## Supplementary Materials for:

# DNA-driven condensation assembles the meiotic DNA break machinery

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**Other Supplementary Materials for this manuscript include the following:**

## Supplementary Discussion

### 1. Previously unclear but evolutionarily conserved relationships between Rec114, Mei4, and Mer2.

Previous work resulted in Rec114, Mei4, and Mer2 being loosely grouped together as a functionally related subset of the DSB-essential proteins, but it was never clear precisely how they interacted with one another[2]. Mer2 foci are independent of Rec114 and Mei4, but Rec114 and Mei4 appear to largely depend on one another and on Mer2 for their chromatin association[11,12,16]. These different dependencies are also the case in mice[21,25,50]. Thus, our results provide a molecular framework for understanding these previously unclear relationships: heterotrimeric Rec114–Mei4 complexes and Mer2 homotetramers are biochemically distinct entities that each bind cooperatively to DNA, but they collaborate in DSB formation in the context of mixed, DNA-dependent condensates.

Chromatin-associated Rec114, Mei4 and Mer2 foci localize to chromosome axes in *S. cerevisiae*, *S. pombe* (Rec7, Rec24, and Rec15) and mice (REC114, MEI4 and IHO1), and probably also in other organisms including plants (PHS1, PRD2 and PRD3/PAIR1)[11,12,14-16,25,26,29,30,51,52]. We therefore consider that the DNA-driven condensation we uncovered here is likely to be a fundamental, evolutionarily conserved property of these proteins.

### 2. Mechanisms of condensate assembly: growth by fusion vs. exchange with soluble protein pools.

We envisioned several scenarios to account for how the condensates assemble (**Extended Data Fig. 5a**): (i) Nucleation could be limiting, with focus growth resulting principally from incorporation of protein from soluble pools. (ii) Frequent nucleation events could occur at early time points leading to large numbers of small foci, whereupon some foci dissolve and others grow by incorporation from soluble pools. (iii) Frequent nucleation events could occur leading to numerous small foci that then collide and fuse to yield fewer, larger foci. Results of our DNA immobilization experiments indicated that focus growth in this assay is determined largely by fusion (**Fig. 2d, Extended Data Fig. 5b**). However, the rates of dynamic exchanges from soluble pools are likely to be a function of experimental conditions, and such exchange may be particularly important *in vivo* where viscous drag on chromosomes may inhibit fusion.

Under these experimental conditions, the condensates appear to efficiently deplete essentially all of the free protein from solution, inferred from the paucity of free protein particles in the AFM images with plasmid DNA (**Fig. 2c and Extended Data Fig. 2g**) and inability of fluorescence intensity to recover after foci are photobleached (**Extended Data Fig. 5c**). *In vivo*, where new protein synthesis will provide ongoing input of free protein complexes, focus growth by addition of new protein is likely to occur for much of prophase I. There may also be active disassembly processes *in vivo* that release protein complexes from condensates, not recapitulated in our *in vitro* system.

### 3. Phase separation and the molecular basis of Rec114–Mei4–Mer2 assemblies.

Two types of mechanisms have been proposed for self-assembly of chromatin subcompartments by phase separation: one involves a multivalent chromatin binder, the second

involves a self-associating chromatin binder. Both types can induce phase separation of nuclear bodies[53].

Both Rec114–Mei4 and Mer2 are multimeric, so they may be multivalent binders that crosslink the DNA scaffold. Such interactions are predicted to fall apart upon removal of the DNA, as we observed, whereas protein droplets of self-associating chromatin binders should persist[53]. However, multivalent DNA binding alone cannot explain the highly cooperative assembly of Rec114–Mei4 and Mer2 that restricts itself to just a subset of available DNA molecules, so protein self-association must also be invoked. Protein self-association is apparent from the ability of pre-existing Mer2 condensates to incorporate the DNA binding-defective Mer2-KRRR protein, which by itself is incapable of condensate formation (**Extended Data Fig. 6e**). These self interactions are presumably too weak to persist upon DNA removal, at least at the protein concentrations we tested. The context-dependent effects of $Mg^{2+}$ on Mer2 condensates might reflect direct effects on protein-protein interactions or, non-exclusively, modulation of protein-DNA interactions that affect the balance between nucleation of new condensates and growth of condensates by fusion or addition of soluble protein.

