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**Replication** - Alan Kolber - 2012-03-04

**Mechanism and Regulation of DNA Replication** - Alan Kolber - 2012-03-04

**Cell Cycle Regulation** - Philipp Kaldis - 2010-11-18
This book is a state-of-the-art summary of the latest achievements in cell cycle control research
identify the mechanisms responsible for the cancer research. The chapters are written by internationally leading experts in the field. They provide an updated view on how the cell cycle is regulated in vivo, and about the involvement of cell cycle regulators in cancer.

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**The Regulation of DNA Replication and Transcription** - Mirko Beljanski - 2013-03-26
"The Regulation of DNA Replication and Transcription explores basic processes of DNA replication and transcription in an effort to release of genetic information and its role in the regulation of cellular events. Concerned with discovering the fundamental concept that might integrate and explain the wide range of existing lines of evidence, the author reports and interprets the results of experiments conducted in an impressive range of biological systems. Focused on complex mechanisms at the biochemical level, these studies allow analysis of the pathways involved when cells, organs and animal systems react to various trigger molecules derived from both living cells and exogenous sources. These include hormones, RNA, RNA fragments, alkaloids, actinomycin D, and phorbol esters, as well as chemical carcinogens and drugs. Combining the results of these studies with his own extensive work in this field, the author is able to formulate a uniquely integrative biochemical model for the gene expression, demonstrating that both biological and chemically synthesized molecules can trigger
biochemical level, these studies allow analysis of DNA and thus influence cell transformation. Apart from its academic significance, the model offers high potential assistance in the search for ways to induce or control the expression of certain genes and, moreover, to promote differentiation of given cells in vitro as well as in situ."

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**DNA Replication Control in Microbial Cell**
cellular metabolism may involve interactions of replication proteins with other macromolecular complexes, responsible for various cellular processes. Thus, the exact set of factors participating in triggering the replication initiation may differ depending on growth conditions. Therefore, understanding the regulation of DNA duplication requires placing this process in the context of the current knowledge on bacterial metabolism, as well as cellular and chromosomal structure. Moreover, in both Escherichia coli and eukaryotic cells, replication initiator proteins were shown to play other roles in addition to driving the assembly of replication complexes, which constitutes another, yet not sufficiently understood, layer of coordinating DNA replication with the cell cycle.

DNA Replication Control in Microbial Cell Factories - Monika Glinkowska - 2014-09-23
This work describes the current knowledge of biochemical mechanisms regulating initiation of DNA replication in Escherichia coli, which
participating in triggering the replication protein. Examples of direct linkages between DNA replication and other cellular processes are provided. In addition, similarities of the mechanisms of regulation of DNA replication operating in prokaryotic and eukaryotic cells are identified, and implications for understanding more complex processes, like carcinogenesis are suggested. Studies of recent years provided evidence that regulation of DNA replication in bacteria is more complex than previously anticipated. Multiple layers of control seem to ensure coordination of this process with the increase of cellular mass and the division cycle. Metabolic processes and membrane composition may serve as points where integration of genome replication with growth conditions occurs. It is also likely that coupling of DNA synthesis with cellular metabolism may involve interactions of replication proteins with other macromolecular complexes, responsible for various cellular processes. Thus, the exact set of factors

initiation may differ depending on growth conditions. Therefore, understanding the regulation of DNA duplication requires placing this process in the context of the current knowledge on bacterial metabolism, as well as cellular and chromosomal structure. Moreover, in both Escherichia coli and eukaryotic cells, replication initiator proteins were shown to play other roles in addition to driving the assembly of replication complexes, which constitutes another, yet not sufficiently understood, layer of coordinating DNA replication with the cell cycle.

