made the simple assumption that ssDNA coverage should be maximal within a plateau extending from the DSB site. We manually set this plateau by recording the maximum value that was obtained to the right of the artifact peak (dashed line in right panel), and extending this value back to the left. We also set the maximum to 1 to convert

the arbitrary units of *R´*to the desired probability distribution *R*. The graph in the middle shows the empirical estimate of *R* and the superimposed logistic regression smoothing. The graph on the right shows the underlying distribution of resection tract lengths, calculated as the first derivative of 1 – *R*.
(D) Spikiness in the SSDS map. (i) When SPO11-oligo hotspots were ordered by the position of maximal mean normalized signal (binned by 10 bp) across each hotspot, spikes of signal along the resection tract segments were apparent. These spikes are also readily apparent in SSDS coverage maps around individual hotspots (**Figures 1E, S6, and S5A**). (ii) When these hotspots were divided into four groups based on the position of maximal signal relative to hotspot center, all of the group averages displayed a hotspot-proximal peak overlapping or in addition to the peak from the maximal signal that was the basis of the grouping. These plots demonstrate that certain segments are preferentially sequenced over others. Furthermore, there appear to be two distinct components to this bias: a DSB-proximal preference (addressed in the next panels) that is shared by all hotspots, and a preference for particular segments that are positioned at variable distances away from the hotspot midpoint. The latter bias is likely caused by a requirement for foldback annealing during enrichment for ssDNA during SSDS library preparation.
(E) Sonication as a cause of preferential recovery of DSB-proximal ssDNA fragments.
(F) Simulations establish that a sonication end effect is sufficient to explain the DSB-proximal spike in SSDS coverage. To evaluate sonication as the source of the end effect, we simulated the isolation and sequencing of ssDNA by the published SSDS protocol (see STAR Methods for details). The plot shows, for positions relative to DSB ends, the frequencies of ssDNA recovered by our simulation. This analysis demonstrates that the 100–150 bp adjacent to the DSB site are expected to be preferentially sequenced by SSDS solely as a consequence of sonication bias.

Figure S6. Spatial Relationships Between Sequential Steps in Meiotic Recombination at Known Recombination Hotspots, Related to Figure 6
SPO11 oligos (this study) and strand-specific normalized SSDS coverage (Brick et al., 2012) are shown in comparison to maps of crossover breakpoints and noncrossover gene conversions for 15 of the 16 crossover hotspots shown in **Figure S1B**. (*HS9* is not shown because detailed crossover data were not available.) For crossovers, the orientations of allele-specific primers used to amplify recombinant molecules from sperm DNA are indicated as described in the legend to **Figure 6B**. In the top graph for each hotspot, the horizontal red bar denotes the location of the SPO11-oligo hotspot called at 50× over genome average. In the bottom graph for each hotspot, ticks denote tested DNA sequence polymorphisms. Data for *A3* are reproduced from **Figure 6B** to facilitate side-by-side comparison with the other hotspots. Noncrossover data were available only for the three hotspots depicted at the top. Plots are centered on SPO11-oligo hotspot center, except for *HS18.2*, where the plot is centered on the middle of the crossover breakpoint distribution. The 5001-bp window around *HS59.4* does not encompass all crossovers, which extended on the left side to a polymorphism 427 bp away. Note that the y-axes vary to accommodate the wide range of values among the hotspots. See **Table S2B** and STAR Methods for additional information.

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 **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**.

Supplemental Table Legends

Table S1. Mapping statistics for SPO11-oligo reads, Related to STAR Methods

Table S1A. Mapping statistics for SPO11-oligo reads in five datasets

Table S1B. Mapping statistics for shortened SPO11-oligo reads in three datasets

Table S2. Crossover hotspots identified by pedigree analysis or by sperm typing, Related to Figures 1D, S1B, 6 and S6

Table S2A. Four crossover hotspots on chromosome 1, *A1–A4*, previously delineated by pedigree analysis
Four hotspots on chromosome 1 were previously defined by pedigree analysis in crosses involving haplotypes from C57BL/6 ("B6"), C3H/HeJ ("C3H"), SPRET/EiJ ("SPRET") and CAST/EiJ ("CAST") strains (Kelmenson et al., 2005).

Table S2B. Crossover hotspots previously delineated by sperm typing
In the literature we identified 16 crossover hotspots previously defined by fine-scale analysis of crossover products in sperm isolated from F_1 hybrids of crosses between C57BL/6J ("B6") and either A/J, C3H/HeJ ("C3H") or DBA/2J ("DBA") (Kauppi et al., 2007; Bois, 2007; Wu et al., 2010; Getun et al., 2010; Cole et al., 2010; Getun et al., 2012; de Boer et al., 2015). See **Figure S6** for SPO11-oligo maps, SSDS maps, crossover rates, and noncrossover conversion frequencies in 5001-bp windows around these hotspots.

Table S3. Hotspots called at 50× over genome average in five datasets, Related to STAR Methods

Table S3A. Hotspots in B6 SPO11-oligo map
Hotspot coordinates are from genome build GRCm38/mm10.
Hits: SPO11-oligo counts (RPM)
center: coordinate of maximum SPO11-oligo value within Start and End after smoothing over 201 bp
median: coordinate of weighted mean in SPO11-oligo counts within Start and End

Table S3B. Hotspots in *Atm* wt SPO11-oligo map
Hotspot coordinates are from genome build GRCm38/mm10.
Hits: SPO11-oligo counts (RPM)
center: coordinate of maximum SPO11-oligo value within Start and End after smoothing over 201 bp
median: coordinate of weighted mean in SPO11-oligo counts within Start and End

Table S3C. Hotspots in *Atm* het SPO11-oligo map
Hotspot coordinates are from genome build GRCm38/mm10.
Hits: SPO11-oligo counts (RPM)
center: coordinate of maximum SPO11-oligo value within Start and End after smoothing over 201 bp
median: coordinate of weighted mean in SPO11-oligo counts within Start and End

Table S3D. Hotspots in *Atm* null 1 SPO11-oligo map
Hotspot coordinates are from genome build GRCm38/mm10.
Hits: SPO11-oligo counts (RPM)
center: coordinate of maximum SPO11-oligo value within Start and End after smoothing over 201 bp
median: coordinate of weighted mean in SPO11-oligo counts within Start and End

Table S3E. Hotspots in *Atm* null 2 SPO11-oligo map
Hotspot coordinates are from genome build GRCm38/mm10.
Hits: SPO11-oligo counts (RPM)
center: coordinate of maximum SPO11-oligo value within Start and End after smoothing over 201 bp
median: coordinate of weighted mean in SPO11-oligo counts within Start and End

Table S4. PRDM9 binding motifs identified in 201-bp windows around centers of hotspots called at 50× over genome average, Related to STAR Methods

Table S4A. Instances of the primary 12-bp motif
Coordinates for PRDM9 motif instances and associated hotspots are from genome build GRCm38/mm10.
For hotspots with >1 instance of the motif, only the central-most motif (i.e., closest to hotspot center) is included in this table.
strand: orientation of PRDM9 motif
class: cluster by SPO11-oligo pattern relative to motif
center: coordinate of center of associated SPO11-oligo hotspot

Table S4B. Instances of the secondary 15-bp motif found within hotspots that lacked identifiable primary motif
Coordinates for PRDM9 motif instances and associated hotspots are from genome build GRCm38/mm10.
For hotspots with >1 instance of the motif, only the central-most motif (i.e., closest to hotspot center) is included in this table.
strand: orientation of PRDM9 motif
class: cluster by SPO11-oligo pattern relative to motif
center: coordinate of center of associated SPO11-oligo hotspot

Table S4C. Instances of the 15-bp motif found within hotspots newly arising in *Atm* null 1
Coordinates for PRDM9 motif instances and associated hotspots are from genome build GRCm38/mm10.
For hotspots with >1 instance of the motif, only the central-most motif (i.e., closest to hotspot center) is included in this table.
strand: orientation of PRDM9 motif
class: cluster by SPO11-oligo pattern relative to motif
center: coordinate of center of associated SPO11-oligo hotspot

Table S5. Data underlying chromosome-scale analyses, Related to Figure 4

Table S6. Hotspots called at 50× over genome average in three datasets after read-length shortening, related to STAR Methods

Table S6A. Hotspots in *Atm* wt SPO11-oligo map after read-length shortening
Hotspot coordinates are from genome build GRCm38/mm10.
Hits: SPO11-oligo counts (RPM)
center: coordinate of maximum SPO11-oligo value within Start and End after smoothing over 201 bp
median: coordinate of weighted mean in SPO11-oligo counts within Start and End

Table S6B. Hotspots in *Atm* null 1 SPO11-oligo map after read-length shortening
Hotspot coordinates are from genome build GRCm38/mm10.
Hits: SPO11-oligo counts (RPM)
center: coordinate of maximum SPO11-oligo value within Start and End after smoothing over 201 bp
median: coordinate of weighted mean in SPO11-oligo counts within Start and End

Table S6C. Hotspots in *Atm* null 2 SPO11-oligo map after read-length shortening
Hotspot coordinates are from genome build GRCm38/mm10.
Hits: SPO11-oligo counts (RPM)
center: coordinate of maximum SPO11-oligo value within Start and End after smoothing over 201 bp
median: coordinate of weighted mean in SPO11-oligo counts within Start and End