Depending on the strength of the interactions, phase-separated systems can exist as liquid, gel-like or even solid forms, and transitions from liquid to solid may occur spontaneously[35,37-39]. We found that the reversibility of the Rec114–Mei4–Mer2 condensates decreases over time, influenced by molecular crowding, potentially consistent with the progressive transitions to gel-like or solid states.

### 4. A model for the assembly of the meiotic break machinery by DNA-driven Rec114–Mei4–Mer2 condensation

A longstanding question has been how the different DSB proteins promote Spo11 activity. On the basis of their axis-association and the physical links between Mer2, Spp1 and histone H3 lysine 4 trimethylation (which marks regions of preferential DSB activity within chromatin loops), Rec114, Mei4 and Mer2 have been proposed to form part of the tethered loop-axis structure that is thought to assemble in preparation for Spo11-mediated DNA cleavage[3,11,12,15-19]. Our data adds to this model by highlighting the organizing role of Rec114–Mei4 and Mer2 in the assembly of punctate clusters. We propose that it is these RMM clusters that provide the structural assembly for recruitment of Spo11 and other regulatory components, and that it is in the context of these structures that the tethered loop-axis configuration is eventually adopted (**Extended Data Fig. 9a**). Binding of Mer2 to both Spp1 and Hop1 has recently been recapitulated *in vitro*, confirming the direct role of Mer2 in the loop-axis connection[54]. In addition, Mer2 was found to bind histone octamers, suggesting that condensates may incorporate chromatinized templates, not only naked DNA.

Hyperstoichiometric condensates would be expected to recruit multiple core complexes, which is supported by our *in vitro* data and the detection of Spo11 foci *in vivo*[55]. Each cluster may thus be expected to be capable of forming multiple DSBs. Indeed, closely spaced pairs of DSBs on the same chromatid (double cuts) occur at a much higher frequency than expected by chance, estimated to account for ~10% of total Spo11 activity in wild-type cells[41]. The distance between pairs of DSBs ranges from about 30 to >100 bp with a 10-bp periodicity, corresponding to the pitch of the double helix. This periodicity can be explained if two Spo11 complexes engage DNA in the same orientation, which in turn can be explained if the complexes are

constrained on a surface[41]. In the light of our findings, we propose that Rec114–Mei4–Mer2 condensates provide platforms that recruit and display co-oriented arrays of Spo11 complexes that then capture and break loop DNA (**Extended Data Fig. 9a**). The core complex might be mostly at the surface of the condensates or might be active only when exposed on the surface.

**5. Hotspot competition and DSB interference.**

Our model has implications for numerical and spatial control of DSB patterning. It has long been appreciated that the presence of a strong hotspot can reduce DSB activity of a neighboring hotspot. This phenomenon—hotspot competition—is a population-average effect[56-61]. Another process—DSB interference—is observed when cleavage of individual DNA molecules within a cell is considered: the presence of a DSB decreases the chances of another DSB occurring nearby, at distances up to a hundred kilobases[40].

Hotspot competition and DSB interference are genetically separable in *S. cerevisiae* because DSB interference depends on the DNA-damage response kinase Tel1[40], while hotspot competition does not[61]. Tel1 governs a *cis*-acting DSB-dependent negative feedback loop[62,63]. In a *tel1* mutant, not only is DSB interference eliminated, but negative interference is detected over distances on the ~10 kb scale, meaning that coordinated cutting of the same chromatid at adjacent hotspots is observed at high frequency[40].