**Regulation of DNA Replication and Repair by DNA Polymerase Delta Interacting Protein 1 (PDIP1)** - Tianli Xia - 2006

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**DNA Replication** - Hisao Masai - 2018-01-22
the existing concepts ranging from the old directions of DNA replication research. The contents reflect upon the principles that have been established through the genetic and enzymatic studies of bacterial, viral, and cellular replication during the past decades. The book begins with a historical overview of the studies on eukaryotic DNA replication by Professor Thomas Kelly, a pioneer of the field. The following chapters include genome-wide studies of replication origins and initiation factor binding, as well as the timing of DNA replications, mechanisms of initiation, DNA chain elongation and termination of DNA replication, the structural basis of functions of protein complexes responsible for execution of DNA replication, cell cycle-dependent regulation of DNA replication, the nature of replication stress and cells’ strategy to deal with the stress, and finally how all these phenomena are interconnected to genome instability and development of various diseases. By reviewing principles to the newest ideas, the book gives readers an opportunity to learn how the classical replication principles are now being modified and new concepts are being generated to explain how genome DNA replication is achieved with such high adaptability and plasticity. With the development of new methods including cryoelectron microscopy analyses of huge protein complexes, single molecular analyses of initiation and elongation of DNA replication, and total reconstitution of eukaryotic DNA replication with purified factors, the field is enjoying one of its most exciting moments, and this highly timely book conveys that excitement to all interested readers.

DNA Replication - Hisao Masai - 2018-01-22
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**Mechanism and Regulation of DNA Replication**
- A. R. Kolber - 1974

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**Co-regulation of Chromatin Assembly and DNA Replication**
- Alexa Anne Franco - 2004

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- Alexa Anne Franco - 2004
The E2F/RB Pathway Regulation of DNA Replication and Protein Biosynthesis - William O. Ward - 2003

The Regulation of DNA Replication and Transcription - Carl M. Mansfield - 1976

Regulation of DNA Replication Through the Cell Cycle in Yeast S. Cerevisiae - Hai Rao - 1996

Regulation of DNA Replication in Xenopus Laevis - Jing Fang - 1996

Regulation of DNA Replication by CDC-48 Cofactors - Paul A. Pirson - 2014

Regulation of DNA Replication Initiation in Yeast Saccharomyces Cerevisiae - Min Ding - 1996

Regulation of DNA Replication and Repair by Diadenosine Tetraphosphate - Ryan Cunningham - 2010
DNA replication, the process of copying one double stranded DNA molecule to produce two identical copies, is at the heart of cell proliferation. This book highlights new insights into the replication process in eukaryotes, from the assembly of pre-replication complex and features of DNA replication origins, through polymerization mechanisms, to propagation of epigenetic states. It also covers cell cycle control of replication initiation and includes the latest on mechanisms of replication in prokaryotes. The association between genome replication and transcription is also addressed. We hope that readers will find this book interesting, helpful and inspiring.

Regulation of Deoxyribonucleic Acid Replication and Cellular Responses to Perturbations in Replication in the Bacterium B. Subtilis - Alexi I. Goranov - 2006
(Cont.) My results demonstrate that the conserved recombination protein, RecA,
Regulation of Deoxyribonucleic Acid under the tested conditions. More than 75% of the RecA-mediated transcriptional response is due to the expression of phage and mobile element genes and their indirect effects. Under conditions of replication elongation arrest, there is still a significant recA-independent response, at least part of which is mediated by the replication protein DnaA. The DnaA-mediated response appears to be conserved in other bacteria, as homologues if the affected genes also have DnaA binding sites in their promoter regions. Previously, one of the DnaA regulated genes, sda, has been shown to affect cell viability after perturbations in replication. Here I showed that another DnaA-regulated gene, ftsL, also affects cell survival after replication arrest by coordinating replication and cell-division. I believe that my results have furthered our understanding of how replication is coordinated with other cell-cycle processes, and have raised interesting questions for future investigation.

Replication and Cellular Responses to Perturbations in Replication in the Bacterium B. Subtilis - Alexi I. Goranov - 2006 (Cont.) My results demonstrate that the conserved recombination protein, RecA, mediates most of the transcriptional response under the tested conditions. More than 75% of the RecA-mediated transcriptional response is due to the expression of phage and mobile element genes and their indirect effects. Under conditions of replication elongation arrest, there is still a significant recA-independent response, at least part of which is mediated by the replication protein DnaA. The DnaA-mediated response appears to be conserved in other bacteria, as homologues if the affected genes also have DnaA binding sites in their promoter regions. Previously, one of the DnaA regulated genes, sda, has been shown to affect cell viability after perturbations in replication. Here I showed that another DnaA-regulated gene, ftsL, also...
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The Regulation of DNA Replication and Cell Division in E. Coli B/r - - 1968

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The Regulation of DNA Replication and Transcription - Mirko Beljanski - 1983

The Regulation of DNA Replication and Transcription - Mirko Beljanski - 1983

Regulation of DNA Replication During Meiosis in Fission Yeast - Hui Hua - 2012
The interval between meiotic nuclear divisions can be regarded as a modified mitotic cell cycle regulating this critical aspect of meiosis that allows haploid cells to be generated from a diploid progenitor were investigated in this project. Licensing is restricted after meiosis I due to down-regulation of Cdc18 and Cdt1. Late meiotic expression of Cdc18 and Cdt1, which load the MCM helicase onto replication origins, can lead to partial DNA replication after meiosis I. This implies that block to initiation via licensing forms an important component of this regulation. As detecting any minor DNA re-
replication after meiosis I requires a technique more sensitive than flow cytometry for detection of total cell DNA contents, I also investigated a procedure to allow incorporation and detection of 5-ethynyl-2'-deoxyuridine (EdU) in fission yeast. Additional inactivation of Spd1 or stabilization of Dfp1 after MI when Cdc18 and Cdt1 are also expressed does not enhance re-replication, but cyclin-dependent kinase Cdc2 plays a role in preventing re-replication during the MI-MII
can lead to partial DNA replication after meiosis is subverted, replication forks only move a short distance in the interval between meiosis I and II, implying that the elongation step of DNA replication is also inefficient. In addition, I show that the regulation of entry into meiosis II is not delayed by a partial round of DNA replication or DNA damage, indicating that replication and DNA damage checkpoints do not operate in late meiosis.

Regulation of DNA Replication During Meiosis in Fission Yeast - Hui Hua - 2012

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DNA damage checkpoints do not operate in late meiosis.

Regulation of DNA Replication Initiation in a Baculovirus, AcMNPV - - 1998

Regulation of DNA Replication Initiation in a Baculovirus, AcMNPV - - 1998

DNA Replication Control in Microbial Cell Factories - Monika Glinkowska - 2014-10-31

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Cell Cycle Regulation of DNA Replication in Xenopus Egg Extracts - Piotr Romanowski - 1998

Cell Cycle Regulation of DNA Replication in Xenopus Egg Extracts - Piotr Romanowski - 1998

Replicon Theory, Regulation of DNA Replication and Genomic Instability - Michel Debatisse - 2013

Replicon Theory, Regulation of DNA Replication and Genomic Instability - Michel Debatisse - 2013

Regulation of DNA Replication and Transcription: Functional and Structural Analysis of SV40- and Monkey-derived CIS-acting Elements - Paul J. Szymanski - 1990

Regulation of DNA Replication and Transcription: Functional and Structural Analysis of SV40- and Monkey-derived CIS-acting Elements - Paul J. Szymanski - 1990

The Regulation of DNA Replication During Drosophila Development - Janet Lynn Carminati - 1995

The Regulation of DNA Replication During
DNA replication is a fundamental part of the life cycle of all organisms. Not surprisingly many aspects of this process display profound conservation across organisms in all domains of life. The chapters in this volume outline and review the current state of knowledge on several key aspects of the DNA replication process. This is a critical process in both normal growth and development and in relation to a broad variety of pathological conditions including cancer. The reader will be provided with new insights into the initiation, regulation, and progression of DNA replication as well as a collection of thought provoking questions and summaries to direct future investigations.

**The Mechanisms of DNA Replication** - David Stuart - 2013-02-20

**Transcription Factors and DNA Replication** - David Scott Pederson - 1994

Initiation of DNA synthesis: a general model from studies with prokaryotes. Regulatory transcription factors and the initiation of transcription in eucaryotic cells. Replication origins and the initiation of DNA synthesis in...
Duplication of the genome during S phase of the mitotic cell cycle begins at thousands of sites along chromosomes termed origins of replication. Although many of the essential protein components catalyzing events at these sites are known and are conserved throughout eukaryotes, the likelihood or efficiency of initiation of DNA synthesis at any given genomic site is expected to be influenced by other novel factors, including aspects of chromatin and DNA structure. Here I show that increased histone H4 acetylation at replication origin loci occurs after treatment with the histone deacetylase inhibitor TSA and coincides with a loss of specific initiation site selection both within origin loci and throughout the genome. Furthermore, new replication initiation sites become activated or used with greater frequency after treatment with TSA, and TSA promotes the activation of replication origins earlier during the S phase of the cell.
during evolution with the development of a novel histone acetylation in controlling the initiation of DNA synthesis at specific chromosomal sites. Regions of helically unstable DNA termed DNA unwinding elements (DUEs) are commonly found at replication origins, and our laboratory identified a DUE-binding protein (DUE-B) using the c-myc DUE in a yeast one-hybrid screen. Here I demonstrate that DUE-B is required for efficient entry into S phase in human cells and for efficient replication in the Xenopus egg extract replication system. Structural analyses show the N-terminal portion of the protein to be identical to that of bacterial D-aminoacyl-tRNA deacylases. Human DUE-B possesses this function in vitro and the ability to hydrolyze ATP, suggesting that DUE-B may be a multifunctional esterase. Unique to vertebrate homologs of DUE-B is a C-terminal extension of 62 amino acids that binds DNA and is targeted for phosphorylation by CK2. The addition of the C-terminal domain to DUE-B in higher eukaryotes may have coincided function for this protein in the initiation of DNA replication. Together these two sets of data argue that previously uncharacterized factors regulate the initiation of DNA replication in higher eukaryotes, possibly to deal with the complex chromosomal architecture found in these organisms.

**Regulation of DNA Replication Initiation by Histone Acetylation and the DNA Unwinding Element Binding Protein DUE-B** - Michael George Kemp - 2006

Duplication of the genome during S phase of the mitotic cell cycle begins at thousands of sites along chromosomes termed origins of replication. Although many of the essential protein components catalyzing events at these sites are known and are conserved throughout eukaryotes, the likelihood or efficiency of initiation of DNA synthesis at any given genomic site is expected to be influenced by other novel factors, including aspects of chromatin and DNA structure. Here I
show the N-terminal portion of the protein to be replication origin loci occurs after treatment with the histone deacetylase inhibitor TSA and coincides with a loss of specific initiation site selection both within origin loci and throughout the genome. Furthermore, new replication initiation sites become activated or used with greater frequency after treatment with TSA, and TSA promotes the activation of replication origins earlier during the S phase of the cell cycle. These data suggest a physiological role for histone acetylation in controlling the initiation of DNA synthesis at specific chromosomal sites. Regions of helically unstable DNA termed DNA unwinding elements (DUEs) are commonly found at replication origins, and our laboratory identified a DUE-binding protein (DUE-B) using the c-myc DUE in a yeast one-hybrid screen. Here I demonstrate that DUE-B is required for efficient entry into S phase in human cells and for efficient replication in the Xenopus egg extract replication system. Structural analyses identical to that of bacterial D-aminoacyl-tRNA deacylases. Human DUE-B possesses this function in vitro and the ability to hydrolyze ATP, suggesting that DUE-B may be a multifunctional esterase. Unique to vertebrate homologs of DUE-B is a C-terminal extension of 62 amino acids that binds DNA and is targeted for phosphorylation by CK2. The addition of the C-terminal domain to DUE-B in higher eukaryotes may have coincided during evolution with the development of a novel function for this protein in the initiation of DNA replication. Together these two sets of data argue that previously uncharacterized factors regulate the initiation of DNA replication in higher eukaryotes, possibly to deal with the complex chromosomal architecture found in these organisms.

**Regulation of DNA Replication During Conventional and Unconventional Cell Cycles in Tetrahymena** - Po-Hsuen Lee - 2015

As the nucleating protein for pre-replicative
regulation-of-dna-replication-and-transcription

During the vegetative cell cycle of Tetrahymena thermophila, previously published work has shown that DNA replication initiates from defined chromosomal sites in an ORC-dependent manner. Tetrahymena exhibits nuclear dimorphism, a polyploid somatic macronucleus (MAC), which is transcriptionally active and maintains vegetative growth, and a diploid germline micronucleus (MIC) responsible for the transmission of genetic information during conjugation. In order to provide more information about the fundamental mechanisms of micro- and macro-nuclear replication programs, I study the impacts of changing in ORC protein contents on the fate of micro- and macro-nuclear chromosomes during the vegetative cell cycle and development in Tetrahymena. I examined the effect of down-regulation of ORC1 on genome stability and intra-S phase checkpoint activation by disrupting Recognition Complex (ORC) specifies where replication initiates in eukaryotic chromosomes. ORC1 gene in the macronucleus. Partial depletion of Orc1p leads to genome instability in the diploid mitotic micronucleus, abnormal division of the polyploid amitotic macronucleus, and failure to mount a robust intra-S phase checkpoint response. In addition, the ORC1 knockdown strain fails to execute two developmentally-regulated DNA replication programs, endoreplication and ribosomal DNA (rDNA) gene amplification. I also examined the regulation of ORC and MCM during development. Remarkably, the result suggests that the demand on the ORC-dependent replication machinery differs during development and the vegetative S phase. To further gain new insights into fundamental mechanisms that protect chromosomes from replication stress, I examined the impact of replication stress on the regulation of ORC and MCM. This study led to the discovery of a novel DNA replication program that is activated under HU treatment. While...
replication initiates in eukaryotic chromosomes. In response to HU, cells were competent to complete S phase in the absence of Orc1p and Mcm6p after HU was removed. In addition, the rDNA origin used exclusively during the S phase of vegetative cell cycle and developmentally programmed gene amplification is suppressed when these replication proteins are selectively degraded under HU treatment. Instead, an alternative program was used to resume the cell cycle progression. These data provide compelling evidence for an ORC-independent DNA replication program in cells recovering from replication stress. The electronic version of this dissertation is accessible from http://hdl.handle.net/1969.1/152509

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Transcriptional Silencing and Regulation of DNA Replication - David Clifford Zappulla - 2002

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Regulation of DNA replication is a highly regulated process across all organisms. Improper regulation of DNA replication can be detrimental. I identified an overinitiating, conditional synthetic lethal mutant of Bacillus subtilis. I isolated suppressors of this mutant and uncovered novel genes associated with DNA replication. These suppressors acted both at the steps of initiation and elongation to overcome the detrimental replication initiation of the synthetic lethal [delta]yabA dnaA 1 mutant. One class of suppressors decreased levels of the replicative
DNA replication is a highly regulated process. Helicase are sufficient to limit replication initiation under fast growth conditions. I also explored the regulation of DnaA as a transcription factor. The replication initiation inhibitor, YabA, binds to DnaA and prevents its cooperative binding at the origin. In addition to its role in replication initiation, DnaA also directly regulates expression of several genes. YabA has been shown to inhibit DnaA binding at several promoters but its effect on DnaA-mediated gene expression is unclear. I found that YabA inhibits sda activation by DnaA but does not significantly affect repression of ywIC by DnaA. Lastly, I showed that YabA appears to stimulate autoregulation of dnaA. This preliminary data illustrates a role for YabA regulation in DnaA-mediated gene expression.

Regulation of DNA Replication and the Replication Initiator, DnaA, in Bacillus Subtilis - Mary Elizabeth Anderson (Ph. D.) - 2019

across all organisms. Improper regulation of DNA replication can be detrimental. I identified an overinitiating, conditional synthetic lethal mutant of Bacillus subtilis. I isolated suppressors of this mutant and uncovered novel genes associated with DNA replication. These suppressors acted both at the steps of initiation and elongation to overcome the detrimental replication initiation of the synthetic lethal [delta]yabA dnaA 1 mutant. One class of suppressors decreased levels of the replicative helicase, DnaC. I showed that decreased levels of helicase are sufficient to limit replication initiation under fast growth conditions. I also explored the regulation of DnaA as a transcription factor. The replication initiation inhibitor, YabA, binds to DnaA and prevents its cooperative binding at the origin. In addition to its role in replication initiation, DnaA also directly regulates expression of several genes. YabA has been shown to inhibit DnaA binding at
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The Initiation of DNA Replication - Dan S Ray - 2012-12-02
The Initiation of DNA Replication contains the proceedings of the 1981 ICN-UCLA Symposia on Structure and DNA-Protein Interactions of Replication Origins, held in Salt Lake City, Utah on March 8-13, 1981. The papers explore the initiation of DNA replication and address relevant topics such as whether there are specific protein recognition sites within an origin; how many proteins interact at an origin and whether they interact in a specific temporal sequence; or whether origins can be subdivided into distinct functional domains. The specific how they are catalyzed are also discussed. This book is organized into six sections and comprised of 41 chapters. The discussion begins by analyzing the replication origin region of the Escherichia coli chromosome and the precise location of the region carrying autonomous replicating function. A genetic map of the replication and incompatibility regions of the resistance plasmids R100 and R1 is described, and several gene products produced in vivo or in vitro from the replication region are considered. The sections that follow focus on the DNA initiation determinants of bacteriophage M13 and of chimeric derivatives carrying foreign replication determinants; suppressor loci in E. coli; and enzymes and proteins involved in initiation of phage and bacterial chromosomes. The final chapters examine the origins of eukaryotic replication. This book will be of interest to scientists, students, and researchers in fields ranging from microbiology and
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**Roles of Chromatin and Nuclear Structure in Regulation of DNA Replication by Xenopus Egg Extract** - Zhi Hong Lu - 1999
The study of genome duplication has been largely hindered by the lack of appropriate monitoring techniques, and any comprehensive understanding ultimately requires quantitative approach. In this master’s thesis, we analyzed replication patterns in mouse somatic and embryonic stem cells (mESCs) with newly developed three-dimensional structured illumination microscopy (3D-SIM) to register the progression of S-phase in more detail than previously described. We successfully established an automated workflow to produce reliable and reproducible replication foci (RF) counts in C2C12 cells from 3DSIM data and TANGO (Tools for Analysis of Nuclear Genome Organization). Such an approach has not been described before, and could be used to evaluate further cell types and schemes. We observed significant differences in replication timing and progression between somatic (C2C12, C127) and mESCs (HI5). In this report we show that in mESCs S-phase lasts significantly longer (15 h), with a
Epigenetic regulation of DNA replication compared to somatic cells. Furthermore, differentiated HI5 female mESCs into epiblast-like cells (EpiLCs) exhibit inactive X chromosome and differential replication timing of Xi within two distinct EpiLC populations, and a much shorter S-phase (10 h). As a final aim of this work, we interfered with specific histone modifications with inhibitors and knockout cell lines. Inhibition of EZH2 methyltransferase resulted in global reduction of H3k27me3 levels in both somatic and mESCs, however replication dynamics were not affected. In contrast to somatic cells, viability of mESCs in presence of inhibitor was greatly reduced, suggesting a more important role of H3K27me3 in mESCs. Suv39H1/H2 double knockout mESCs had no observable effect on replication dynamics or proliferation. Moreover, differentiation of these cells into EpiLCs resulted in a distinct S-phase progression, with replication resembling HI5 EpiLCs.
like cells (EpiLCs) exhibit inactive X chromosome approach. In this master’s thesis, we analyzed replication patterns in mouse somatic and embryonic stem cells (mESCs) with newly developed three-dimensional structured illumination microscopy (3D-SIM) to register the progression of S-phase in more detail than previously described. We successfully established an automated workflow to produce reliable and reproducible replication foci (RF) counts in C2C12 cells from 3DSIM data and TANGO (Tools for Analysis of Nuclear Genome Organization). Such an approach has not been described before, and could be used to evaluate further cell types and schemes. We observed significant differences in replication timing and progression between somatic (C2C12, C127) and mESCs (HI5). In this report we show that in mESCs S-phase lasts significantly longer (15 h), with a ‘leaky’ chromocenter replication profile compared to somatic cells. Furthermore, differentiated HI5 female mESCs into epiblast- and differential replication timing of Xi within two distinct EpiLC populations, and a much shorter S-phase (10 h). As a final aim of this work, we interfered with specific histone modifications with inhibitors and knockout cell lines. Inhibition of EZH2 methyltransferase resulted in global reduction of H3k27me3 levels in both somatic and mESCs, however replication dynamics were not affected. In contrast to somatic cells, viability of mESCs in presence of inhibitor was greatly reduced, suggesting a more important role of H3K27me3 in mESCs. Suv39H1/H2 double knockout mESCs had no observable effect on replication dynamics or proliferation. Moreover, differentiation of these cells into EpiLCs resulted in a distinct S-phase progression, with replication resembling HI5 EpiLCs.