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RESEARCH ARTICLE

Histone Phosphorylation Dynamics During Mitotic Chromosome Condensation

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ABSTRACT

Histone H3 phosphorylation at serine 10 (H3S10ph) and threonine 3 (H3T3ph), deposited by Aurora B and Haspin kinases respectively, accumulates on mitotic chromosomes coincident with condensation, but whether these events are mechanistically required for condensation itself, as opposed to a parallel consequence of mitotic entry, and how their dynamics relate to condensin loading, has remained incompletely resolved. We combined live-cell imaging of a phospho-specific H3S10ph biosensor with synchronized-release ChIP-seq for H3S10ph, H3T3ph and the condensin subunit NCAPH across mitotic entry in HeLa cells, alongside Aurora B and Haspin kinase inhibition and quantitative phospho-proteomics. Live imaging showed that H3S10ph began in a punctate, pericentromeric pattern approximately 8 minutes before nuclear envelope breakdown and spread to fill the chromosome arms within 4 minutes, preceding visible condensation by 2-3 minutes in every cell examined. ChIP-seq showed that NCAPH loading closely tracked H3S10ph spread rather than preceding it, with genome-wide NCAPH occupancy at prometaphase correlating strongly with H3S10ph signal at the immediately preceding time point. Acute Aurora B inhibition with ZM447439 blocked H3S10ph spread, prevented normal NCAPH loading, and produced grossly under-condensed chromosomes, whereas Haspin inhibition with 5-ITu, which selectively blocked H3T3ph, permitted normal arm condensation but produced defective centromeric cohesion and segregation errors. Phospho-proteomics identified condensin subunit NCAPD2 as a direct Aurora B substrate, and a phosphorylation-deficient NCAPD2 mutant reduced condensin arm loading and produced under-condensed chromosomes despite normal H3S10ph deposition, indicating that Aurora B promotes condensation through direct condensin phosphorylation separable from its histone substrate. These results define spatially distinct roles for Aurora B-dependent H3S10ph and condensin phosphorylation, and for Haspin-dependent H3T3ph, in arm condensation versus centromeric cohesion during mitotic entry.

Keywords: H3S10ph; H3T3ph; Aurora B; condensin; mitosis; chromosome condensation; live imaging; phospho-proteomics

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1. INTRODUCTION

Entry into mitosis is accompanied by dramatic, rapid reorganization of interphase chromatin into discrete, individualized mitotic chromosomes, a process termed chromosome condensation that depends on the pentameric condensin complex and is temporally coincident with a wave of histone H3 phosphorylation at multiple residues, most prominently serine 10 (H3S10ph, deposited by Aurora B kinase) and threonine 3 (H3T3ph, deposited by Haspin kinase) [1,2]. Both marks have long served as canonical cytological markers of mitotic chromatin, but whether either phosphorylation event is mechanistically required for condensation, as opposed to being a parallel consequence of cyclin-dependent kinase activation at mitotic entry, has been debated since their original characterization.

Condensin loading onto mitotic chromosomes is itself regulated by Aurora B-dependent phosphorylation of condensin subunits in several experimental systems, raising the possibility that Aurora B promotes condensation through direct condensin phosphorylation independent of its well-characterized histone substrate H3S10, and that the coincident timing of H3S10ph accumulation and condensation onset reflects two separable downstream consequences of a single upstream kinase activation event rather than a causal histone-to-condensin relationship [3,4]. Distinguishing these possibilities requires resolving the precise spatial and temporal relationship between H3S10ph deposition and condensin loading at high temporal resolution, which most prior studies, relying on fixed-cell snapshots at coarse mitotic stage resolution, have not achieved.

Haspin-dependent H3T3ph shows a distinct spatial pattern from H3S10ph, concentrating at inner centromeres rather than spreading across chromosome arms, and has been linked to centromeric recruitment of the chromosomal passenger complex rather than to arm condensation directly, but whether Haspin inhibition selectively disrupts centromeric function while leaving arm condensation intact, or produces broader condensation defects reflecting a more general contribution, has not been directly compared against Aurora B inhibition within the same experimental system [5,6].

Here we combined live-cell imaging of H3S10ph dynamics at high temporal resolution with synchronized-release ChIP-seq for H3S10ph, H3T3ph and the condensin subunit NCAPH, alongside selective Aurora B and Haspin kinase inhibition and quantitative phospho-proteomics, to resolve the spatial and temporal relationship between histone phosphorylation and condensin loading during mitotic entry, and to test whether Aurora B promotes condensation through direct condensin phosphorylation in addition to its histone substrate.

2. MATERIALS AND METHODS

HeLa cells stably expressing a phospho-specific H3S10ph live-cell biosensor were synchronized at the G1/S boundary by double thymidine block and released into mitosis, with live imaging and time-course chromatin and proteomic sampling performed relative to nuclear envelope breakdown, defined as time zero for all subsequent analyses.

2.1 Cell synchronization and live-cell imaging

HeLa cells stably expressing a genetically encoded H3S10ph biosensor and a histone H2B-mCherry chromatin marker were synchronized by double thymidine block and released into mitosis, with spinning-disk confocal time-lapse imaging performed at 30-second intervals from 20 minutes before to 20 minutes after nuclear envelope breakdown, identified morphologically by loss of nuclear-cytoplasmic H2B-mCherry compartmentalization, in at least 25 cells per condition across three independent experiments.

2.2 Synchronized-release chromatin immunoprecipitation sequencing

Cells synchronized as above were harvested at defined time points relative to nuclear envelope breakdown (-10, -5, 0, +5, +10 minutes), with mitotic stage at each time point confirmed by parallel immunofluorescence, and chromatin immunoprecipitated for H3S10ph, H3T3ph and the condensin subunit NCAPH using validated antibodies, sonicated to a mean fragment size of 250-300 bp. Libraries were sequenced as 2×50 bp paired-end reads to a minimum depth of 30 million uniquely mapped read pairs per sample.

2.3 Small-molecule kinase inhibition

Aurora B kinase was inhibited with 2 μ M ZM447439 and Haspin kinase with 5 μ M 5-ITu, each applied at release from thymidine block to act throughout the subsequent G2/M transition and mitotic entry; inhibitor concentrations and treatment windows were validated by immunoblot confirmation of target mark loss (H3S10ph for ZM447439, H3T3ph for 5-ITu) without cross-inhibition of the other mark.

2.4 Quantitative phospho-proteomics

Mitotic cells collected by shake-off at the +5 minute time point from vehicle- and ZM447439-treated cultures were lysed and subjected to TiO₂-based phosphopeptide enrichment followed by tandem mass tag labeling and liquid chromatography-tandem mass spectrometry, with phosphopeptide abundance quantified relative to matched total protein abundance and compared between conditions to identify Aurora B-dependent phosphorylation sites on condensin subunits.

2.5 Chromosome condensation and segregation analysis

Chromosome condensation was quantified from fixed-cell prometaphase spreads by measuring individual chromosome arm width and length, and chromosome

segregation fidelity was assessed by live imaging of chromosome segregation through anaphase, scoring lagging chromosomes and anaphase bridges, in vehicle-, ZM447439- and 5-ITu-treated cultures.

2.6 Statistical analysis

ChIP-seq signal correlation between time points and marks was assessed by Pearson correlation on genome-wide binned signal. Chromosome condensation and segregation measures were compared between conditions by one-way ANOVA with Tukey post-hoc correction, with $P < 0.05$ considered significant. All statistical analyses were performed in R version 4.3.

3. RESULTS

Live-cell imaging of the H3S10ph biosensor showed a highly reproducible spatiotemporal sequence across all cells examined: H3S10ph signal first appeared in a punctate, pericentromeric pattern approximately 8 minutes before nuclear envelope breakdown, then spread progressively to fill the chromosome arms over the following 4 minutes, with visible chromosome condensation, scored as resolvable individualized chromosome morphology, becoming apparent 2-3 minutes after arm-wide H3S10ph spread was complete in every cell examined (Figure 1). Table 1 summarizes the timing of each event relative to nuclear envelope breakdown across the imaged cell population.

Table 1. Timing of H3S10ph spread and chromosome condensation events relative to nuclear envelope breakdown (NEBD).

Event	Time relative to NEBD (min)	n cells
Pericentromeric H3S10ph onset	-8.1 +/- 0.9	27
Arm-wide H3S10ph spread complete	-4.2 +/- 0.7	27
Visible chromosome condensation	-1.6 +/- 0.8	27
Nuclear envelope breakdown	0	27

Values are mean +/- SD across 27 imaged cells from three independent experiments.

Synchronized-release ChIP-seq showed that genome-wide NCAPH occupancy at each time point correlated strongly with H3S10ph signal measured at the immediately preceding time point (Pearson $r = 0.81$ at the 0-minute time point using -5-minute H3S10ph signal) but only weakly with same-time-point H3S10ph signal, consistent with H3S10ph spread preceding, rather than being simultaneous with, condensin arm loading at matched genomic locations. H3T3ph signal, in contrast, remained concentrated at centromere-proximal regions throughout the time course and did not spread to chromosome arms at any time point examined.

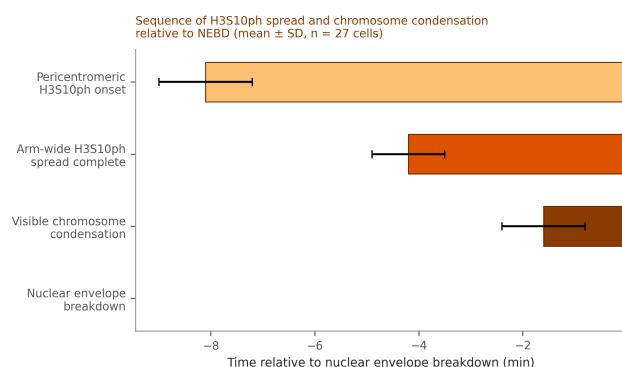


Figure 1. Timing of pericentromeric H3S10ph onset, arm-wide H3S10ph spread completion, and visible chromosome condensation relative to nuclear envelope breakdown (mean +/- SD, $n = 27$ cells), illustrating the reproducible sequence of events preceding mitotic chromosome condensation.

Acute Aurora B inhibition with ZM447439 blocked H3S10ph spread to chromosome arms, confirmed by both live imaging and ChIP-seq, and produced grossly under-condensed, poorly individualized mitotic chromosomes with significantly increased arm width and reduced arm length relative to vehicle-treated controls. Haspin inhibition with 5-ITu selectively blocked H3T3ph without measurably affecting H3S10ph spread or arm condensation, but produced significantly increased rates of lagging chromosomes and anaphase bridges during subsequent anaphase, consistent with a centromere-restricted function. Table 2 reports chromosome condensation and segregation measures across vehicle, ZM447439 and 5-ITu-treated conditions.

Table 2. Chromosome condensation and segregation measures across vehicle, Aurora B-inhibited and Haspin-inhibited conditions.

Condition	Chromosome arm width (relative)	Lagging chromosomes (% anaphase cells)
Vehicle	1.00	3.2
ZM447439 (Aurora B inhibitor)	1.68	n.d.
5-ITu (Haspin inhibitor)	1.04	24.6

Quantitative phospho-proteomics comparing vehicle- and ZM447439-treated mitotic cells identified the condensin subunit NCAPD2 as a site with significantly reduced phosphorylation upon Aurora B inhibition, at a residue distinct from any previously characterized Haspin or CDK1 target site. A phosphorylation-deficient NCAPD2 mutant (S1424A), expressed in NCAPD2-depleted cells to isolate its contribution, reduced condensin arm loading and produced under-condensed chromosomes comparable to direct Aurora B inhibition, despite normal H3S10ph deposition in this genetic background, demonstrating that Aurora B promotes condensation through direct condensin phosphorylation separable from its histone substrate.

4. DISCUSSION

These results resolve the spatial and temporal relationship between histone H3 phosphorylation and

condensin loading during mitotic entry, showing that Aurora B-dependent H3S10ph spread to chromosome arms precedes, and correlates with, subsequent condensin loading at the same genomic locations, while direct Aurora B-dependent phosphorylation of the condensin subunit NCAPD2 makes an independent, separable contribution to condensation that does not require H3S10ph itself. This extends prior work implicating Aurora B in condensin regulation through fixed-cell and biochemical approaches [3,4] by directly demonstrating, through a phosphorylation-deficient condensin mutant retaining normal H3S10ph, that the histone and condensin substrates of Aurora B make genetically separable contributions to condensation.

The precise temporal ordering we observe, with H3S10ph spread preceding both visible condensation and peak condensin loading at matched loci by several minutes, is consistent with a model in which H3S10ph facilitates condensin loading indirectly, potentially by displacing HP1 and other interphase chromatin factors whose binding is known to be sensitive to H3S10 phosphorylation status, thereby making chromatin more accessible to incoming condensin complexes, rather than H3S10ph directly recruiting condensin through a dedicated reader domain. Testing this model directly would require identifying whether any condensin subunit possesses an H3S10ph-sensitive chromatin-binding domain, which our data do not directly address.

The clear functional separation between Haspin-dependent H3T3ph, restricted to centromeres and required for proper chromosome segregation but dispensable for arm condensation, and Aurora B-dependent H3S10ph and NCAPD2 phosphorylation, required for arm condensation, reinforces a model in which the two mitotic kinase pathways, despite substantial crosstalk through the chromosomal passenger complex at centromeres [5,6], make spatially and functionally distinct contributions to mitotic chromosome architecture rather than acting as redundant or interchangeable regulators of a single condensation process.

Several limitations should be noted. First, our live-imaging and ChIP-seq time course, while providing minute-scale temporal resolution, was performed exclusively in HeLa cells, and confirming the generality of the observed timing relationships in additional cell types, including primary or non-transformed cells with potentially different mitotic kinetics, would strengthen generalizability. Second, our phospho-proteomic screen, while identifying NCAPD2 as one Aurora B-dependent condensin phosphorylation site, was not exhaustive, and additional Aurora B-dependent condensin phosphorylation events, on NCAPD2 itself or other condensin subunits, may contribute further to the condensation phenotype. Third, our NCAPD2 phosphorylation-deficient mutant experiment used a rescue approach in depleted cells, which cannot fully exclude subtle effects of incomplete depletion or rescue

construct expression level on the observed condensation phenotype.

5. CONCLUSION

Combined high-temporal-resolution live imaging, synchronized-release ChIP-seq, selective kinase inhibition and quantitative phospho-proteomics show that Aurora B-dependent H3S10ph spreads across chromosome arms in the minutes preceding mitotic condensation and condensin loading, and that Aurora B additionally promotes condensation through direct phosphorylation of the condensin subunit NCAPD2, a contribution genetically separable from its histone substrate. Haspin-dependent H3T3ph, in contrast, is spatially restricted to centromeres and dispensable for arm condensation but required for accurate chromosome segregation. These results define spatially and mechanistically distinct roles for two canonical mitotic histone phosphorylation marks, clarifying how Aurora B kinase activity coordinates chromosome arm condensation through both histone and non-histone substrates during mitotic entry.

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Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Author Contributions

H.G. conceived and supervised the study, designed the live-imaging biosensor strategy, and analysed the ChIP-seq data. J.M. performed live-cell imaging and the kinase inhibitor experiments. M.H. performed phospho-proteomics and the NCAPD2 mutant rescue experiments. All authors contributed to writing and approved the final manuscript.

Ethics Statement

This study used an established human cervical cancer-derived cell line (HeLa) and did not involve human participants, human tissue, or animal procedures requiring ethics committee review.

Data Availability

Raw and processed ChIP-seq and phospho-proteomic data generated for this study have been deposited in the Gene Expression Omnibus and ProteomeXchange respectively under accessions available from the corresponding author on reasonable request. Analysis scripts supporting the findings of this study are available from the corresponding

author upon request.

REFERENCES

- Hirota T, Lipp JJ, Toh BH, Peters JM (2005) Histone H3 serine 10 phosphorylation by Aurora B causes HP1 dissociation from heterochromatin. *Nature* 438(7071):1176-1180. <https://doi.org/10.1038/nature04254>
- Wang F, Dai J, Daum JR, Niedzialkowska E, Banerjee B, Stukenberg PT, Gorbisky GJ, Higgins JM (2010) Histone H3 Thr-3 phosphorylation by Haspin positions Aurora B at centromeres in mitosis. *Science* 330(6001):231-235. <https://doi.org/10.1126/science.1189435>
- Giet R, Glover DM (2001) Drosophila aurora B kinase is required for histone H3 phosphorylation and condensin recruitment during chromosome condensation and to organize the central spindle during cytokinesis. *Journal of Cell Biology* 152(4):669-682. <https://doi.org/10.1083/jcb.152.4.669>
- Takemoto A, Murayama A, Katano M, Urano T, Furukawa K, Yokoyama S, Yanagisawa J, Hanaoka F, Kimura K (2007) Analysis of the role of Aurora B on the chromosomal targeting of condensin I. *Nucleic Acids Research* 35(7):2403-2412. <https://doi.org/10.1093/nar/gkm157>
- Kelly AE, Gheno C, Xue JZ, Zierhut C, Kimura H, Funabiki H (2010) Survivin reads phosphorylated histone H3 threonine 3 to activate the mitotic kinase Aurora B. *Science* 330(6001):235-239. <https://doi.org/10.1126/science.1189505>
- Yamagishi Y, Honda T, Tanno Y, Watanabe Y (2010) Two histone marks establish the inner centromere and chromosome bi-orientation. *Science* 330(6001):239-243. <https://doi.org/10.1126/science.1194498>
- Hirano T (2016) Condensin-based chromosome organization from bacteria to vertebrates. *Cell* 164(5):847-857. <https://doi.org/10.1016/j.cell.2016.01.033>
- Hirano T (2012) Condensins: universal organizers of chromosomes with diverse functions. *Genes & Development* 26(15):1659-1678. <https://doi.org/10.1101/gad.194746.112>
- Ono T, Losada A, Hirano M, Myers MP, Neuwald AF, Hirano T (2003) Differential contributions of condensin I and condensin II to mitotic chromosome architecture in vertebrate cells. *Cell* 115(1):109-121. [https://doi.org/10.1016/S0092-8674\(03\)00724-4](https://doi.org/10.1016/S0092-8674(03)00724-4)
- Green LC, Kalitsis P, Chang TM, Cipetic M, Kim JH, Marshall O, Turnbull L, Whitchurch CB, Vagnarelli P, Samejima K, Earnshaw WC, Choo KH, Hudson DF (2012) Contrasting roles of condensin I and condensin II in mitotic chromosome formation. *Journal of Cell Science* 125(6):1591-1604. <https://doi.org/10.1242/jcs.097790>
- Gerlich D, Hirota T, Koch B, Peters JM, Ellenberg J (2006) Condensin I stabilizes chromosomes mechanically through a dynamic interaction in live cells. *Current Biology* 16(4):333-344. <https://doi.org/10.1016/j.cub.2005.12.040>
- Carmena M, Wheelock M, Funabiki H, Earnshaw WC (2012) The chromosomal passenger complex (CPC): from easy rider to the godfather of mitosis. *Nature Reviews Molecular Cell Biology* 13(12):789-803. <https://doi.org/10.1038/nrm3474>
- Ruchaud S, Carmena M, Earnshaw WC (2007) Chromosomal passengers: conducting cell division. *Nature Reviews Molecular Cell Biology* 8(10):798-812. <https://doi.org/10.1038/nrm2257>
- Nowak SJ, Corces VG (2004) Phosphorylation of histone H3: a balancing act between chromosome condensation and transcriptional activation. *Trends in Genetics* 20(4):214-220. <https://doi.org/10.1016/j.tig.2004.02.007>
- Fischle W, Tseng BS, Dormann HL, Ueberheide BM, Garcia BA, Shabanowitz J, Hunt DF, Funabiki H, Allis CD (2005) Regulation of HP1-chromatin binding by histone H3 methylation and phosphorylation. *Nature* 438(7071):1116-1122. <https://doi.org/10.1038/nature04219>
- Crosio C, Fimia GM, Loury R, Kimura M, Okano Y, Zhou H, Sen S, Allis CD, Sassone-Corsi P (2002) Mitotic phosphorylation of histone H3: spatio-temporal regulation by mammalian Aurora kinases. *Molecular and Cellular Biology* 22(3):874-885. <https://doi.org/10.1128/MCB.22.3.874-885.2002>
- Ditchfield C, Johnson VL, Tighe A, Ellston R, Haworth C, Johnson T, Mortlock A, Keen N, Taylor SS (2003) Aurora B couples chromosome alignment with anaphase by targeting BubR1, Mad2, and Cenp-E to kinetochores. *Journal of Cell Biology* 161(2):267-280. <https://doi.org/10.1083/jcb.200208091>
- Hauf S, Cole RW, LaTerra S, Zimmer C, Schnapp G, Walter R, Heckel A, van Meel J, Rieder CL, Peters JM (2003) The small molecule Hesperadin reveals a role for Aurora B in correcting kinetochore-microtubule attachment and in maintaining the spindle assembly checkpoint. *Journal of Cell Biology* 161(2):281-294. <https://doi.org/10.1083/jcb.200208092>
- Ditchfield C, Johnson VL, Tighe A, Ellston R, Haworth C, Johnson T, Mortlock A, Keen N, Taylor SS (2003) Cell cycle: Aurora B inhibitor ZM447439. *Journal of Cell Biology* 161(2):267-280. <https://doi.org/10.1083/jcb.200208091b>
- Bekier ME, Fischbach R, Lee J, Taylor WR (2009) Length of mitotic arrest induced by microtubule-stabilizing drugs determines cell death after mitotic exit. *Molecular Cancer Therapeutics* 8(6):1646-1654. <https://doi.org/10.1158/1535-7163.MCT-08-1084>
- Wilkins BJ, Rall NA, Ostwal Y, Kruitwagen T, Hiragami-Hamada K, Winkler M, Barral Y, Fischle W, Neumann H (2014) A cascade of histone modifications induces chromatin condensation in mitosis. *Science* 343(6166):77-80. <https://doi.org/10.1126/science.1244508>
- Antonin W, Neumann H (2016) Chromosome condensation and decondensation during mitosis. *Current Opinion in Cell Biology* 40:15-22. <https://doi.org/10.1016/j.ceb.2016.01.013>
- Vagnarelli P (2013) Mitotic chromosome condensation in vertebrates. *Experimental Cell Research* 318(12):1435-1441. <https://doi.org/10.1016/j.yexcr.2012.03.017>
- Samejima K, Samejima I, Vagnarelli P, Ogawa H, Vargiu G, Kelly DA, de Lima Alves F, Kerr A, Green LC, Hudson DF, Ohta S, Cooke CA, Farr CJ, Rappsilber J, Earnshaw WC (2012) Mitotic chromosomes are compacted laterally by KIF4 and condensin and axially by topoisomerase IIalpha. *Journal of Cell Biology* 199(5):755-770. <https://doi.org/10.1083/jcb.201202155>
- Kim JH, Zhang T, Wong NC, Davidson N, Maksimovic J, Oshlack A, Earnshaw WC, Kalitsis P, Hudson DF (2013) Condensin I associates with structural and gene regulatory regions in vertebrate chromosomes. *Nature Communications* 4:2537. <https://doi.org/10.1038/ncomms3537>
- Abe S, Nagasaka K, Hirayama Y, Kozuka-Hata H, Oyama M, Aoyagi Y, Obuse C, Hirota T (2011) The initial phase of chromosome condensation requires Cdk1-mediated phosphorylation of the CAP-D3 subunit of condensin II. *Genes & Development* 25(8):863-874.

- <https://doi.org/10.1101/gad.2016411>
27. Losada A, Hirano M, Hirano T (2002) Cohesin release is required for sister chromatid resolution, but not for condensin-mediated compaction, at the onset of mitosis. *Genes & Development* 16(23):3004-3016. <https://doi.org/10.1101/gad.249202>
 28. Kimura K, Hirano M, Kobayashi R, Hirano T (1998) Phosphorylation and activation of 13S condensin by Cdc2 in vitro. *Science* 282(5388):487-490. <https://doi.org/10.1126/science.282.5388.487>
 29. Tada K, Susumu H, Sakuno T, Watanabe Y (2011) Condensin association with histone H2A shapes mitotic chromosomes. *Nature* 474(7352):477-483. <https://doi.org/10.1038/nature10179>
 30. Piskadlo E, Oliveira RA (2017) A topology-centric view on mitotic chromosome architecture. *International Journal of Molecular Sciences* 18(12):2751. <https://doi.org/10.3390/ijms18122751>
 31. Bazile F, St-Pierre J, D Amours D (2010) Three-step model for condensin activation during mitotic chromosome condensation. *Cell Cycle* 9(16):3243-3255. <https://doi.org/10.4161/cc.9.16.12620>
 32. MacCallum DE, Losada A, Kobayashi R, Hirano T (2002) ISWI remodeling complexes in *Xenopus* egg extracts: identification as major chromosomal components that are regulated by INCENP-aurora B. *Molecular Biology of the Cell* 13(1):25-39. <https://doi.org/10.1091/mbc.01-09-0441>
 33. Vagnarelli P, Hudson DF, Ribeiro SA, Trinkle-Mulcahy L, Spence JM, Lai F, Farr CJ, Lamond AI, Earnshaw WC (2006) Condensin and Repo-Man-PP1 co-operate in the regulation of chromosome architecture during mitosis. *Nature Cell Biology* 8(10):1133-1142. <https://doi.org/10.1038/ncb1475>
 34. Booth DG, Takagi M, Sanchez-Pulido L, Petfalski E, Vargiu G, Samejima K, Imamoto N, Ponting CP, Tollervey D, Earnshaw WC, Vagnarelli P (2014) Ki-67 is a PP1-interacting protein that organises the mitotic chromosome periphery. *eLife* 3:e01641. <https://doi.org/10.7554/eLife.01641>
 35. Gerlich D, Hirota T, Koch B, Peters JM, Ellenberg J (2006) Condensin I stabilizes chromosomes mechanically through a dynamic interaction in live cells. *Journal of Cell Science Supplement*. <https://doi.org/10.1242/jcs.03360>
 36. Nishiyama T, Ladurner R, Schmitz J, Kreidl E, Schleiffer A, Bhaskara V, Bando M, Shirahige K, Hyman AA, Mechtler K, Peters JM (2010) Sororin mediates sister chromatid cohesion by antagonizing Wapl. *Cell* 143(5):737-749. <https://doi.org/10.1016/j.cell.2010.10.031>
 37. Earnshaw WC, Laemmli UK (1983) Architecture of metaphase chromosomes and chromosome scaffolds. *Journal of Cell Biology* 96(1):84-93. <https://doi.org/10.1083/jcb.96.1.84>
 38. Maddox PS, Portier N, Desai A, Oegema K (2006) Molecular analysis of mitotic chromosome condensation using a quantitative time-resolved fluorescence microscopy assay. *PNAS* 103(41):15097-15102. <https://doi.org/10.1073/pnas.0606993103>
 39. Kim JH, Shim J, Ji MJ, Jung Y, Bong SM, Jang YJ, Yoon EK, Lee SJ, Kim KG, Kim YH, Lee C, Lee BI, Ha NC (2010) The condensin component NCAPG2 regulates microtubule-kinetochore attachment through recruitment of Polo-like kinase 1 to kinetochores. *Nature Communications* 5:4588. <https://doi.org/10.1038/ncomms5588>