Our model for locally coordinated break formation through the recruitment of multiple Spo11 complexes within Rec114–Mei4–Mer2 condensates provides a molecular explanation for these behaviors. Hotspot competition could be implemented prior to break formation and can be explained if hotspots compete for poorly diffusing and locally limiting factors[56]. Indeed, Rec114–Mei4–Mer2 have been proposed to constitute this factor based on the observation that they associate to the chromosome axes, which may therefore limit their diffusion within the nucleus, hence their availability[16]. The highly cooperative DNA-driven condensation described here provides a molecular basis to understand how this might work, because the nucleation of a condensate would cause a local depletion of Rec114–Mei4 and Mer2 proteins, reducing the probability of another nucleation event (**Extended Data Fig. 9b**). After a DSB is made, Tel1 is envisioned to act both within and between adjacent condensates to suppress additional DSBs nearby (**Extended Data Fig. 9b**).

Alternatively, it has been proposed that hotspot competition and DSB interference could reflect association of a cluster of several chromatin loops with a limited-catalytic-capacity DSB-forming assembly[3,40]. In this view, loops within a cluster compete with one another for access to the DSB machinery. Hotspot clustering has also been proposed as a means to control DSB patterning in *S. pombe*[64]. Once activated, the machinery has potential to make multiple cuts, but this is suppressed by Tel1 after the first DSB is made. How such loop clustering might occur and how it might be integrated with the DSB machinery have been a matter of conjecture, and mechanisms behind speculated roles of Rec114, Mei4, and Mer2 were likewise unclear[3]. The macromolecular condensates we document could readily account for all of these properties. The synaptonemal complex is another example of how phase separation is proposed to govern spatial patterning of meiotic recombination[65].

## 6. Macromolecular condensates as a platform to integrate DSB formation with repair.

An intriguing possibility is that forming DSBs in the context of a superstructure may facilitate control of subsequent repair. The condensates themselves, being axis-associated, could hold the bases of the broken DNA loop, providing a coherent unit that would prevent the diffusion of the DNA ends from each other (**Extended Data Fig. 9c**, bottom panel). In addition, one or both DSB ends might remain embedded within the condensate via the persistence of Spo11-oligo complexes that might cap the ends after resection. End-capping by Spo11[66] has recently received support from patterns of recombination intermediates detected in mice[67], and is consistent with the observation that Spo11 binds tightly to DNA ends *in vitro*, even in the absence of a covalent link[10].

Following DSB formation, one might imagine that the condensates hand over or evolve into recombination nodules where DSB repair takes place. In *Sordaria macrospora*, the Mer2 ortholog Asy2 re-localizes from the axis to the central region of the synaptonemal complex as the latter assembles during zygonema[23]. Similar movements are also seen for recombination proteins Mer3 and Msh4[68,69], but Mer2 re-localization to the axis is independent of Mer3 and Msh4, suggesting that Mer2 may have a role in transporting recombination complexes to the nascent synaptonemal complex. We propose that this transport takes advantage of the coherence provided by the DNA-dependent condensates.

## Supplementary References


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**Supplementary Table S1. (separate file)**

Excel file of crosslinking mass spectrometry data for Rec114–Mer2 complexes alone, for Mer2 alone, or for the mixture of all three proteins with DNA.

## Supplementary Table S2. Oligonucleotides used in this study

| Oligo | Sequence |
|---|---|
| cb095 | CTAGTATAGAGCCGGCGCGCCATGTCTAGATAGCGTTAGGTCTGCCGAATAGTACTACTCGGATCCCGAGCGAACCACGC |
| cb100 | GCGTGGTTCGCTCGGGATCCGAGTAGTACTATTCGGCAGACCTAACGCTATCTAGACATGGCGCGCCGGCTCTATACTAG |
| cb342 | TCGCGCGTTTCGGTGATGA |
| cb343 | TCGGAACAGGAGAGCGCACG |
| cb906 | CATCGGGCGCGGATCCCCATGGACCATGTACGAGTACTGCTC |
| cb907 | GTTTCAACCTTATTAACCCATTTTATGAATTC |
| cb908 | GAATTCATAAAATGGGTTAATAAGGTTGAAAC |
| cb909 | CCGCGACTAGTGAGCTCTCACTTTTCGAACATTTTATTG |
| cb910 | CATCGGGCGCGGATCCGAATTCACCATGAGTAGAGGCAAAC |
| cb911 | GAATTAAAGCAAAACAGATTATCCAGTCAAC |
| cb912 | GTTGACTGGATAATCTGTTTTGCTTTAATTC |
| cb913 | CTAGTGAGCTCGTCGACTTAAATGTTTTCTGTCTC |
| cb922 | CAACGTGGGCAAAGATGTCCTAGCAATGTAATCGTCTATG |
| cb935 | CATAGACGATTACATTGCTAGGACATCTTTGCCCACGTTG |
| cb939 | AAAGATGTCCTAGCAATGTA |
| cb940 | TACATTGCTAGGACATCTTT |
| cb978 | CATCGGGCGCGGATCCGAATTCACCATGGTCGCTAGAGGTAG |
| cb979 | AAGGTTTTCACGTTTCCGTTCGTAGCAGCATC |
| cb980 | GATGCTGCTACGAACGGAAACGTGAAAACCTT |
| cb981 | CCGCGACTAGTGAGCTCTCACAGCTCAGATTC |
| cb1161 | CACAGAGAACAGATTGGTGGATCCATGGTCGCTAGAGGTAG |
| cb1162 | CAGTGGTGGTGGTGGTGGTGCTCGAGTCACAGCTCAGATTCCAG |
| cb1175 | AAAGCTAGCGTACATTATCGCCAATACGC |
| cb1177 | GGATCCCCGGGTACCGAGCTCG |
| cb1186 | GATGGTCACAAGGTCCATGGCAGCCGCAGCATCCAGCTCCCCAACCCTATC |
| cb1187 | GATAGGGTTGGGGAGCTGGATGCTGCGGCTGCCATGGACCTTGTGACCATC |
| cb1223 | GATAGACAAGTGGCAAGCCCTTTCCGCTAACTGC |
| cb1224 | GCAGTTAGCGGAAAGGGCTTGCCACTTGTCTATC |
| cb1259 | AGAGAACAGATTGGTGGATCCATGGTGAGCAAGGGCG |
| cb1260 | ACCTCTAGCGACCATGGATCCCTTGTACAGCTCGTC |
| cb1279 | AGGACGATGATGACAAAGGTGGATCCATGGTGTCTAAAGGTG |
| cb1280 | CAGTACTCGTACATGGTCCATGGATCCAATCTAGACTTGTAC |
| cb1283 | GTACAGGTTTTCGGTCCATGGGGATCCACC |
| cb1284 | TTCCAATCCAATATGTACGAGTACTGCTCA |
| cb1285 | GTACAGGTTTTCGGATCCACCTTTGTCATC |
| cb1286 | TTCCAATCCAATATGGTGTCTAAAGGTGAAG |
| cb1287 | GTACAGGTTTTCGGTGAATTCCCTTCCCTC |

| | |
|---|---|
| cb1288 | TTCCAATCCAATATGAGTAGAGGCAAACTG |
| cb1330 | /5Cy3/TCGGAACAGGAGAGCGCACG |
| cb1331 | /5Cy5/TCGGAACAGGAGAGCGCACG |
| cb1332 | TATTGCTGCTGACGCATTCCGATTAAC |
| cb1334 | TCTGCGGCGCTAATCAAGGAAAAGTTG |
| cb1338 | CCCTGCCGCTTACCGG |
| cb1339 | AAAAATCGACGCTCAAGTCAG |
| cb1340 | CCATAAAATGGGTATGTATG |
| cb1341 | CTTCTTCGTCTTTCAAC |
| sp16 | CACCATCATCACCACAGCCAGGATCCGATGTACGAGTACTGCTC |
| sp17 | CCTGCAGGCGCGCCGAGCTCGAATTCACTTTTCGAACATTTTATTGAG |
| sp25 | GTATAAGAAGGAGATATACATATGAGTAGAGGCAAACTG |
| sp26 | CAGCGGTTTCTTTACCAGACTCGAGTTAAATGTTTTCTGTCTC |

## Supplementary Table S3. Plasmids used in this study.

| Plasmid | Description | Reference |
|---|---|---|
| pCCB649 | Rec114 in pFastbac1 | This study |
| pCCB650 | HisFlag-Rec114 in pFastbac-HTb | This study |
| pCCB651 | MBP-Rec114 in pFastbac1 | This study |
| pCCB652 | Mei4 in pFastbac1 | This study |
| pCCB653 | HisFlag-Mei4 in pFastbac1-HTb | This study |
| pCCB654 | MBP-Mei4 in pFastbac1 | This study |
| pCCB681 | Mer2 in pFastbac1 | This study |
| pCCB682 | HisFlag-Mer2 in pFastbac1-HTb | This study |
| pCCB683 | MBP-Mer2 in pFastbac1 | This study |
| pCCB750 | HisSUMO-Mer2 in pSMT3 | This study |
| pCCB777 | HisSUMO-eGFP-Mer2 in pSMT3 | This study |
| pCCB779 | HisSUMO-Mer2-(K265A/R266A/R267A/R268A) in pSMT3 | This study |
| pCCB783 | HisSUMO-eGFP-Mer2-(K265A/R266A/R267A/R268A) in pSMT3 | This study |
| pCCB786 | HisFlag-mScarlet-Rec114 in pFastbac1 | This study |
| pCCB789 | HisFlag-Tev-Rec114 in pFastbac1 | This study |
| pCCB790 | HisFlag-Tev-mScarlet-Rec114 in pFastbac1 | This study |
| pCCB791 | MBP-Tev-Mei4 in pFastbac1 | This study |
| pCCB825 | HisSUMO-Rec114(375-428)-Mei4(1-43) in pETDuet1 | This study |
| pCCB835 | HisFlag-Tev-Rec114-(H39A/L40A/S41A) in pFastBac1 | This study |
| pCCB836 | HisFlag-Tev-mScarlet-Rec114-(H39A/L40A/S41A) in pFastBac1 | This study |
| pCCB848 | HisFlag-Tev-Rec114-(R395A/K396AK399A/R400A) in pFastBac1 | This study |
| pCCB849 | HisFlag-Tev-mScarlet-(Rec114-R395A/K396AK399A/R400A) in pFastBac1 | This study |
| pCCB850 | Rec114-(R395A/K396AK399A/R400A) in pRS305 | This study |
| pCCB851 | Rec114-(R395A/K396AK399A/R400A)-myc8 in pRS305 | This study |
| pCCB856 | Rec114-(F411A) in pRS305 | This study |
| pCCB857 | Rec114-(F411A)-8myc in pRS305 | This study |
| pCCB858 | Rec114-(F411A) in pSK304 | This study |
| pJX005 | Mer2myc5-K265A/R266A/R267A/R268A in pSK351 (pRS306) | This study |
| pSK276 | Gal4AD empty Y2H vector | Arora *et al.*, 2004 |
| pSK281 | LexA-Mei4 Y2H vector | Arora *et al.*, 2004 |
| pSK282 | LexA-Rec102 Y2H vector | Arora *et al.*, 2004 |
| pSK283 | LexA-rec104 Y2H vector | Arora *et al.*, 2004 |
| pSK304 | Gal4AD-Rec114 Y2H vector | Arora *et al.*, 2004 |
| pSK351 | Mer2-5myc in pRS306 | Henderson *et al.*, 2006 |
| pSK591 | Rec114-8myc in pRS305 | Maleki *et al.*, 2007 |
| pSK592 | Rec114 in pSR305 | Maleki *et al*., 2007 |
| pSP1 | Rec114 del 1-50, del 152-277 in pSK304 | This study |

