Mitosis is the process of one cell dividing into two daughters, such that each inherits a single and complete copy of the genome of their mother. This is achieved through the equal segregation of the sister chromatids between the daughter cells. However, beyond this simple principle, the partitioning of other cellular components between daughter cells appears to follow a large variety of patterns. We discuss here how the organization of the nuclear envelope during mitosis influences cell division and, subsequently, cellular identity.

Introduction

Eukaryotic chromosomes are surrounded by a double membrane (consisting of an inner and outer nuclear membrane) called the nuclear envelope. Depending on the organism and cell type, the nuclear envelope either remains intact or disassembles before chromosome segregation; these forms of mitosis are termed closed and open division, respectively. Closed mitosis is considered to be the most ancient mechanism of eukaryotic cell division, whereas open mitosis appears to have been invented several times during evolution. Animals and plants, for example, are related more distantly to each other than to fungi. Nevertheless, both undergo open mitosis, whereas most fungi retain an intact nuclear envelope during division.

In addition to the symmetric distribution of chromosomes between sister cells, cell division often also involves the asymmetric segregation of cellular components. In some cases, this asymmetry relies on the presence and organization of the nuclear envelope. During metazoan embryogenesis, asymmetric cell divisions diversify a pool of precursor cells into different cell types. In adult organisms, asymmetric divisions constantly regenerate various tissues. Indeed, stem cells are generally thought to divide asymmetrically during both embryonic development and adult tissue homeostasis. They maintain an unlimited division potential, whereas their differentiating progeny only divide a few times before undergoing terminal differentiation. Similarly to differentiating cells, individual budding yeast cells can only produce a limited number of direct progeny. During the budding yeast cell cycle, a smaller daughter cell grows from a larger mother cell. This daughter cell survives its mother and expresses its own transcriptional programs. Consequently, daughter cells represent an eternal lineage in a population of budding yeast cells.

Keywords: closed mitosis, open mitosis, compartmentalization, nuclear envelope, asymmetric cell division

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One of the mechanisms through which eternal and differentiating or aging lin-
erages differ from each other involves the asymmetric segregation of factors defining
these fates. Depending on whether these factors are fixed or freely diffusing, differ-
ent mechanisms ensure the asymmetry of their inheritance. If cell identity factors are
immobilized on a structure, asymmetric inheritance is achieved by segmenting
this structure into only one of the two sister cells. Cell identity factors diffusing
freely in two-dimensional membranes or three-dimensional liquid phases can be
segregated asymptomatically only if their exchange between the future sister-cells is
restricted in some way. Budding yeast cells, for example, take advantage of their
closed mitosis to extensively compartmentalize their nuclear during division. This
compartmentalization is used to asym-
metrically segregate cellular components
such as the transcription factor Ace2 and non-chromosomal DNA.13-15

The daughter-specific transcription
factor Ace2 (activator of CUP1 expres-
sion) diffuses freely in the budding yeast nuclear but strongly accumulates in the
daughter half of the dividing nucleus.13,14

The asymmetry of Ace2 localization is
established at the end of anaphase by a network of kinases and phosphatases
that differentially regulate its nuclear import and export in the two halves of the
nucleus.15-15 Importantly, initiating and maintaining or increasing this asym-
metry requires the compartmentalization of the nucleoplasm between the future
mother and daughter parts of the divid-
ing nucleus.13 This compartmentalization is
ensured by the dumbbell-morphology of the late anaphase nucleus. Mutants with
a wider and/or shorter bridge exchange
Ace2 and other nucloplasmic molecules
more rapidly between the two halves of the
nucleus, resulting in a more homogenous
distribution of Ace2 between mother and
bud. Hence, the presence of the nuclear
envelope during mitosis is essential for the
asymmetry of Ace2 segregation.

Ace2 homologs are present in the
genomes of many fungi.16 In Saccharomyces
cerevisiae, Schizosaccharomyces pombe, Schizosaccharomyces japonicus, Candida
albicans and Candida glabrata, Ace2
induces the transcription of genes
encoding cell wall degrading enzymes
genes involved in sister cell separation
after mitosis.17-20 During the yeast-like
growth phase of C. albicans, Ace2 accumu-
lates in the daughter nucleus of large
budding cells, similarly to its budding
yeast homolog.7 The C. albicans nucleus
also adopts a dumbbell-like shape during
division, probably providing the basis for
the maintenance of Ace2 asymmetry by
nucleoplasmic compartmentalization.
In contrast, S. pombe has a dumbbell shaped
nucleus but distributes Ace2 symmetric-
cally.21 Ace2 being not the sole nuclear
factor to segregate asymptomatically in bud-
ing yeast (see below), the dumbbell mor-
phology of the fusion yeast nucleus might
in this case help to maintain other asym-
metries than that of Ace2.

