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The genetic map and comparative analysis with the physical map of *Trypanosoma brucei*

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The publishers would like to apologise for two paragraphs being deleted from the print version of this paper. The complete and correct final four paragraphs of the Results and Discussion are given below:

The vast majority of markers demonstrate the inheritance of equal numbers of parental alleles in the progeny, in agreement with Mendelian predictions (13). However, there are two regions of the genetic map (on chromosomes III and IX) where there is segregation distortion, i.e. where one haplotype is inherited more frequently in the progeny than predicted (Figure 4). The origin of this distortion is unknown at the present time but could reflect selection acting on the uncloned progeny populations before cloning for alleles at loci in these regions of the chromosomes.

From the segregation analysis of markers, one progeny clone, F532/53 mcl 1, appears to be trisomic for chromosome I, having inherited both alleles from parental stock TREU 927 for all chromosome I markers analysed. Analysis of the inheritance of markers on other chromosomes, however, indicates that this hybrid has inherited only one homologue from each parental stock, clearly demonstrating that this progeny clone is trisomic for chromosome I, but not triploid. This is the first case of trisomy as opposed to triploidy reported in *T.brucei* and probably arose due to chromosomal non-disjunction at meiosis. The frequency of trisomy in this genetic cross is 2.5% and, while this clone cannot be used for mapping purposes for chromosome I, it is informative for all other chromosomes and so was included in the panel of informative hybrids.

Thirty-nine progeny clones for the 182 markers typed has generated a dataset of 6797 scored alleles (Supplementary Tables 1–11), and has identified two spontaneous mutation events, generating novel sized alleles distinct from parental alleles. These mutation events occurred at loci TB9/9 and TB9/13, giving a mutation frequency for each locus of 0.027 mutants/alleles genotyped. This generates an overall mutation frequency for all markers combined of 0.0003 mutants/alleles genotyped. These finding raises the question of the origin of the mutant allele. As these two mutant alleles replaced the original parental allele they could not have arisen during vegetative growth of the progeny clone but arose before the cloning process, possibly at meiosis. In both cases the mutant alleles had gained repeats generating alleles larger than the parental alleles and for mapping purposes it was assumed the parental allele closest in size to the mutant allele was the progenitor allele.

The high resolution genetic map we have generated for *T.brucei* for this pathogen has opened up the possibility of identifying genes that determine traits of importance by genetic analysis and positional cloning (1). The next challenge is to exploit this new toolset to understand better traits such as human infectivity (28) drug resistance or virulence (1) that all contribute to the severity of sleeping sickness and Nagana.