| | | |
|---|---|---|
| pSP3 | Rec114 del 101-277 in pSK304 | This study |
| pSP6 | Rec114 del 152-377 in pSK304 | This study |
| pSP9 | Rec114 del 152-277 in pSK304 | This study |
| pSP25 | Rec114-(H39A/L40A/S41A) in pSK304 | This study |
| pSP34 | Rec114-Mei4 in pEtDuet1 | This study |
| pSP53 | HisSumo-Rec114-Mei4 in pEtDuet1 | This study |
| pSP113 | Rec114-(H39A/L40A/S41A)-8myc in pRS305 | This study |

## Supplementary Table S4. Yeast strains used in this study.

| Strain | Genotype | Reference |
|---|---|---|
| SKY661 | *MATa, ho::LYS2, lys2, leu2::hisG, trp1::hisG, ndt80::KanMX4, LexA(op)-LacZ::URA3* | Arora *et al.*, 2004 |
| SKY662 | *MATα, ho::LYS2, lys2, leu2::hisG, trp1::hisG, ndt80::KanMX4, LexA(op)-LacZ::URA3* | Arora *et al.*, 2004 |
| SKY865 | *MATa, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4* | Maleki *et al.*, 2007 |
| SKY866 | *MATα, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4* | Maleki *et al.*, 2007 |
| SKY1524 | *MATa, ho::LYS2, lys2, ura3, leu2::hisG, mer2Δ::KanMX4* | Henderson *et al.*, 2006 |
| SKY1525 | *MATα, ho::LYS2, lys2, ura3, leu2::hisG, mer2Δ::KanMX4* | Henderson *et al.*, 2006 |
| SKY1560 | *MATa, ho::LYS2, lys2, ura3, leu2::hisG, mer2Δ::KanMX4, MER2-myc5::URA3* | Henderson *et al.*, 2006 |
| SKY1595 | *MATα, ho::LYS2, lys2, ura3, leu2::hisG, mer2Δ::KanMX4, MER2-myc5::URA3* | Henderson *et al.*, 2006 |
| SKY6411 | *MATa, ho::LYS2, lys2, ura3, leu2::hisG, mer2Δ::KanMX4, Mer2(K265A/R266A/R267A/R268A)-myc5::URA3* | This study |
| SKY6413 | *MATα, ho::LYS2, lys2, ura3, leu2::hisG, mer2Δ::KanMX4, Mer2(K265A/R266A/R267A/R268A)-myc5::URA3* | This study |
| SKY6562 | *MATa, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::REC114* | This study |
| SKY6563 | *MATα, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::REC114* | This study |
| SKY6749 | *MATa, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::REC114-8myc* | This study |
| SKY6750 | *MATα, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::REC114-8myc* | This study |
| SKY6797 | *MATa, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::Rec114(H39A/L40A/S41A)-8myc* | This study |
| SKY6798 | *MATα, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::Rec114(H39A/L40A/S41A)-8myc* | This study |
| SKY6859 | *MATa, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::Rec114(R395A/K396A/K399A/R400A)-8myc* | This study |
| SKY6860 | *MATα, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::Rec114(R395A/K396A/K399A/R400A)-8myc* | This study |
| SKY6885 | *MATa, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::Rec114(F411A)* | This study |
| SKY6886 | *MATα, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::Rec114(F411A)* | This study |
| SKY6889 | *MATa, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::Rec114(F411A)-8myc* | This study |
| SKY6890 | *MATα, ho::LYS2, ura3, lys2, leu2::hisG, rec114Δ::KanMX4, LEU2::Rec114(F411A)-8myc* | This study |