During budding yeast mitosis, nuclear
compartmentalization also promotes the
retention of acentromeric DNA in the
mother cell.11,13 The accumulation of these
acentromeric episomes in turn contrib-
utes to aging of the yeast mother cell as it
divides.21,24 Extrachromosomal rDNA
circles (ERCs) are naturally occurring
episomes that are excised from the rDNA
locus by homologous recombination.22,23

Aging yeast mother cells accumulate large
numbers of ERCs, since ERCs are retained
in the mother nucleus, where they prolif-
erate through replication.

Several non-exclusive mechanisms
converge to ensure the retention of epis-
omes in budding yeast mother cells (as
discussed by Ouellet and Barral25). The
asymmetry of episomes diffusing freely
in the nucleoplasm strongly depends on the
geometrical constraints of the divid-
ing nuclear envelope and the time spent
in nuclear division, as both parameters
determine to which extent episomes can
equilibrate between mother and daugh-
ter parts of the nucleus before division.26

The morphology of the dividing nucleus
and the time needed for nuclear divi-
sion set a basal retention frequency for
individual plasmids to about 85% per
division. The retention frequencies of a
second class of episomes, which also
includes ERCs, is much higher, with
about 96% to over 99% retained in the
mother cell.22-25 In addition to nuclear
geometry and timing, the high reten-
tion frequencies of these episomes also
depend on the compartmentalization of
the nuclear envelope. Highly retained
acentromeric episomes are tethered to
the nuclear envelope and a diffusion bar-
rrier in the ONM restricts their separa-
tion to the daughter part of the nucleus.12
Failure to attach to the nuclear periphery
and mutations weakening the diffusion
barrier in the ONM decrease the asym-
metry of their segregation to levels similar
to episomes diffusing freely in the nucleo-
plasm.12,27 Consequently, it has been pro-
posed that the diffusion barrier in the
ONM is required for high episode reten-
tion frequencies in budding yeast cells.26

In S. pombe, acentromeric self-replic-
cating episomes are also asymmetrically
segregated to one of the daughter cells.28
Furthermore, ERCs have been isolated
from fusion yeast.29,30 If integrated into a
bacterial vector backbone, fusion yeast
ERC sequences are able to promote plas-
mid replication in vivo, suggesting ERCs
are able to self-amplify in fusion yeast.30

How episomes are segregated asymmetry-
cally in fusion yeast is still unclear. The
comparison to budding yeast, however,
strongly suggests that nuclear compart-
mentalization may contribute to the
asymmetry of episode inheritance in both
budding and fusion yeast.

**Chromosome Segregation during Closed Mitosis**

In addition to the segregation of individual proteins and relatively small acentromeric
episomes, the nuclear envelope has also
been implicated in chromosome segrega-
tion during closed mitosis. In organisms
undergoing both closed and open mitoses,
the mitotic spindle generally segregates
chromatids during mitosis. However,
when cells undergo closed mitosis in the
absence of nuclear spindle microtubules,
contacts between the nuclear envelope and
chromatin can also promote the segrega-
tion of the chromosomes.

Certain dinoflagellates, for example
divide without disassembling their nuclear
envelope and without assembling an intra-
nuclear spindle (Fig. 1).31,32 Chromosome
segregation in these organisms involves
the interaction of chromosomes with the
nuclear envelope, which is in con-
tact with ordered arrays of cytoplasmic
microtubules. These microtubules transverse the dividing nucleus in parallel while they are wrapped by pipes of nuclear envelope. Chromosomes intimately contact the nuclear envelope surrounding the individual microtubule bundles and appear to slide along the membrane, resulting in their segregation into two future nuclei.

