

# Supplementary Figure 1

*C. e xnd-1* MSSEPIVANLDN-----SMTTEPAPAAFPPISPMSFAFMNEEERAAVKFP---  
*C. b xnd-1* MN--DLAKN-----LLKHSQT TDSAPPISPLSFAFMDEEKAAFKFPASE  
*C. r xnd-1* MN--DIMN CNPNFRSEEP RRQLTLNGVSWPVRTTEETISAPPISPLSFAFMSEERTALNFPGRH

*C. e xnd-1* -----KVDPKPQTAKDDEPSTSQKTL SVDDLLIVDDDETDSPPASSSNYE  
*C. b xnd-1* LAEGDTKKDEEQMSPNKNDNSNSHSSPSTSPDSSSRPRKTFKVDDLLIVDDDEPFSSP---DGYE  
*C. r xnd-1* FQP-----EINRPRNISK SASFTPEPSLDSTPPYNRKTFKIDDLLVDDDED DDDSPPSANSFE

*C. e xnd-1* PKAHIGWASFGDSCLPPPKP-----VKKATDPNLPRRKVYVIRSQNTDKSKSPLVNNQOVT  
*C. b xnd-1* PTAHLGWASFGESIRPPPPTTPPPPPVLSKVVSAPVPLKRKVYVIHQANRAKTKPYDASNHVSA  
*C. r xnd-1* PKAHLGWASF-----EVYVVRQONREK----DTSDHVVTH

*C. e xnd-1* LEKAQNSPKNPNPVVSK---PIVLTDSDEDVDVGFDEEEEAIKIMADPTR-PPDLPPQRSLSR  
*C. b xnd-1* TKRVSGSPVSP TSKLAKKD VADVSSDSDADVDVGLDEEESTLLLDVSDSANPPDLPPQSSLGSR  
*C. r xnd-1* NDNMPGPPKEDSPITT--INDIS--DSDADVDVVGIDEDNS-ISIEANEDGNPPDLPPQSSIGSR

▽

*C. e xnd-1* FESNRTRKRDIFRNSYDSDEEEFLRSRYQRAVTPPPILERQSGSTRESVSEPESEKILEEDATLGSE  
*C. b xnd-1* FG-LKSKRDNRRG-YDSEDDDPSSLRYANNFTPPRLEPQTTE-----NQNRDLPKKIDED--  
*C. r xnd-1* FS-YKSKKARYR--YDSEEEEMINARFLSNISPPPLEAQATSGS ISSAKDNAGADEEKKCEDG--

*C. e xnd-1* HTERVGRKIELEEINPSQLLRTKISASAIPI LPLSKSIMERKKVALEMTKNAVIRQANNFRPKAKN  
*C. b xnd-1* ---SVELRS---SLDAPQSLKSNKPG---PVLPLSKSIMEQKVALGMTRNAVIKKVDSKSTTKN  
*C. r xnd-1* ---QIEQR LGLEELNSAQILKTKVSSSTIPV LPLSKSIMERKKVALEMTRHAVIKKYNSP--SFKN

◆

*C. e xnd-1* STVAPKSVQIPAYNHKTPMTFYSNMAAPA--FVKGVNGRCYRCAEIQKPVDMSSFRFVDDSTVIAI  
*C. b xnd-1* FTCAPRSVQIPAYTDKAPMTLYSNVKNKPG-AFHKDARGRCVRCRDRSPVDMSSFKFVDDSTVISV  
*C. r xnd-1* FTVAPKSVQIPAYNDNTPMTLYSNINKPLNSISKDVRGRCVRCRDRSPVDMSTFKFIDDTTVVSV

↓

*C. e xnd-1* RALLDQTRVMVARTAIWNREYAKRLGDRAAGTVWQKPDEVSAQMTGF SATVRRVIEIANMSIIP  
*C. b xnd-1* RALLDQTRVMIARTAVWNREYSKRLGDRAAGTVWQKPDEVTAQMTGF SATVRRVIEIANLSIIP  
*C. r xnd-1* RALLDQTRVMIARTAMWNREYSKRLGDRAAGTVWQRPDEVTAQMTGF SATVRRVIEIANLSIIP

↓

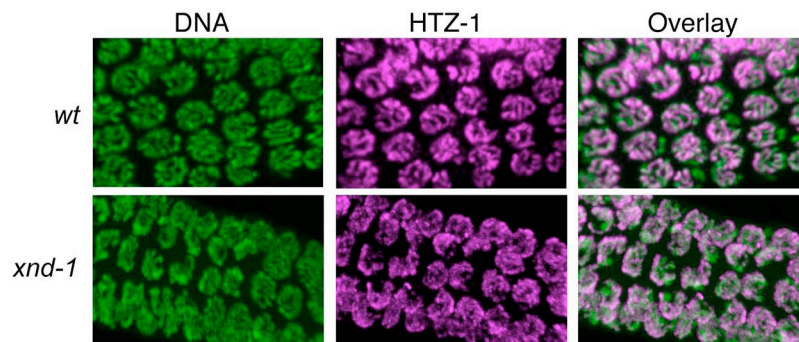
*C. e xnd-1* KCADRVGVS RNELTSLKSSFGSEKFCGQTMRA PHVLTQFYSVNQASMAARNREGSDGPSSSAARPVG  
*C. b xnd-1* RCADRIGVSKNELASLKSAFENEKFFGQTMRTPHVLTHTHYSAKSQNSAN-KGTTNAHG PPLLPRIG  
*C. r xnd-1* RCADRIGVSKNELVSLKSTFGTEKFLGQTMRTPHVLTHTHYAQKSQSIAGSRPSSSNVTPPIPRMG

*C. e xnd-1* RPPTTQP VETAVEKKNDEDEKRHHPLTNFT-----TASTSSQNLQD-QPPKNTVLRW  
*C. b xnd-1* RPP-----AQKVD-EEEEKRHHPLTNYLPSTSLQS----TSQETSTSPDITVPPLKKGVLRW  
*C. r xnd-1* NPP-----VETEKNEDED RHHPLTYYPSSSFPSSTSFQSSSPSVSPPESLVPPLKKTVLRW

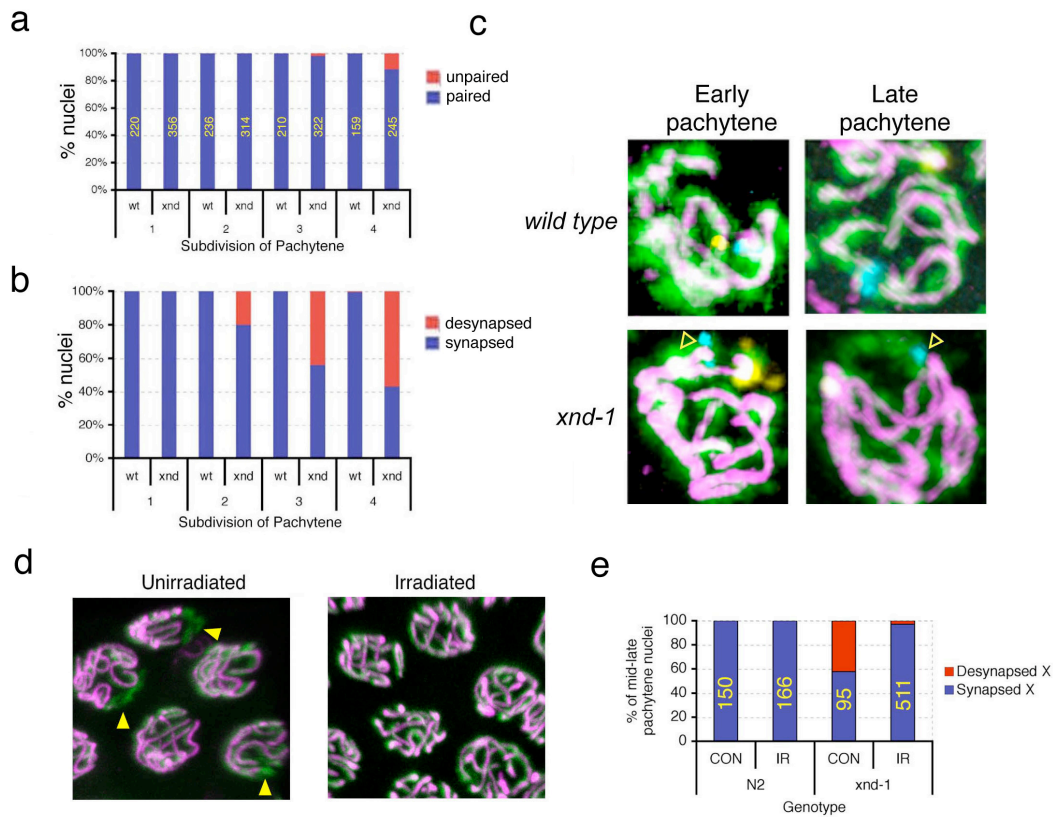
*C. e xnd-1* SNITTPRILRPPTLKTPYSLPRTAVIPVMKKKQPE-----IEQVQPKKDT-----TA  
*C. b xnd-1* TSIQNPRILRTA--HAPYPLPRMAVIPVLKKKDVATTSETASTSSGPLYVP PKKETKKDQAAAPT  
*C. r xnd-1* TSIQTPRILRPP--NPSYVVPKMAVIPVMKKVDST-----PCKKE-----IVM

