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High-resolution estimates of crossover and noncrossover recombination from a captive baboon colony

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Significance

Most homologous recombination events are noncrossovers (NCO), but little is known about NCO conversion tract lengths. Here we utilize whole-genome sequence data from large baboon pedigrees to estimate the NCO tract length distribution and to study other aspects of recombination.

27 **Abstract**

28

29 Homologous recombination has been extensively studied in humans and a handful of
30 model organisms. Much less is known about recombination in other species, including
31 non-human primates. Here we present a study of crossovers and non-crossover (NCO)
32 recombination in olive baboons (*Papio anubis*) from two pedigrees containing a total of
33 20 paternal and 17 maternal meioses, and compare these results to linkage-
34 disequilibrium (LD) based recombination estimates from 36 unrelated olive baboons.
35 We demonstrate how crossovers, combined with LD-based recombination estimates,
36 can be used to identify genome assembly errors. We also quantify sex-specific
37 differences in recombination rates, including elevated male crossover and reduced
38 female crossover rates near telomeres. Finally, we add to the increasing body of
39 evidence suggesting that while most NCO recombination tracts in mammals are short
40 (e.g., < 500 bp), there are a non-negligible fraction of longer (e.g., > 1 Kb) NCO tracts.
41 We fit a mixture-of-two-geometric distributions model to the NCO tract length distribution
42 and estimate that >99% of all NCO tracts are very short (mean 24 bp), but the
43 remaining tracts can be quite long (mean 11 Kb). A single geometric distribution model
44 for NCO tract lengths is incompatible with the data, suggesting that LD-based methods
45 for estimating NCO recombination rates that make this assumption may need to be
46 modified.

47

48 **Introduction**

49

50 Homologous recombination is a fundamental biological process, thought to be
51 necessary for the proper segregation of chromosomes during meiosis and essential for
52 the efficacy of natural selection. Recombination rates in higher eukaryotes are
53 generally measured using (1) genetic comparisons between parents and offspring (e.g.,
54 using genotype or sequence data), (2) genotyping or sequencing of single or pooled
55 sperm (i.e., potential gametes), or (3) indirect estimation via statistical methods that
56 quantify the relationship between linkage disequilibrium and recombination. Each of
57 these three approaches involve tradeoffs regarding cost/effort and the breadth and

58 depth of information they can provide. In particular, only pedigree-based studies
59 provide both sex-specific recombination estimates and direct identification of both
60 crossover (CO) and non-crossover (NCO) recombination events, but they are more
61 difficult to conduct due to sample acquisition challenges.

62 Recombination is thought to arise from double strand breaks (DSB) that occur
63 after the pairing of homologous chromosomes during meiosis. Depending on how these
64 breaks are resolved, the result can either be CO recombination, which involves the
65 reciprocal transfer of large chromosomal regions between homologs, and NCO
66 recombination (colloquially called 'gene conversion'), involving the non-reciprocal
67 replacement of short tracts of DNA from one homolog to another (Orr-Weaver et al.
68 1981; Szostak et al. 1983). Since crossovers are also associated with gene conversion
69 tracts at the DSB location, we will use the term NCO recombination to describe
70 homologous gene conversion not associated with a nearby crossover.

71 Theory predicts a close relationship between recombination and patterns of linkage
72 disequilibrium (LD), since homologous recombination will tend to shuffle haplotypes and
73 break down allelic associations. Population genetic analyses of dense genotype and
74 sequence data, along with sperm typing studies, have shown that most human
75 crossovers happen in narrow (1-2 Kbp) 'hotspots' (e.g., Chakravarti et al. 1984; Jeffreys
76 et al. 2001; Crawford et al. 2004; Myers et al. 2005), and that this fine-scale structuring
77 of recombination rates can help explain the block-like structure of LD in many parts of
78 the genome (Wall and Pritchard 2003). In most vertebrates, the locations of these
79 hotspots are mediated by the zinc finger *PRDM9* (reviewed in Paigen and Petkov 2018),
80 and recombination hotspot locations are generally not shared across closely related
81 species (e.g., Ptak et al. 2005; Auton et al. 2012; Stevison et al. 2016).

82 Much less is known about NCO recombination. A handful of studies in humans
83 and model organisms have found that most recombination events are NCOs, but mean
84 tract lengths are quite short – tens or hundreds of base pairs (e.g., Jeffreys and May
85 2004; Baudat and de Massy 2007; Cole et al 2010; Comeron et al. 2012; Wijnker et al.
86 2013; Li et al. 2019). This short tract length makes NCO recombination especially
87 difficult to study. In particular, for species with low levels of heterozygosity (e.g., most
88 mammals) many NCO tracts are undetectable because the donor and converted

89 sequences are identical. In most of the remainder only a single heterozygous site is
90 converted, making NCO recombination difficult to distinguish from simple
91 genotype/sequencing errors. While statistical methods have been developed for
92 estimating NCO recombination parameters indirectly from segregating patterns of
93 genetic variation (e.g., Frisse et al. 2001; Gay et al. 2007; Yin et al. 2009;
94 Padhukasahasram and Rannala 2013), these methods are not very accurate primarily
95 because of the small/negligible effect that most NCO tracts have on patterns of genetic
96 variation. In addition, these methods generally assume that NCO tract lengths follow a
97 geometric distribution, which may not be biologically realistic. Because of this, studies
98 of NCO recombination have generally focused on identifying events by comparing the
99 patterns of genetic inheritance of offspring (or potential offspring in the case of sperm
100 typing) from their parents (e.g., Jeffreys and May 2004; Comeron et al. 2012; Wijnker et
101 al. 2013; Williams et al. 2015; Halldorsson et al. 2016; Li et al. 2019).

102 Among mammals, NCO recombination has been most-studied in humans, with
103 several sperm typing studies (Jeffreys and May 2004; Jeffreys and Neumann 2005;
104 Webb et al. 2008; Odenthal-Hesse et al. 2014), two large pedigree-based studies
105 (Williams et al. 2015; Halldorsson et al. 2016), and a study of genetic variation in
106 autozygous tracts of consanguineous individuals (Narasimhan et al. 2017). Two
107 observations from these studies stand out. First, both pedigree-based studies found
108 evidence for complex NCO events, involving multiple non-contiguous gene conversion
109 tracts that are physically near each other, from the same meiosis, and not associated
110 with a nearby CO (Williams et al. 2015; Halldorsson et al. 2016). Second, both studies
111 also found evidence for apparent long (i.e., > 20 Kbp), contiguous NCO tracts. If real,
112 these long tracts are suggestive of a separate molecular mechanism distinct from the
113 gene conversion expected under the standard DSB model. It is possible though that
114 they reflect a rare, CO interference independent recombination process, or that they are
115 actually complex NCO events with smaller tract sizes that are miscalled due to low
116 marker density.

117 In this study, we examine patterns of recombination with a focus on NCO tracts
118 using olive baboons (*Papio anubis*) in the baboon colony housed at the Southwest
119 National Primate Research Center (SNPRC). We generate and analyze high-coverage

120 whole-genome sequence data from two pedigrees with large sib-ships (Figure 1), which
121 allows us to estimate sex-specific recombination rates, identify NCO recombination
122 events, and evaluate the long-range accuracy of the current Panubis1.0 genome
123 assembly (cf. Batra et al. 2020). This assembly used Hi-C contact data to join contigs
124 into scaffolds, and the low-resolution linkage map we generate here allows us to assess
125 the accuracy of this approach.

126 Our choice of baboons was motivated in part by the availability of an extremely
127 large pedigreed colony at the SNPRC, as well as the higher levels of diversity found in
128 baboons relative to humans (e.g., Robinson et al. 2019). Our expectation is that the
129 increased marker density will provide greater resolution on the size distribution of NCO
130 tracts, and that our study of a nonhuman primate will help elucidate whether some of
131 the specific recombination patterns observed in humans can be generalized to a wider
132 group of species.

133

134 **Results**

135

136 ***Baboon genetic map***

137

138 We identified crossovers and NCO recombination events from a total of 20 paternal and
139 17 maternal meioses (Supplementary Table S1). In total, we identified 842 autosomal
140 crossovers with a median resolution (i.e., the size of the region over which the
141 crossover location could be placed) of 7.7 Kb. This corresponds to a sex-averaged
142 autosomal genetic map length of 2,293 cM (2,080 cM in males, 2,506 cM in females).
143 Our estimate was 16% larger than a previous estimate based on microsatellite data
144 (Rogers et al. 2000), which reflects both the longer and more complete baboon genome
145 assembly that we used and the much greater marker density of our study. Overall, our
146 results are consistent with the growing body of evidence suggesting that old world
147 monkeys have shorter genetic map lengths, as measured by direct identification of
148 crossovers in pedigrees, than do humans and great apes (e.g., Broman et al. 1998;
149 Rogers et al. 2000, 2006; Kong et al. 2002; Jasinska et al. 2007; Venn et al. 2014).

150 We also estimated local recombination rates from patterns of LD in 36 unrelated
151 olive baboons using pyrho (Spence and Song 2019). We found that rate estimates are
152 significantly higher within distal regions (≤ 10 Mb from chromosome ends) relative to
153 proximal regions (> 10 Mb from chromosome ends) (two-sided Mann-Whitney U test, $p <$
154 $2.2 * 10^{-16}$, Supplementary Figure 1).

155

156 ***Identifying potential genome assembly errors***

157

158 In performing quality control for our genetic map, we identified several abnormal
159 apparent crossover patterns that likely reflect errors in the Panubis1.0 genome
160 assembly (Figure 2). These included a total of 16 potential inversions, 3 misplaced
161 contigs and 1 potential translocation (Supplementary Table S2). We then used LD-
162 based estimates of recombination using pyrho (Spence and Song 2019) to examine
163 whether patterns of LD provided any additional support. On average, estimated
164 recombination rates at putative synteny breaks are roughly 20 times higher than the
165 estimated rates in the flanking sequences (Figure 3A), consistent with the decrease in
166 LD expected across genome assembly error breakpoints. For 6 proposed inversions
167 and the translocation (Supplementary Table S2), pyrho estimates provide corroborating
168 evidence in finding low levels of estimated recombination (i.e., evidence for synteny)
169 across the 'corrected' breakpoints (Figure 3B).

170

171 ***NCO recombination***

172

173 After stringent filtering, we identified a total of 325 sites contained in 263 tracts
174 (Supplementary Table S3) that were inferred to be converted due to NCO
175 recombination in tracts < 10 Kb in length. Of the 39 events involving the conversion of
176 more than one heterozygote, the minimal length of the inferred NCO tract was generally
177 small (median = 42 bp), but had a long tail of occasionally longer tracts (mean = 167 bp,
178 including 10 tracts longer than 1 Kbp).

179 Overall, we estimated a sex-averaged NCO rate of $7.52 * 10^{-6}$ per site per
180 generation (paternal NCO rate = $5.34 * 10^{-6}$ and maternal NCO rate = $9.71 * 10^{-6}$). As

181 with previous human studies (Williams et al. 2015; Halldorsson et al. 2016), we found a
182 handful of more complex NCO recombination events, including 7 regions containing
183 multiple non-contiguous NCO tracts and 9 NCO regions associated with a nearby
184 crossover (Supplementary Table S3; note that 3 regions involve non-contiguous NCO
185 tracts that are also associated with a nearby crossover). In addition, we identified 10
186 regions consistent with a potential NCO tract of length 10 – 100 Kb (Table 1). Of these,
187 6 were identified as potential inversion errors in the underlying genome assembly, and
188 three others overlapped with non-inversion potential genome assembly errors
189 (Supplementary Table S2). If we include the remaining long NCO tract into the rate
190 calculation, the estimated sex-averaged NCO rate increases to $8.01 * 10^{-6}$ per site per
191 generation (paternal NCO rate = $5.34 * 10^{-6}$ and maternal NCO rate = $1.07 * 10^{-5}$).
192 These estimates are roughly comparable to NCO rate estimates in humans (e.g.,
193 Williams et al. 2015; Halldorsson et al. 2016; Narasimhan et al. 2017).

194

195 ***GC bias of NCO tracts***

196

197 GC-biased gene conversion (gBGC) is a selectively neutral process whereby gene
198 conversion events containing an AT/GC heterozygote in the parent are preferentially
199 resolved to contain the G or C allele in the gamete (Galtier and Duret 2007; Duret and
200 Galtier 2009). Both sperm typing studies (Odenthal-Hesse et al. 2014) and pedigree-
201 based studies (Williams et al. 2015; Halldorsson et al. 2016) in humans have quantified
202 the strength of gBGC in humans. Of the 224 NCO tracts that were informative on gBGC
203 in our study, 129 of them (57.6%) show a transmission bias toward G or C alleles.
204 While this proportion is significantly more than 50% ($p = 0.014$, one-tailed binomial test),
205 it is also significantly less than ($p = 6.8 * 10^{-4}$, one-tailed binomial test) the 68% GC bias
206 estimated from human pedigree studies (Williams et al. 2015; Halldorsson et al. 2016).

207

208 ***Age vs. recombination rate***

209

210 Previous human recombination studies have documented increases in both CO rate
211 (Kong et al. 2004; Martin et al. 2015) and NCO rate (Halldorsson et al. 2016) with

212 increasing maternal age. While we are underpowered to detect any true correlations
213 between recombination rate and parental age, we did find a marginally significant
214 association between NCO rate and paternal age ($p = .036$; raw data in Supplementary
215 Table S3). All other comparisons of CO or NCO rate with paternal or maternal age
216 were not significant ($p > 0.1$).

217

218 ***Regional variation in recombination rates***

219

220 We tabulated the relative numbers of CO and NCO recombination events as a function
221 of distance from telomeres, and stratified the results by sex. We then compared these
222 with sex-averaged recombination rate estimates based on patterns of linkage
223 disequilibrium (Figure 4). As with humans, we find that the male / female CO ratio is
224 higher in distal regions and lower in proximal regions further from the chromosome
225 ends. Near baboon telomeres, males have significantly higher CO rates *and* females
226 have significantly lower CO rates (Figure 4A). We observe a significantly higher male
227 NCO rate near the ends of chromosomes as well (Figure 4B), but did not observe any
228 correlation between female NCO rate and chromosome ends or between CO rate and
229 NCO rate. Consistent with a (partial) decoupling of local CO and NCO rates, we find
230 that rho recombination rate estimates are higher near inferred CO locations (Figure
231 5A) than near inferred NCO locations (Figure 5B).

232

233 ***NCO tract length distribution***

234

235 We used a maximum-likelihood approach for estimating the NCO tract length
236 distribution from the observed patterns of converted NCO sites. We first assumed a
237 geometric tract length distribution, similar to previous studies (e.g., Hilliker et al. 1994;
238 Gay et al. 2007; Miller et al. 2012; Li et al. 2019). If we confine our analyses to NCO
239 tracts less than 10 Kbp in length, we estimate a mean tract length of 309 bp (95% CI =
240 290 – 341 bp). However, we found that our estimate was roughly proportional to the
241 minimum length of the longest NCO tract in our data set. For example, if we only
242 consider NCO tracts < 5 Kbp in length the estimate is 182 bp (95% CI = 173 – 201 bp),

243 or for tracts < 1 Kbp the mean length estimate is 58 bp (95% CI = 48 – 71 bp). This is in
244 part because the geometric distribution does not fit the data well. In particular, while
245 most NCO sites are consistent with a short (i.e., < 100 bp) tract length, there is a tail of
246 longer NCO tracts that must be kilobases long (Figure 6A).

247 We next considered a more general scenario where NCO tract lengths are
248 modeled as a mixture of two geometric distributions. This would be appropriate if NCO
249 recombination could occur through two separate molecular pathways, each of which
250 produced tracts whose length followed geometric distributions. Our maximum likelihood
251 estimate had 99.8% of NCO tracts following a distribution with a mean length of 24 bp
252 (95% CI = 18 – 31 bp), while the remaining NCO tracts had a mean length of 11 Kbp
253 (95% CI = 3 – 100+ Kbp). Under this best-fit model, just 1.6% of NCO tracts are longer
254 than 100 bp, but these longer tracts account for 31.6% of all sites that are converted by
255 NCO recombination (see blue and orange curves in Figure 6B respectively).

256

257 **Discussion**

258

259 Pedigree-based studies of recombination, while common in previous decades due to
260 technological and computational limitations, have been mostly superseded now by
261 studies that indirectly estimate recombination rates from patterns of linkage
262 disequilibrium (e.g., Apuli et al. 2020; Beeson et al. 2019; Dreissig et al. 2019; Jones et
263 al. 2019; Pfeifer 2020; Robinson et al. 2019; Schield et al 2020; Schwarzkopf et al.
264 2020; Shanfelter et al. 2019; Spence and Song 2019; Xue et al. 2020). We argue
265 though that despite the substantial amount of time and effort required to conduct
266 pedigree-based studies, they can provide invaluable information that is inaccessible by
267 other methods. LD-based recombination estimates, by their nature, are averages
268 across time and individuals, are influenced by any evolutionary force that affects
269 patterns of genetic variation (e.g., changes in population size, migration, admixture,
270 natural selection, etc.), and require assumptions about the effective population size to
271 be converted into an actual per generation rate. They cannot provide any information
272 on sex-specific differences in CO rates, nor are they very informative about NCO
273 recombination. For example, while baboon pyrro estimates are, on average, slightly

274 elevated in sub-telomeric regions, this obscures the observation that male crossovers
275 are 10-15 times more prevalent than female crossovers in the distal 5 Mbp of each
276 chromosome arm. Human pedigree studies show the same general pattern (Broman et
277 al. 1998; Kong et al. 2002), but the sex-bias is much larger in baboons than in humans.

278 One ancillary benefit of our pedigree-based examination of recombination in
279 baboons is that it helped provide some independent information on the quality of the
280 existing Panubis1.0 genome assembly. Panubis1.0 utilized a combination of Illumina
281 short-read, Oxford Nanopore long-read, 10x Genomics linked-read, Bionano optical
282 map and Hi-C sequence data to create an assembly with N50 contig size of 1.46 Mbp
283 and single scaffolds that span each of the autosomes (Batra et al. 2020). The Hi-C data
284 in particular enabled Panubis1.0 to be a truly *de novo* genome assembly, unlike the
285 previous reference-guided baboon assembly (Rogers et al. 2019). However, there is
286 some concern that Hi-C based scaffolding is susceptible to incorrect orientation of
287 contigs, leading to inversion errors in the resulting assembly (e.g., Burton et al. 2013).
288 Here, traditional linkage analyses enabled us to identify more than 20 likely assembly
289 errors, most of which were putative inversions (Supplementary Table S3). This
290 suggests that caution should be taken in accepting Hi-C based scaffolding without the
291 presence of orthogonal sources of corroborating data.

292 Evidence for an inversion assembly error in linkage data comes from the presence
293 of three closely spaced crossovers in one or more individuals (Broman et al. 1998,
294 2003; Figure 2). Six of these cases are also consistent with a single crossover
295 associated with a long (24 – 86 Kbp), nearby NCO tract. While the data that we have
296 cannot rule out either of these explanations, the relative dearth of putative long NCO
297 tracts that are not associated with potential genome assembly errors strongly suggests
298 that most (if not all) of the apparent long baboon NCO tracts are artefacts and not real.
299 Similarly, we postulate that at least some of the long human NCO tracts identified in
300 previous studies (Williams et al. 2015; Halldorsson et al. 2016) are actually due to
301 microassembly errors or polymorphic structural variants. It is also likely that some of
302 them represent complex NCO events (with multiple smaller conversion tracts) that are
303 misclassified due to low marker density.

304 Finally, we note that even after removing all apparent long (e.g., > 10 Kbp) NCO

305 tracts, our data show that a simple geometric model of NCO tract lengths is
306 inappropriate, at least for baboons. While the mixture-of-two-geometric-distributions
307 model we considered is somewhat arbitrary, it captures the qualitative observation that
308 most baboon NCO tracts are quite short, but a small minority can be much longer. The
309 extent to which our findings reflect general patterns of NCO recombination is unclear at
310 this time. To date, only two other mammalian species have been studied in depth. Our
311 results are qualitatively similar to the findings of NCO studies in humans (Williams et al.
312 2015; Halldorsson et al. 2016), but long NCO tracts seem to be much rarer in studies of
313 hybrid mice (Li et al. 2019; Gergelits et al. 2021). High-resolution studies in additional
314 species will be needed to better understand how empirical patterns of recombination
315 vary across species, and whether additional molecular models (e.g., a crossover
316 pathway without interference) may be necessary to explain the inferred patterns of
317 recombination in large pedigrees.

318

319 **Materials and Methods**

320

321 ***Samples, sequencing and variant calling***

322

323 All samples for this study are putative olive baboons (*Papio anubis*) from the pedigreed
324 baboon colony housed at the Southwest National Primate Research Center (SNPRC).
325 We extracted DNA from blood or tissue samples and sent them to MedGenome, Inc. for
326 sequencing (using standard protocols and libraries) on Illumina HiSeq 4000 and X
327 machines. We generated novel whole-genome sequence data from 23 individuals,
328 generated additional sequence data from several previously published baboon
329 genomes, and combined these with data from previous studies (Robinson et al. 2019;
330 Wu et al. 2020) to obtain a final data set that included 66 baboons with a median of
331 35.6X depth of coverage. These samples are included in two large pedigrees (Figure 1)
332 as well as in a panel of 36 unrelated olive baboons. SRA accession numbers for all
333 sequences used in this study are presented in Supplementary Table S4 and archived in
334 NCBI BioProject PRJNA433868.

335 For each sample, we mapped all sequenced reads to the Panubis1.0 genome
336 assembly (Batra et al. 2020) using BWA MEM (Li and Durbin 2009) before marking
337 duplicate reads with Picard (<https://broadinstitute.github.io/picard>) and then genotyping
338 with HaplotypeCaller from the Genome Analysis Toolkit (GATK; McKenna et al., 2010).
339 We then produced a joint genotype call set with GATK GenotypeGVCFs before applying
340 filters. Specifically, we excluded sites in soft-masked regions of the genome, which
341 correspond to repetitive and low complexity regions identified with WindowMasker
342 (Morgulis et al., 2006), plus variants that were not bi-allelic SNPs. We also excluded
343 genotypes with genotype quality (GQ) score less than 30, and sites with excess
344 heterozygosity (defined as sites where the number of heterozygotes more than 3.5
345 standard deviations above random mating expectations). For the last criterion, we are
346 aware that the 66 samples are not all unrelated, but since relatedness (and population
347 structure) generally leads to less heterozygosity than random mating expectations, our
348 approach is conservative.

349

350 ***Pedigree-based identification of crossover events***

351

352 We utilized the single nucleotide variants in the call set described above to identify
353 recombination events from the meioses involving 10173, 12242, 9841, 1X2816, and
354 their offspring (20 paternal meioses and 17 maternal meioses in total). Note that each
355 sub-pedigree had a minimum of 5 offspring.

356 For a target meiosis, we first filtered the data to include only 'informative' sites
357 where the parentally transmitted allele could be directly inferred. For example, when
358 trying to identify paternal recombination events in 16517, we require the sire's (10173)
359 genotype to be heterozygous and the dam's (12242) genotype to be homozygous. That
360 way, the maternally transmitted allele is known and the paternally transmitted allele
361 must be the other allele in 16517's genotype. While the parental genome is unphased,
362 it is straightforward to infer haplotypic phase by examining the patterns of alleles
363 transmitted by the parent, and to identify potential recombination events by switches in
364 which haplotype is inherited in each of the offspring (Coop et al. 2008). We employed

365 additional filters by requiring genotype calls in all of a pedigree's offspring, and by
366 removing the 5 informative sites nearest to the ends of each chromosome.

367 We started by identifying all switches in transmitted haplotype that could be
368 parsimoniously explained by a single crossover (Supplementary Table S1). We then
369 manually examined all intervals (i.e., regions between consecutive informative markers)
370 where we inferred the occurrence of two crossovers. Four of these intervals were long
371 (e.g., > 60 Kb), not near chromosome ends (i.e., at least 2 Mb away), and involved a
372 sub-pedigree with at least 7 offspring (i.e., with parent 10173, 1X2816 or 1X4519). For
373 these, the evidence is quite strong that there were in fact two crossovers (rather than a
374 genome assembly error or >2 crossovers). An additional eight intervals are shorter (2 –
375 30 Kb) and/or involve smaller sub-pedigrees with only 5 offspring. We have labelled
376 these as 'provisional' crossovers, listed them in the "Provisional_COs" sheet in
377 Supplementary Table S1, and included them for estimating the total genetic map length.

378

379 ***Pedigree-based identification of genome assembly errors***

380

381 We identified several unusual patterns in our crossover data that are suggestive of
382 either genome assembly errors or polymorphic chromosomal rearrangements (Figure
383 2). For example, three closely spaced crossovers in a single meiosis are extremely
384 unlikely due to crossover interference (Muller 1916), but could easily arise through the
385 combination of a single crossover plus an inversion (Broman et al. 1998, 2003). We
386 hypothesized that potential breakpoints are likely to occur in-between assembled
387 contigs, and classified these synteny breaks as putative inversions (three crossovers
388 within 10 Mb in the same meiosis), misplaced contigs (two closely linked crossovers
389 occurring at the same locations in multiple individuals), translocations (misplaced
390 contigs where the correct genomic location could be inferred), or single breaks of
391 synteny (Supplementary Table S2). The genomic location of contig breaks within
392 hypothesized breakpoints are shown in parentheses in the second and third columns of
393 Table S2.

394

395 ***LD-based recombination estimates***

396

397 We used pyrho (Spence and Song 2019) to estimate local recombination rates from
398 patterns of linkage disequilibrium in a panel of 36 unrelated olive baboon founders.
399 Recombination rate inference with pyrho requires a demographic model, and for this we
400 used SMC++ (Terhorst et al., 2017). For this analysis, we included genotypes with a
401 minimum quality score of 40 and read depth ≥ 8 , and applied a series of filters based on
402 recommendations from GATK to minimize the inclusion of errors (QUAL < 30.0, QD <
403 2.0, FS > 60.0, MQ < 40.0, MQRankSum < -12.5, ReadPosRankSum < -8.0, SOR > 3.0,
404 ExcHet < 0.05), leaving 14.4 million variants in total. To run SMC++, we used a random
405 set of ten individuals as the “distinguished” lineages, and set the polarization parameter
406 (-p) to 0.5 to handle uncertainty in the polarization of derived versus ancestral alleles.
407 We then followed the developer’s instructions to incorporate the demographic model
408 from SMC++ into the recombination rate inference with pyrho. We also incorporated two
409 additional filters before running pyrho; we excluded singletons, which are uninformative
410 for LD-based recombination rate inference, and we thinned variants so that no two
411 SNPs were closer than 10 bp, leaving 10.4 million variants in total. To handle the large
412 number of haplotypes in our dataset (n=72), we enabled the Moran approximation with:
413 --approx --moran_pop_size 98. After exploring a number of block penalty (smoothing)
414 and window size parameters with hyperparam, we found that a block penalty of 10 and
415 window size of 75 were optimal. After manual inspection of the output, we removed the
416 pyrho value for a single interval on chromosome 19 (approximate positions 24.92 –
417 25.02 Mb) where the estimated genetic map length was ~102 cM. There is no evidence
418 for any crossovers near this region, so we deemed the extremely large estimate to be
419 unreliable.

420 pyrho and other LD-based methods most naturally estimate the population scaled
421 recombination parameter ρ ($= 4Nr$, where N is the effective population size and r is the
422 recombination rate per generation). So, conversion of these values to actual per
423 generation recombination rate estimates requires assumptions about other fundamental
424 parameters such as N and/or the mutation rate. With the assumptions described above,
425 the pyrho estimated total genetic map length was several times shorter than the sex-
426 averaged crossover-based genetic map length. To make these results more compatible

427 with each other, we rescaled the pyrho values to have the same total autosomal map
428 length (2,293 cM) as estimated from crossovers by multiplying all pyrho-based
429 estimates by the constant 5.577. This preserves local patterns of recombination rate
430 variability while acknowledging the large uncertainties in estimating past population
431 sizes.

432

433 ***Identifying non-crossover (NCO) recombination***

434

435 NCO recombination can be identified from pedigree data in an analogous way as
436 crossover identification. Specifically, single NCO tracts show up as two very tightly
437 linked crossovers in a single individual, or equivalently as one or more closely linked
438 sites where an offspring inherits one parental haplotype, surrounded on both sides by
439 much larger regions where the offspring inherits the other parental haplotype. Finally,
440 we arbitrarily fixed the maximum NCO tract length size as 10 Kb, and analyzed
441 apparent larger NCO tracts separately (see Results).

442 Pedigree 1 contained three offspring (32043, 32849 and 33863) of the 2nd
443 generation individuals used for estimating NCO rates (Figure 1). For all sites contained
444 in putative NCO tracts involving the parents of these offspring (19181 and 19348), we
445 checked for Mendelian inconsistencies across the whole pedigree as a limited way to
446 test whether putative NCO tracts might be caused by sequencing/genotyping errors in
447 the offspring. (We did not find any.) To reduce the effect of potential genotyping errors
448 in the parent, we excluded sites with more than two segregating alleles, as well as all
449 apparent NCO tracts that are shared across multiple half or full siblings.

450

451 ***NCO tract length distribution***

452

453 We use an approximate maximum-likelihood approach to estimating the probability of
454 the data as a function of NCO tract length distribution parameters. Here the data
455 consist of the pattern of which informative sites are converted (or not converted) for
456 each autosome of each meiosis. We assume that both the probability of NCO
457 recombination and the NCO tract length distribution do not change across base pairs or

458 meioses, and fix the former at $7.52 * 10^{-6}$ per base pair per generation, as estimated
459 below. Without loss of generality, we assume that NCO tracts are initiated at a certain
460 base pair, and then continue along the chromosome 5' to 3' until they end.

461 Suppose $D(\Lambda)$ is a specific NCO tract length distribution governed by parameter(s)
462 Λ . If $m(\Lambda)$ is the mean tract length given Λ , then the per base pair probability of initiation
463 of an NCO tract of length k is

$$464 \quad f_k(\Lambda) = \frac{7.52 * 10^{-6} \Pr(k|\Lambda)}{m(\Lambda)}$$

466 per meiosis. For a specific NCO tract, define $\{o_k\}$ as the number of k -mers (i.e., k
467 consecutive base pairs) that overlap all of the informative sites converted in the tract
468 and no others. Then the probability of observing an NCO tract is $\sum_k f_k(\Lambda) o_k$. Similarly,
469 define $\{e_k\}$ as the number of k -mers (across a chromosome) that must be excluded
470 because they overlap with a non-converted site. The probability that none of the
471 informative sites that shouldn't be converted are actually converted is $\prod_k (1 - f_k(\Lambda))^{e_k} \approx$
472 $e^{-\sum_k f_k(\Lambda) e_k}$. Finally, the likelihood of the full data is then the product of the separate
473 likelihoods of observing each of the observed NCO tracts multiplied by the products of
474 all of the probabilities of not converting the non-converted sites, across all meioses and
475 autosomes.

476 Our analyses of actual data treated each contiguous NCO tract as separate, even
477 if it was part of a complex NCO event, and arbitrarily required tract lengths to be 10 Kbp
478 or shorter. This led to a total of 263 NCO tracts that we had information for. We first
479 considered a geometric distribution for NCO tract lengths, requiring the mean length to
480 be an integer. Confidence intervals were obtained using the standard asymptotic
481 maximum likelihood assumptions. Next, we considered a mixture of two geometric
482 distributions, parameterized by $\Lambda = (\alpha, m_1, m_2)$, where m_1 and m_2 are the means of the
483 two distributions, and α is the proportion of tracts that have mean m_1 . We then
484 calculated the likelihood of the data over a grid of parameter values, where α varied
485 from 0 to 1 in increments of 0.001 and m_1 and m_2 varied from 10 – 100000 for all
486 integers with 2 significant digits in this range. After obtaining the maximum likelihood
487 estimate, we created profile likelihood curves for m_1 and m_2 to estimate approximate
488

489 confidence intervals.

490

491 **Data Availability**

492

493 All sequence data used in this study have been deposited in the Sequence Read
494 Archive under NCBI BioProject PRJNA433868 with accession numbers given in
495 Supplementary Table S4. The vcf file and pyrho genetic map will be made publicly
496 available prior to publication.

497

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499

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502

503 **Figure Legends**

504

505 **Figure 1.** Schematics of the two baboon pedigrees used in this study.

506

507 **Figure 2.** Detecting assembly errors from abnormal crossover patterns. (A) Type of
508 assembly error, (B) Pattern of inferred crossovers in offspring, and (C) Description of
509 pattern.

510

511 **Figure 3.** Estimates of ρ from patterns of LD at (A) Breaks of synteny in the current
512 baboon assembly identified from abnormal crossover patterns, and (B) Regions where
513 the Panubis1.0 assembly has been “corrected”

514

515 **Figure 4.** Recombination rates as a function of distance from telomeres. Comparison
516 of LD-based recombination estimates (grey and black) with paternal (blue) and maternal
517 (red) (A) crossover counts, and (B) NCO recombination counts.

518

519 **Figure 5.** Elevation of ρ recombination rate estimates near the sites of (A)
520 crossover and (B) NCO recombination events.

521

522 **Figure 6.** Distribution of NCO tract lengths, for actual data and best-fit model. (A)
523 Minimum inferred lengths of observed NCO tracts shorter than 10 Kbp. (B) Distribution
524 of NCO tract lengths for the best-fit mixture of two geometric distributions model (in
525 blue), and weighted by tract length (in orange).

526

527

528 **Table 1.** List of apparent NCO tracts longer than 10 Kbp

529

530

Chrom.	Parent	Offspring	Minimum tract length	# of NCO sites	Overlap with inversion or BOS?
3	9841	19348	40934	22	No
4	9841	15444	24809	39	Yes
6	10173	18385	55304	62	Yes
7	10173	15444	39253	2	Yes
7	12242	26988	45787	22	Yes
8	12242	28246	61766	9	Yes
11	1X2816	10489	24481	4	Yes
13	1X2816	8307	84614	54	Yes
13	10173	16517	86445	48	Yes
16	12242	17903	12403	19	Yes

531

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Figure 1

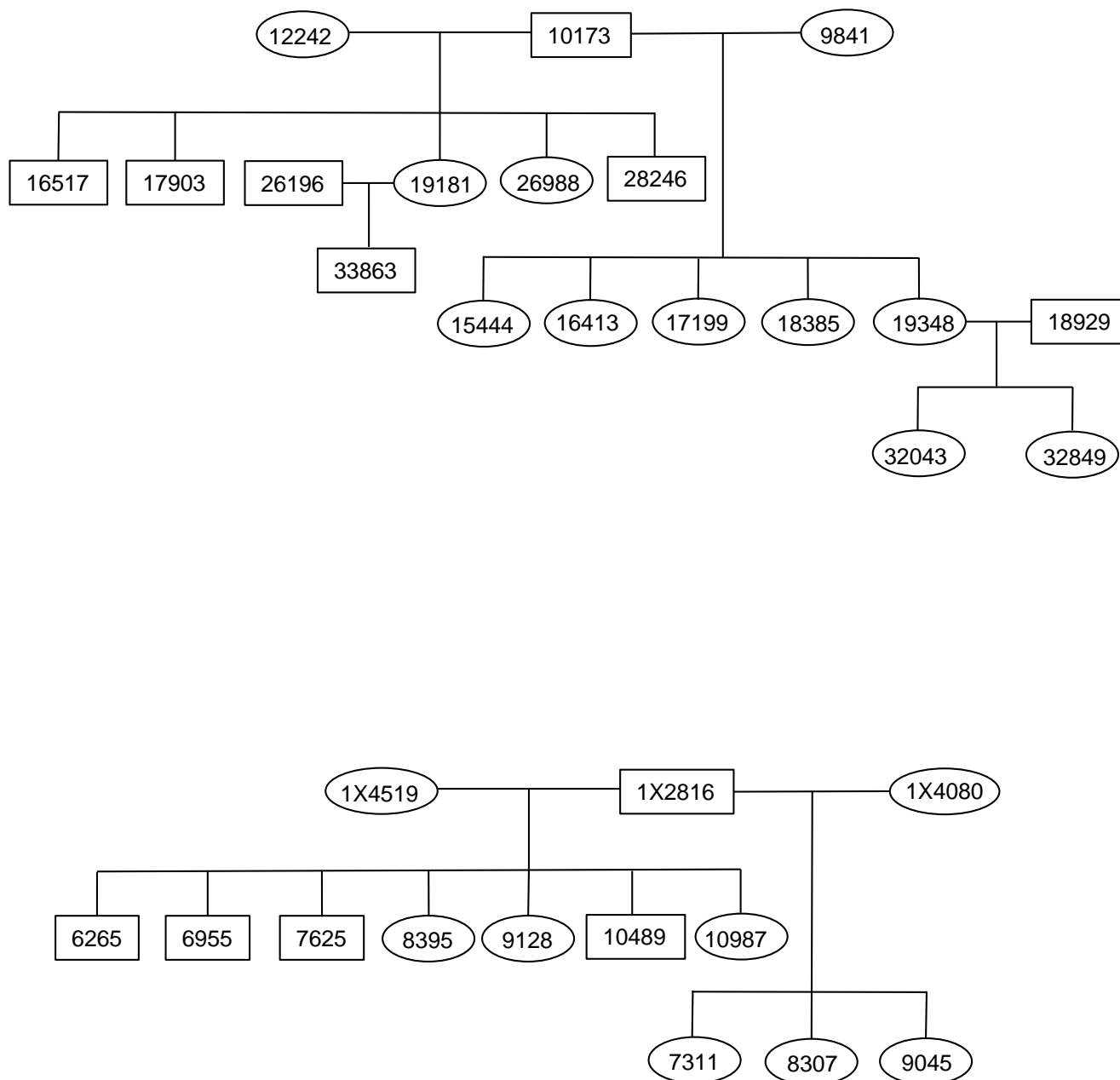


Figure 2

A

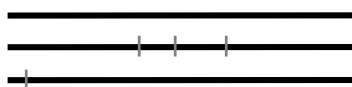
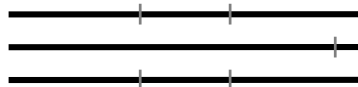
Misplaced contig



Inversion



B



C

Closely spaced crossover pairs in multiple individuals

Three closely spaced crossovers in at least one individual

Figure 3

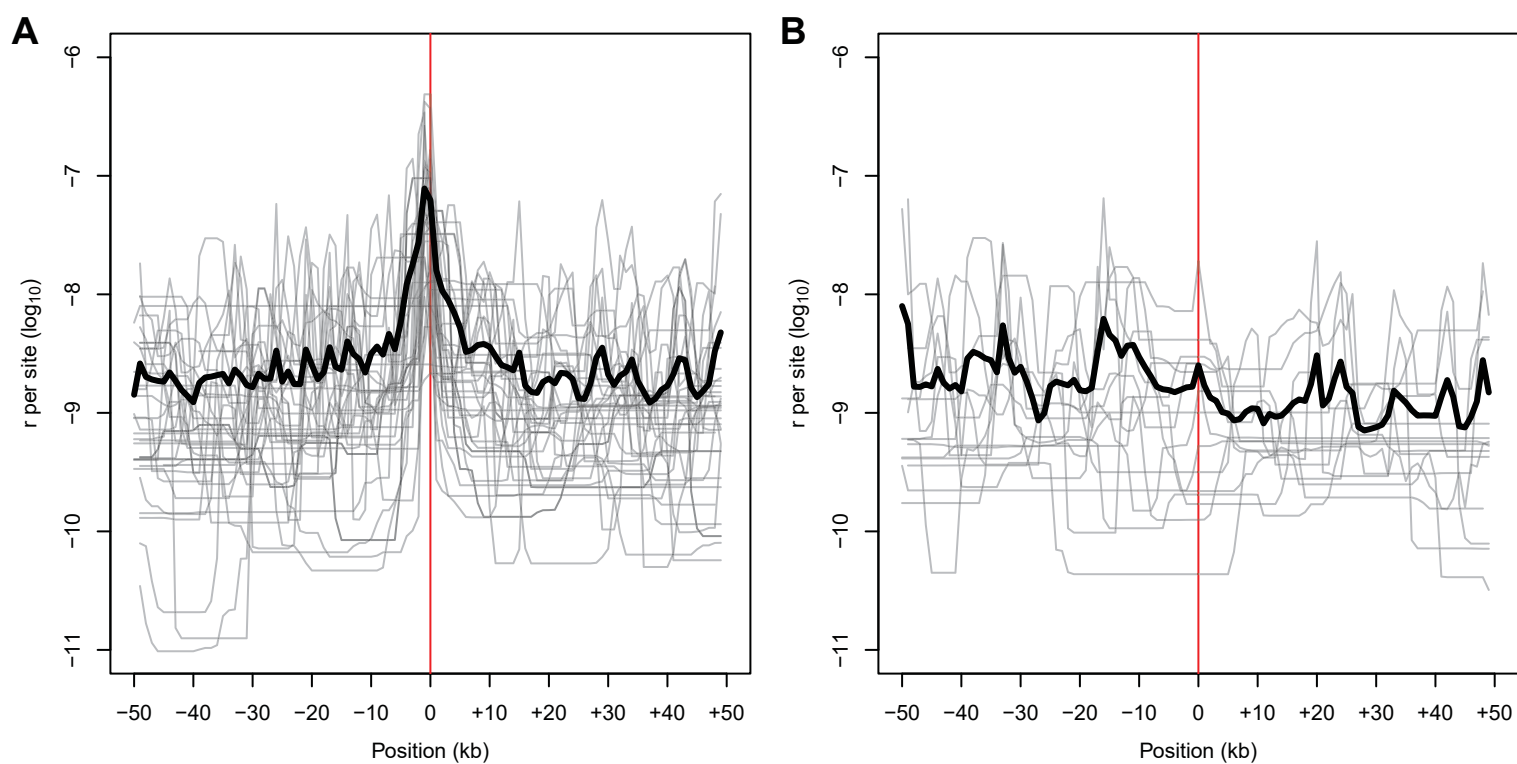
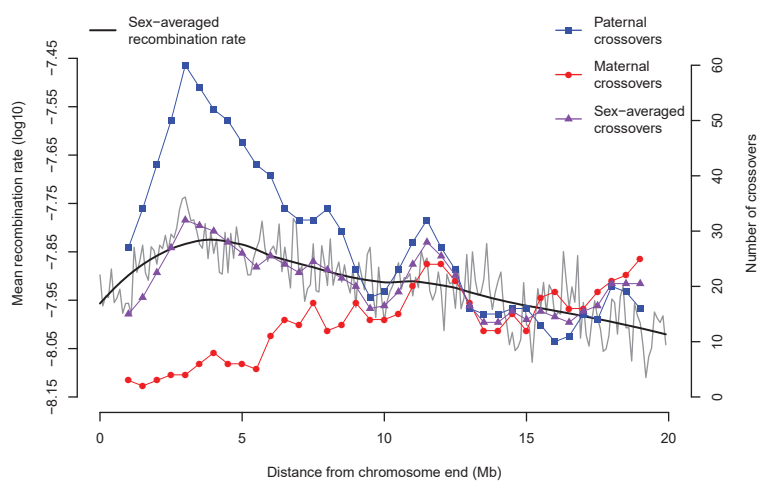


Figure 4

A



B

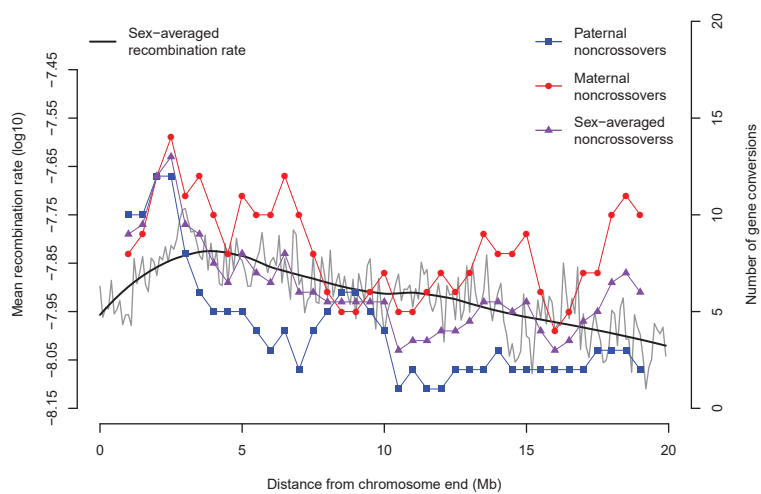


Figure 5

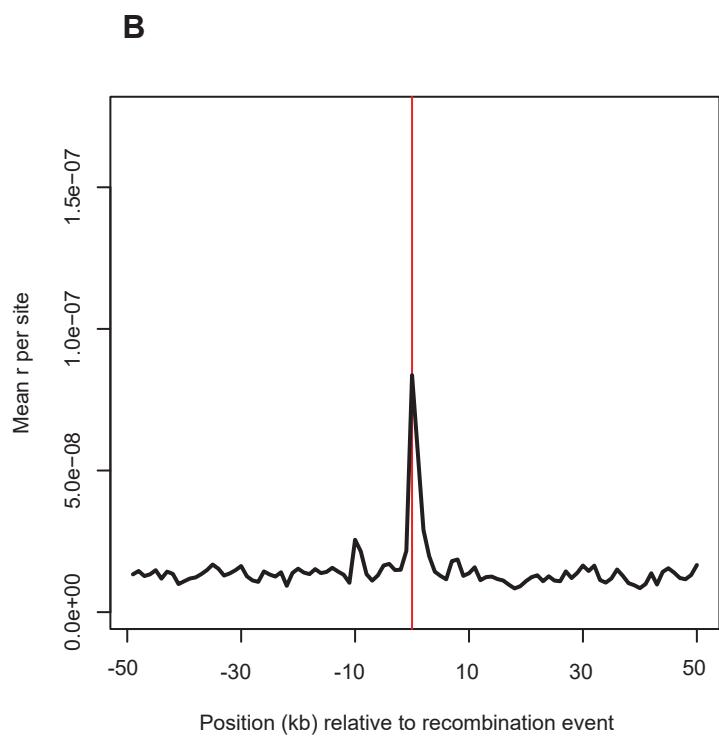
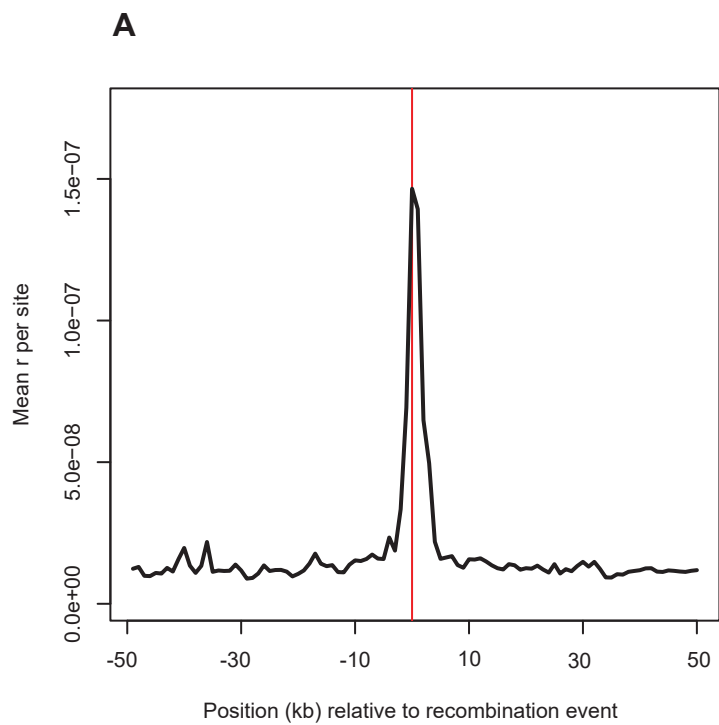
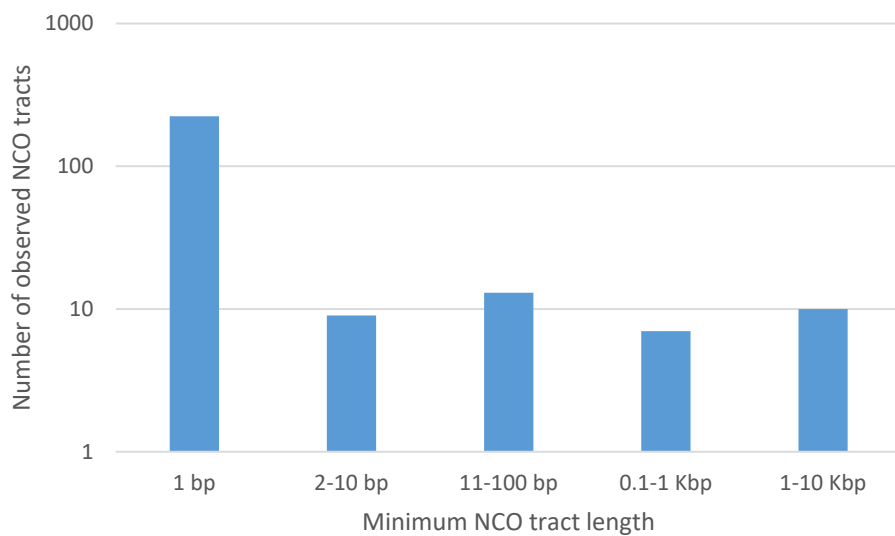


Figure 6

A



B