Such intimate chromatin-nuclear envelope contacts might also be present in *S. pombe*, since these cells are also able to divide their nuclei in the absence of a mitotic spindle in a process termed nuclear fission. The maturation of spindle pole bodies (SPBs) and the separation of sister chromatids are two key factors driving nuclear fission. In the absence of SPB-associated proteins linking chromatin to the nuclear periphery, nuclear fission is impaired. It seems that, in fission yeast, the interaction between chromatin and the nuclear envelope is not only passively maintained during nuclear division but also actively shapes the dividing nucleus and drives karyofission. Supporting this idea, inhibiting chromosome segregation in presence of an elongating anaphase spindle interferes with nuclear division and changes nuclear shape. Upon uncoupling of the SPB from the pole of the elongating spindle, chromatin masses are not separated and remain in the center of the nucleus. At the same time, the growing spindle microtubules push the nuclear envelope and assembles an intranuclear spindle before breaking the nuclear envelope open at a later time point. In contrast to the true open mitosis tip, leading to a fenestration of the nuclear envelope, however, does not disassemble once the nucleus opens at its tip, but remains present throughout mitosis. In contrast to fungi, metazoan cells fully disassemble their nuclear envelope at some point during cell division. The timing of nuclear envelope disassembly varies significantly between organisms and cells types. In addition, some fungi undergo only a partially closed mitosis, starting with an intact nuclear envelope and assembling an intranuclear spindle before breaking the nuclear envelope open at a later time point. In contrast to the true open mitosis of metazoans, the nuclear envelope does not disperse completely in these cases; instead it ruptures and/or fenestrates without disappearing (Fig. 2). A closer look at the spectrum cells explore between spindle breakdown and karyofission. Thereby chromatin-nuclear envelope contacts could contribute to the faithful segregation of the chromosomes between mother and daughter nuclei.

**Anaphase with or without a Nuclear Envelope**

In contrast to fungi, metazoan cells fully disassemble their nuclear envelope at some point during cell division. The timing of nuclear envelope disassembly varies significantly between organisms and cells types. In addition, some fungi undergo only a partially closed mitosis, starting with an intact nuclear envelope and assembling an intranuclear spindle before breaking the nuclear envelope open at a later time point. In contrast to the true open mitosis of metazoans, the nuclear envelope does not disperse completely in these cases; instead it ruptures and/or fenestrates without disappearing (Fig. 2). A closer look at the spectrum cells explore between spindle breakdown and karyofission. Thereby chromatin-nuclear envelope contacts could contribute to the faithful segregation of the chromosomes between mother and daughter nuclei.

**Figure 1. Late anaphase/telophase during closed mitosis without nuclearplasmic spindle microtubules or SPB-microtubule contacts.**

(A) Chromosomes (dark blue) of the dinoflagellate *G. cohnii* are separated as they slide along NE tubes, which emerap microtubule bundles penetrating in the nuclearplasm (light blue). (B) Nuclei of the fission yeast *S. pombe* dividing with or without microtubules or with microtubules attached to the SPB.
partitioning of associated factors and structures despite the loss of nucleo-cytoplasmic compartmentalization.

Despite the different mechanisms with which the nuclear envelope opens, the switch from closed to (semi-)open mitosis takes place later as cells start chromosome segregation. These observations suggest that during closed mitosis, anaphase is a critical point for nuclear envelope integrity. Indeed, during closed mitosis, the most dramatic rearrangements of nuclear shape take place in anaphase, when the spherical metaphase nucleus transforms into an elongated dumbbell shape. Since this represents a process during which the original sphere is transformed into two, the nuclear surface must increase by a third in order to maintain a constant nuclear volume. This increase in nuclear envelope surface is observed during anaphase in *S. cerevisiae* and *S. pombe*, whereas it is absent in *S. japonicus*. Consequently, this fungus may potentially use attachment to NPCs to partition nuclear components between the two daughters. The nuclei of animal and plant cells are even larger than those of *S. japonicus*. Consequently, one reason why they undergo nuclear envelope disassembly may be to avoid having to provide large amounts of new membrane material prior to nuclear envelope reassembly.

In plant cells and human cell lines, the nuclear envelope breaks down during pro-metaphase, whereas in young sea urchin embryos, nuclear envelope disassembly occurs after metaphase. Similarly, nuclear envelope-like structures surrounding mitotic spindle fibers have been observed in insects. During early embryonic and neuroblast divisions of *Drosophila melanogaster*, the nuclear envelope still surrounds chromatin during metaphase. In early *C. elegans* embryogenesis, transmembrane proteins of the INM are detectable at the periphery of chromatin as late as early anaphase. Chromosome condensation and the attachment of chromosomes to spindle microtubules can occur within a nuclear envelope-like structure, whereas anaphase is completed only after nuclear envelope disassembly. This suggests that, in animal cells, progression through anaphase requires complete removal of the nuclear envelope and its resorption into the ER. If any asymmetry would still be provided by the original structure of the nucleus, its propagation through mitosis would now rely on the ER being compartmentalized. Although this is the case in yeast, compartmentalization of the ER has not yet been reported for metazoans.

The second prediction from the observations in *S. japonicus* is that animal cells are unable to increase their nuclear surface during mitosis. We did not find any data precisely measuring nuclear envelope surface in animal cells before and after mitosis. However, a study by Anderson and colleagues suggests that the amount of membrane material available for nuclear envelope formation in human cells is limited. At the end of mitosis, the nuclear envelope is reformed through contacts between ER membrane tubules and decondensing chromatin. This requires the transformation of highly curved ER membrane tubules into flat membrane sheets to form the nuclear envelope. Overexpression of proteins inducing membrane curvature in the ER causes reduced increases in nuclear size after mitosis. Consequently, mechanisms stabilizing the organization of the ER could counteract the incorporation of membrane material into the nuclear envelope and thereby limit the amount available.

To date we have only little knowledge about the factors driving the evolution of mitosis in eukaryotes. Therefore, we can only speculate about why eukaryotes developed a range of mechanisms to progress through the cell cycle. However, even the limited knowledge currently available indicates that the cell biology of nuclear division has clear consequences for segregation of nuclear factors as well as for anaphase progression. Future studies on the variations between open and closed mitosis will allow a more detailed understanding of the relationship between nuclear geometry, mitotic progression and asymmetric cell fate, as well as the forces that have driven the evolution of these diverse mechanisms of mitosis. However, at this point, several parameters appear to play key roles: the possibility for the nuclear envelope to control the symmetric or asymmetric segregation of nuclear components as well as the size of the nucleus. We propose that the interplay between these parameters determines which mitotic mechanisms are chosen and that these mechanisms, in turn, contribute to evolution.

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**Figure 2. Different fates of the nuclear envelope during anaphase:** “intact” (red) during closed mitosis in *S. cerevisiae* separating nucleoplasm (light red) and cytoplasm (light blue), “fenestrated” (orange) during semi-open mitosis in *S. japonicus* and *G. majus*, allowing nucleoplasm and cytoplasm to mix (purple) and “completely disassembled,” incorporated into the ER (green) during open mitosis in human cell lines.
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References

1. Cavalier-Smith T. Origin of the cell nucleus, mitosis and mitotic roles of intracellular coalescence. Red Dross 2010; 17-5. PMID:21029544. http://dx.doi.org/10.1038/nature09997.

2. Balasubramanian B, Raghothama K, Sankararaman S, et al. Phylogenetic analyses support the hypothesis of Eucarya and root relationships among eukaryotes: “supergroups”. Proc Natl Acad Sci U S A 2008; 105:3699-69. PMID:18382750. http://dx.doi.org/10.1073/pnas.0707865105.

3. Martineau RR, Johnson LR. Life span of individual yeast cells. Nature 1993; 183:1753-7. PMID:8548761. http://dx.doi.org/10.1038/367722a0.

4. Harrell LH, Unger ME. Unequal division in Saccharomyces cerevisiae and its implications for the control of cell division. J Cell Biol 1977; 79:442-5. PMID:908047.

5. Kennedy RE, Stromer NR, Jr, Giurazza I, Daugherty cells of Saccharomyces cerevisae from old mother display a reduced life span. J Cell Biol 1994; 124:1059-69. PMID:8008765.

6. Bubke N, Jarren RP, Shin TH, Nonn A. Genetic asymmetric accumulation of Adl2p in pantoaean postmeiotic studies depends on a mutation and restricts yeast mitotype switching to mother cells. Cell 1996; 84:419-29. PMID:8842988. http://dx.doi.org/10.1016/S0092-8674(00)81049-1.

7. Culotta V, Davis RW, Craig EA, Mwl; actin subcellular distributions and genetic programs to induce asymmetric cell division. J Cell Biol 2001; 151:705-16. PMID:11449126. http://dx.doi.org/10.1083/jcb.200006106.

8. Sila H, Herkert J. Identification of asymmetrically localized subproteins, Adl2p, required for image-specific transcription of the 39A2P gene. Cell 1996, 84:755-6. PMID:8628609. http://dx.doi.org/10.1016/S0092-8674(00)81049-1.

9. Broach R, Marques-Sarate T, Davis RW, et al. Nuclear envelope division during cell cycle of the primitive eukaryote dinoflagellate. Proc Natl Acad Sci U S A 2008; 9:646-53. PMID:17486116. http://dx.doi.org/10.1073/pnas.0707865105.

10. Huang J, Biele EL, Villain J, Gigi SF, Ann M, et al. Division in the dinoflagellate Gymnodinium cohnii: a new type of nuclear reproduction. J Cell Biol 2006; 170:287-98. PMID:16605333. http://dx.doi.org/10.1083/jcb.200601002.

11. Michael S, Sulak BE, Xue J, Park K, et al. Mitotic nuclear morphology and cell cycle of the primitive eukaryote dinoflagellate Gymnodinium specious. