Corrigendum

Defective histone supply causes condensin-dependent chromatin alterations, SAC activation and chromosome decatenation impairment

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MNase-seq analyses performed after publication of this manuscript with the same thermosensitive smc2-8 strains revealed that, unexpectedly, they are wild type for SMC2. Instead, they carry a previously published mutation in a cohesin subunit (scc1-73) that also leads to thermosensitivity (1). SMC2 and scc1-73 were confirmed by standard DNA sequencing and their thermosensitivity was complemented by transformation with a plasmid expressing SCC1 (data not shown). These strains were derived by genetic crosses from strain CCG9127 (2). We brought this to the attention of the scientists who provided the strains, who have confirmed that due to an error in their strain collection the strain provided to us is indeed SMC2 scc1-73 (personal communication).

To determine whether the loss of condensin activity suppressed the phenotypes associated with histone depletion as claimed in our manuscript the original smc2-8 mutant (AS330 strain) (3) was backcrossed four times both to BY4147 and W303. We have sequenced this allele and observed that it contains 5 copies of Myc (273 bp) after the amino acid 466 (data not shown). As shown in Figure 4 corrected, smc2-8 suppressed the mitotic arrest and growth defects induced by histone depletion regardless of the accumulation of DNA damage. However, smc2-8 did not suppress neither the alterations of the centromeric structure (Figure 5C) nor the accumulation of catenanes induced by histone depletion (Figure 7C) (data not shown).

Consequently, the following findings remain unaltered: 1) histone depletion causes a metaphase arrest that is independent of the S phase checkpoints and dependent on the Aurora/Ipl1 kinase-mediated spindle assembly (SAC) checkpoint; 2) SAC activation by histone depletion is due to the accumulation of syntelic attachments that impair centromere biorientation; 3) histone depletion causes defective chromosome decatenation; 4) accordingly, the SAC prevents chromosome mis-segregation and cell lethality under conditions of defective histone supply; 5) SAC activation and defective growth are suppressed by the absence of Scc1 and Top2. Our current observation demonstrates that they are also suppressed by the lack of Scc1, extending the interplay between chromatin and Top2/condensin to cohesin. Thus, SAC activation by histone depletion is prevented by reducing the activity of any of the three major structural determinants of the intramolecular loop that provides the tensile properties of the centromere (4).

The reported error makes invalid our conclusion that smc2-8 suppresses the alterations of the centromeric chromatin and the accumulation of catenanes induced by histone depletion; instead, this suppression is mediated by scc1-73, pointing to cohesin as the major structural determinant responsible for histone supply-associated chromatin defects. Therefore, our new results are consistent with a similar model in which chromosome decatenation would require the positive supercoiling introduced by cohesins for Top2-mediated catenanes resolution, and that this supercoiling would be affected by histone depletion.

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The authors apologize for any inconvenience that this error may have caused the scientific community and readers of the journal.

Figure 4 corrected. The experiments were performed as in Figure 4 in BY4741 (similar results were obtained in W303).

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