*C. e xnd-1* SLVRKRGRPRKEKPLEVQ-SPRKGLYLRFK SCTKYIVRTPIDKSVQETVNYLLDEVDKRASTSAT  
*C. b xnd-1* TGPRKRGRPKVKPADG--IPSRSTEMTLRSRKE---IPIDQVQGLVQSLISRISGEASG---  
*C. r xnd-1* TGPRKRGRPRKIRPELTVRPIQPSTRRLRSYKEL--CEIDEDVVDVVEGLVSKVSGTFSINTS

**Supplementary Figure 1.** XND-1 protein sequences from *C.elegans*, *C. briggsae*, and *C. remanei* are compared. Identical amino acids between the three proteins are red, and conserved charge amino acids are highlighted in yellow. The out-of-frame *ok709* allele begins at the open arrowhead; *ok709* encodes an additional 64 amino acids followed by a termination codon. The diamond indicates the amino acid at which the out-of-frame *ok708* deletion begins; *ok708* encodes an additional 20 amino acids followed by a termination codon. The line above the sequences indicates the amino acids that are absent in alternatively spliced transcripts of the *xnd-1* gene. The amino acids between the arrows contain a potential metal-binding (relevant amino acids shaded in blue) domain that may mediate chromatin-binding and/or protein-protein interactions (personal communication, E. Koonin).

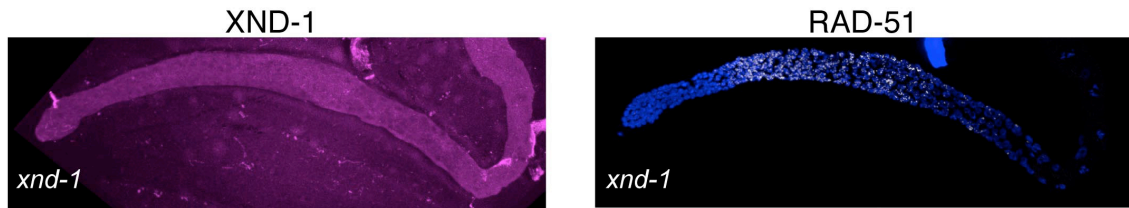


**Supplementary Figure 2.** HTZ-1 staining is unaffected in *xnd-1* mutant germline nuclei. *wt* (top), *xnd-1* (bottom) germlines co-stained with anti-HTZ-1 antibodies (magenta) and DAPI (green). In both *wt* and *xnd-1*, HTZ-1 appears excluded from one chromosome (the X) as it is preferentially localized to the autosomes.

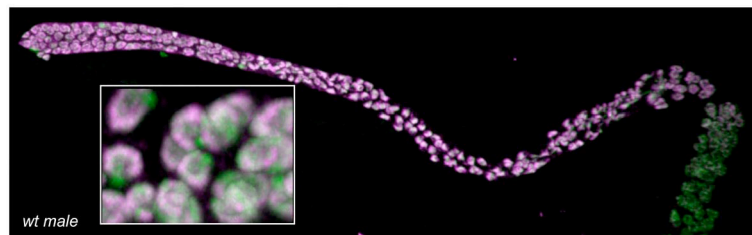


**Supplementary Figure 3.** The SC disassembles on X chromosomes that do not receive a DSB in *xnd-1* mutants. (a) Quantification of pairing and (b) synapsis during pachytene in wild type and *xnd-1* (pairing and synapsis were quantified simultaneously: regions 1-4, *wt*, N=220, 236, 210, 159 nuclei; *xnd-1*, N=356, 314, 322, 245 nuclei). The X chromosome desynapses in ~50% of *xnd-1* late pachytene nuclei. (c) Images of early pachytene and late pachytene nuclei in *wild type* (top) and *xnd-1* (bottom) reveal the loss of the SC on the X. DAPI (green), FISH probes to the 5S locus on Chromosome V (yellow) and the right end of the X chromosome (cyan, yellow arrows) and anti-SYP-1 antibody (magenta). (d, e) Desynapsis is rescued by ionizing radiation (IR) which provides exogenous DSBs. (d) Unirradiated *xnd-1* and irradiated *xnd-1* germlines stained for DNA (green) and anti-SYP-1 antibodies (magenta) demonstrate that the desynapsis defect in *xnd-1* is rescued by exogenous break formation. Yellow arrowheads mark the X chromosome in the unirradiated samples. (e) Quantification of SC status on the X chromosome in control and irradiated animals 16 hrs post-IR (*wt* +/- IR, N= 150, 166; *xnd-1* +/- IR, N= 95, 511).

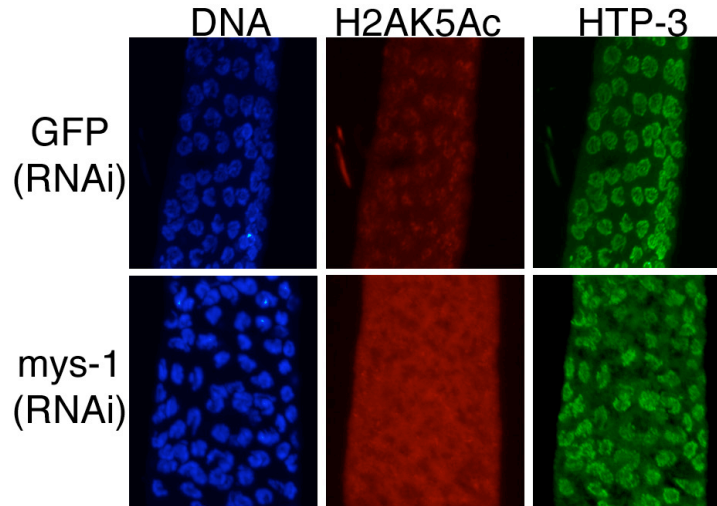




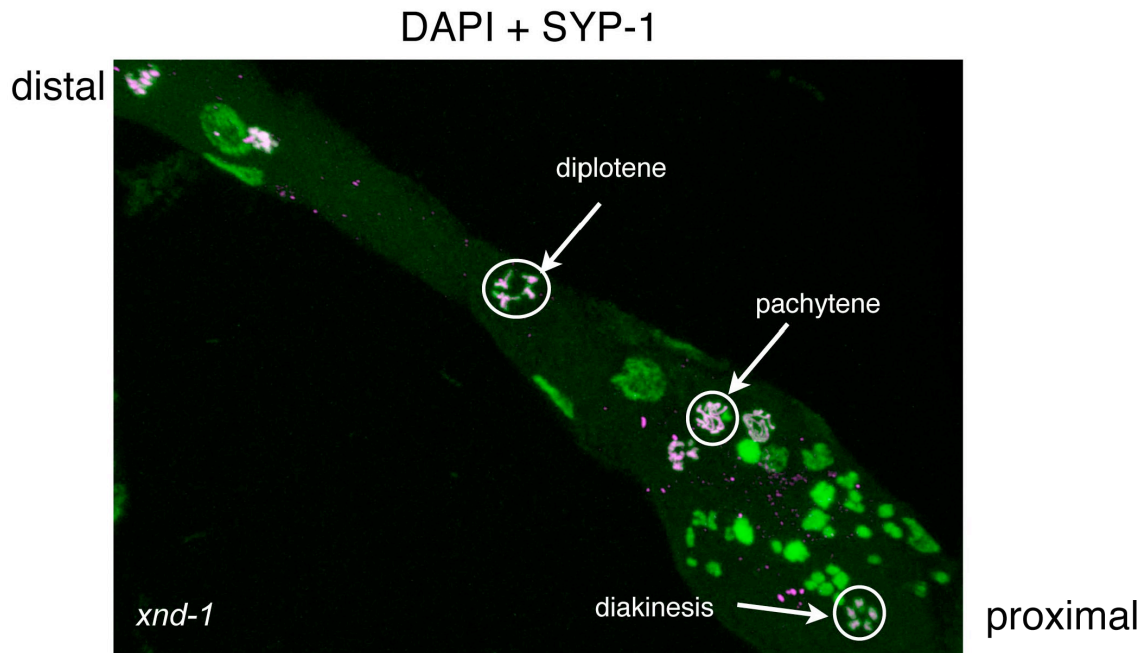
**Supplementary Figure 4.** XND-1 antibodies are specific. anti-XND-1 antibodies do not stain *xnd-1* mutant germlines. *xnd-1* mutant germlines were dissected and co-stained with anti-XND-1 antibodies (left panel, magenta) and anti-RAD-51 antibodies (right panel, white; DAPI, blue).



**Supplementary Figure 5.** XND-1 is enriched on autosomes in *wild type* males. Germlines from *wild type* males were dissected and stained with DAPI (green) and anti-XND-1 antibodies (magenta). As in *wild type* hermaphrodites, XND-1 is preferentially localized to the autosomes (inset).



**Supplementary Figure 6.** *mys-1* treated animals have decreased levels of H2AK5Ac. *GFP(RNAi)* or *mys-1(RNAi)* treated *xnd-1* mutant gonads were dissected, fixed, and stained on the same slides and imaged with identical settings on a Nikon A1 confocal microscope. The 1:2500 dilution of the anti-H2AK5Ac was used in these experiments so that small perturbations in signal could be seen. Anti-Htp-3 immunolocalization shows that the *mys-1(RNAi)* treated samples are not impervious to antibody and that the diffuse red signal with the anti-H2AK5Ac antibody corresponds to background staining.



**Supplementary Figure 7.** Sterile *xnd-1* mutant germlines display meiotic packaging defects. Dissected *xnd-1* germlines were stained with anti-SYP-1 antibody (magenta) and DAPI (green). As shown by the white circles, meiotic progression is aberrant with defects in the temporal and spatial organization of late meiotic events.

**Supplementary Table 1. Crossover distribution on chromosome I from oocytes**

Interval (Mb)		0.17 -1.91	1.91-4.59	4.59-10.72	10.72-12.05	12.05-14.68	<i>N</i>
<b>oocytes</b>	<i>wt</i>	8.9 (21)	10.6 (25)	0.0 (0)	5.5 (13)	26.3 (62)	236
	<i>xnd-1</i>	6.5 (15)	16.9 (39)	21.2** (49)	2.2 (5)	1.3** (3)	231

Values are map units for each interval (number of COs per interval)

The change in crossover distribution between *N2* and *xnd-1* is statistically significant:

$$\chi^2(5, N=111)=24.000, p < .001$$

\*, \*\*Significant difference in map size of the interval between *wild type* and *xnd-1* (\* $p < 0.05$ ; \*\* $p < 0.001$ )

**Supplementary Table 2. Crossover distribution on the X chromosome**

Interval (Mb)		0.06 - 2.34	2.34 - 4.13	4.13 - 6.88	6.88 - 10.64	10.64 - 14.26	14.26 - 17.22	<i>N</i>
<b>oocytes</b>	<i>wt</i> <sup>1</sup>	7.3 (27)	9.5 (35)	6.5 (24)	7.6 (28)	9.8 (36)	10.3 (38)	--
	<i>xnd-1</i>	1.5* (6)	2.5* (10)	4.5 (18)	8.7** (35)	6.5 (26)	1.7** (7)	402

Values are map units for each interval (number of COs per interval)

The change in crossover distribution between *N2* and *xnd-1* is statistically significant:

$$\chi^2(6, N=102)=50.2, p < .001$$

<sup>1</sup>Data from Lim et al.<sup>1</sup>

\*, \*\*Significant difference in map size of the interval between *wild type* and *xnd-1* (\* $p < 0.05$ ; \*\* $p < 0.001$ )

**Supplementary Table 3. Crossover distribution on chromosome I from sperm**

Interval (Mb)		0.17 -1.91	1.91-4.59	4.59-10.72	10.72-12.05	12.05-14.68	<i>N</i>
<b>sperm</b>	<i>wt</i>	14.0 (46)	12.8 (42)	6.1 (20)	4.3 (14)	13.4 (44)	328
	<i>xnd-1</i>	5.7** (20)	17.5* (61)	22.6** (79)	0.9* (3)	3.4** (12)	349

Values are map units for each interval (number of COs per interval)

The change in crossover distribution between *N2* and *xnd-1* is statistically significant:  $\chi^2(5, N=166)=207, p < .001$

\*, \*\*Significant difference in map size of the interval between *wild type* and *xnd-1* (\* $p < 0.05$ ; \*\* $p < 0.001$ )

**Supplementary Table 4. *xnd-1* viability and % HIM**

<b>genotype</b>	<b>#embryos</b>	<b>%hatched</b>	<b>%males</b>
<b><i>N2</i></b>	3347	99.9	0.06
<b><i>ok708 M<sup>+</sup>Z<sup>-</sup></i></b>	2502	87.4	13.8
<b><i>ok708 M<sup>-</sup>Z<sup>-</sup></i></b>	3263	75.5	23.2
<b><i>ok709 M<sup>+</sup>Z<sup>-</sup></i></b>	2132	69.4	12.3
<b><i>ok709 M<sup>-</sup>Z<sup>-</sup></i></b>	2288	65.5	19.9
<b><i>+/+;sEx826</i></b>	1502	ND	24.7
<b><i>rde-2;sEx826</i></b>	2047	ND	0.49

**Supplementary Table 5. Number of RAD-51 foci in *wt* and *xnd-1* meiotic germline nuclei.**

REGION:	TZ	1	2	3	4	5	6
<b>N2#1</b>							
0	86	6	1	1	38	44	21
1-2	30	53	9	24	16	5	1
3-4	0	23	51	30	7	0	0
5-7	0	1	17	16	4	0	0
<b>N2#2</b>							
0	33	2	0	0	2	29	20
1-2	8	27	6	6	11	4	0
3-4	0	8	31	23	9	0	0
5-7	0	0	10	14	13	0	0
<b>N2#3</b>							
0	43	7	0	0	0	34	0
1-2	8	26	26	12	11	3	0
3-4	0	1	29	25	21	0	0
5-7	0	0	1	12	4	0	0
<b>xnd-1#1</b>							
0	32	1	1	0	2	17	25
1-2	2	2	19	9	20	25	12
3-4	0	17	29	12	17	5	1
5-7	0	22	12	16	4	0	0
<b>xnd-1#2</b>							
0	34	0	0	0	2	10	8
1-2	14	15	0	16	12	8	1
3-4	0	8	7	11	7	0	0
5-7	0	5	14	2	0	0	0
<b>xnd-1#3</b>							
0	35	1	0	4	12	21	10
1-2	10	10	2	19	14	7	3
3-4	1	22	12	9	5	1	0
5-7	0	14	14	1	0	0	0
<b>xnd-1#4</b>							
0	37	1	0	3	7	22	17
1-2	16	0	1	29	30	16	1
3-4	7	36	19	34	6	3	0
5-7	2	47	46	22	1	1	0
<b>xnd-1#5</b>							
0	25	2	0	0	2	8	28
1-2	13	7	7	9	8	27	15
3-4	2	17	18	17	24	8	2
5-7	1	16	15	4	4	1	0

**Supplementary Table 6. XND-1 localization in meiotic mutants.**

<b>Genotype</b>	<b>Function</b>	<b>XND-1 temporal staining pattern*</b>	<b>XND-1 exclusion from X chromosome</b>	<b>Reference</b>	
<i>chk-2(me64)</i>	pairing	wild type	yes	2	
<i>him-8(e1489, me4, tm611)</i>		wild type	yes	3	
<i>prom-1(ok1140)</i>		wild type	yes	4	
<i>sun-1(jf18)</i>		wild type	yes	5	
<i>him-3(e1147, e1256)</i>	Axial element/SC formation	wild type	yes	6	
<i>htp-1(gk150)</i>		wild type	yes	7,8	
<i>rec-8(ok978)</i>		wild type	yes	9	
<i>syp-1(me17)</i>		wild type	yes	10	
<i>syp-2(ok307)</i>		wild type	yes	11	
<i>him-6(e1104, e1423)</i>	DSB formation and break repair	wild type	yes	12	
<i>him-14(it44)</i>		wild type	yes	13	
<i>him-17(ok424)</i>		wild type	yes	14	
<i>mre-11(ok179)</i>		wild type	yes	15	
<i>msh-2(ok2486)</i>		wild type	yes	16	
<i>msh-5(me23)</i>		wild type	yes	17	
<i>rad-51(lg8701)</i>		wild type	yes	18	
<i>spo-11(me44)</i>		wild type	yes	19	
<i>dpy-28(y1)</i>	Crossover Control	wild type	yes	20	
<i>dpy-27(y56)</i>	Dosage compensation	wild type	yes	21	
<i>dpy-30(y228)</i>		wild type	yes	22	
<i>him-1(e879)</i>	Chromatin structure	wild type	yes	23	
<i>his-72(tm2066)</i>		wild type	yes	24	
<i>hpl-1(tm1621)</i>		wild type	yes	25	
<i>hpl-2(tm1489)</i>		wild type	yes	25	
<i>htz-1(tm2469)</i>		wild type	yes	26	
<i>met-2(n4256)</i>		wild type	yes	27	
<i>mrg-1(qa6200)</i>		wild type	yes	28	
<i>rbr-2(ok2544)</i>		wild type	yes	29	
<i>set-2(tm1630)</i>		wild type	yes	30	
<i>him-5(e1467, ok1896)</i>		Unknown	wild type	yes	23

\*Nuclear staining pattern is consistent with particular substage of prophase I, rather than position in germline.



**Supplementary Table 7. *xnd-1* mutants do not desilence transgene arrays.**

	<b><i>let-868.1::GFP<sup>31</sup></i> expression</b>
<b><i>wild type</i></b>	-
<b><i>xnd-1</i></b>	-
<b><i>mes-4</i> (RNAi)</b>	++

**Supplementary Movie 1.** RAD-51 foci are detected on X chromosomes in wild type nuclei. 3D reconstructions of confocal stacks showing accumulation of RAD-51 foci (magenta) in wild type animals treated with *rad-54(RNAi)*. Autosomes are labeled with anti-HTZ-1 antibodies (cyan), all chromosomes are labeled with DAPI (green), therefore the X chromosome is revealed by the absence of colocalization.

**Supplementary Movie 2.** RAD-51 foci cannot be detected on a subset of X chromosomes in *xnd-1* nuclei. 3D reconstructions of confocal stacks showing accumulation of RAD-51 foci (magenta) in *xnd-1* mutant animals treated with *rad-54(RNAi)*. Autosomes are labeled with anti-HTZ-1 antibodies (cyan), all chromosomes are labeled with DAPI (green), therefore the X chromosome is revealed by the absence of colocalization. Yellow arrows indicate X chromosomes that do not have RAD-51 foci; blue arrows indicate X chromosomes with RAD-51 foci.

**Supplementary Movie 3.** XND-1 is enriched on autosomes. 3D reconstructions of confocal stacks from the mid-pachytene region of wild type animals labeled with anti-Histone H4K12Ac (magenta), anti-XND-1 antibody (cyan); and DAPI (green). Note the complete colocalization of the antibodies and the absence of staining on the X chromosome (yellow arrows).

## Supplementary References

- 1 Lim, J. G., Stine, R. R. & Yanowitz, J. L. Domain-specific regulation of recombination in *Caenorhabditis elegans* in response to temperature, age and sex. *Genetics* **180**, 715-726 (2008).
- 2 MacQueen, A. J. & Villeneuve, A. M. Nuclear reorganization and homologous chromosome pairing during meiotic prophase require *C. elegans* chk-2. *Genes Dev* **15**, 1674-1687 (2001).
- 3 Phillips, C. M. *et al.* HIM-8 binds to the X chromosome pairing center and mediates chromosome-specific meiotic synapsis. *Cell* **123**, 1051-1063 (2005).
- 4 Jantsch, V. *et al.* *Caenorhabditis elegans* prom-1 is required for meiotic prophase progression and homologous chromosome pairing. *Mol Biol Cell* **18**, 4911-4920 (2007).
- 5 Penkner, A. *et al.* The nuclear envelope protein Matefin/SUN-1 is required for homologous pairing in *C. elegans* meiosis. *Dev Cell* **12**, 873-885 (2007).
- 6 Zetka, M. C., Kawasaki, I., Strome, S. & Muller, F. Synapsis and chiasma formation in *Caenorhabditis elegans* require HIM-3, a meiotic chromosome core component that functions in chromosome segregation. *Genes Dev* **13**, 2258-2270 (1999).
- 7 Couteau, F. & Zetka, M. HTP-1 coordinates synaptonemal complex assembly with homolog alignment during meiosis in *C. elegans*. *Genes Dev* **19**, 2744-2756 (2005).
- 8 Martinez-Perez, E. & Villeneuve, A. M. HTP-1-dependent constraints coordinate homolog pairing and synapsis and promote chiasma formation during *C. elegans* meiosis. *Genes Dev* **19**, 2727-2743 (2005).
- 9 Pasierbek, P. *et al.* A *Caenorhabditis elegans* cohesion protein with functions in meiotic chromosome pairing and disjunction. *Genes Dev* **15**, 1349-1360 (2001).
- 10 MacQueen, A. J., Colaiacovo, M. P., McDonald, K. & Villeneuve, A. M. Synapsis-dependent and -independent mechanisms stabilize homolog pairing during meiotic prophase in *C. elegans*. *Genes Dev* **16**, 2428-2442 (2002).
- 11 Colaiacovo, M. P. *et al.* Synaptonemal complex assembly in *C. elegans* is dispensable for loading strand-exchange proteins but critical for proper completion of recombination. *Dev Cell* **5**, 463-474 (2003).
- 12 Wicky, C. *et al.* Multiple genetic pathways involving the *Caenorhabditis elegans* Bloom's syndrome genes him-6, rad-51, and top-3 are needed to maintain genome stability in the germ line. *Mol Cell Biol* **24**, 5016-5027 (2004).
- 13 Zalevsky, J., MacQueen, A. J., Duffy, J. B., Kempthues, K. J. & Villeneuve, A. M. Crossing over during *Caenorhabditis elegans* meiosis requires a conserved MutS-based pathway that is partially dispensable in budding yeast. *Genetics* **153**, 1271-1283 (1999).
- 14 Reddy, K. C. & Villeneuve, A. M. *C. elegans* HIM-17 links chromatin modification and competence for initiation of meiotic recombination. *Cell* **118**, 439-452 (2004).
- 15 Chin, G. M. & Villeneuve, A. M. *C. elegans* mre-11 is required for meiotic recombination and DNA repair but is dispensable for the meiotic G(2) DNA damage checkpoint. *Genes Dev* **15**, 522-534 (2001).
- 16 Degtyareva, N. P. *et al.* *Caenorhabditis elegans* DNA mismatch repair gene msh-2 is required for microsatellite stability and maintenance of genome integrity. *Proc Natl Acad Sci U S A* **99**, 2158-2163 (2002).
- 17 Kelly, K. O., Dernburg, A. F., Stanfield, G. M. & Villeneuve, A. M. *Caenorhabditis elegans* msh-5 is required for both normal and radiation-induced meiotic crossing over but not for completion of meiosis. *Genetics* **156**, 617-630 (2000).
- 18 Alpi, A., Pasierbek, P., Gartner, A. & Loidl, J. Genetic and cytological characterization of the recombination protein RAD-51 in *Caenorhabditis elegans*. *Chromosoma* **112**, 6-16 (2003).

- 19 Dernburg, A. F. *et al.* Meiotic recombination in *C. elegans* initiates by a conserved mechanism and is  
dispensable for homologous chromosome synapsis. *Cell* **94**, 387-398 (1998).
- 20 DeLong, L., Casson, L. P. & Meyer, B. J. Assessment of X chromosome dosage compensation in  
Caenorhabditis elegans by phenotypic analysis of lin-14. *Genetics* **117**, 657-670 (1987).
- 21 Plenefisch, J. D., DeLong, L. & Meyer, B. J. Genes that implement the hermaphrodite mode of  
dosage compensation in Caenorhabditis elegans. *Genetics* **121**, 57-76 (1989).
- 22 Hsu, D. R. & Meyer, B. J. The dpy-30 gene encodes an essential component of the Caenorhabditis  
elegans dosage compensation machinery. *Genetics* **137**, 999-1018 (1994).
- 23 Broverman, S. A. & Meneely, P. M. Meiotic mutants that cause a polar decrease in recombination on  
the X chromosome in Caenorhabditis elegans. *Genetics* **136**, 119-127 (1994).
- 24 Ooi, S. L., Priess, J. R. & Henikoff, S. Histone H3.3 variant dynamics in the germline of  
Caenorhabditis elegans. *PLoS Genet* **2**, e97 (2006).
- 25 Couteau, F., Guerry, F., Muller, F. & Palladino, F. A heterochromatin protein 1 homologue in  
Caenorhabditis elegans acts in germline and vulval development. *EMBO Rep* **3**, 235-241 (2002).
- 26 Whittle, C. M. *et al.* The genomic distribution and function of histone variant HTZ-1 during *C.*  
*elegans* embryogenesis. *PLoS Genet* **4**, e1000187 (2008).
- 27 Andersen, E. C. & Horvitz, H. R. Two *C. elegans* histone methyltransferases repress lin-3 EGF  
transcription to inhibit vulval development. *Development* **134**, 2991-2999 (2007).
- 28 Takasaki, T. *et al.* MRG-1, an autosome-associated protein, silences X-linked genes and protects  
germline immortality in Caenorhabditis elegans. *Development* **134**, 757-767 (2007).
- 29 Christensen, J. *et al.* RBP2 belongs to a family of demethylases, specific for tri- and dimethylated  
lysine 4 on histone 3. *Cell* **128**, 1063-1076 (2007).
- 30 Simonet, T., Dulermo, R., Schott, S. & Palladino, F. Antagonistic functions of SET-2/SET1 and  
HPL/HP1 proteins in *C. elegans* development. *Dev Biol* **312**, 367-383 (2007).
- 31 Kelly, W. G., Xu, S., Montgomery, M. K. & Fire, A. Distinct requirements for somatic and germline  
expression of a generally expressed Caenorhabditis elegans gene. *Genetics* **146**, 227-238 (1997).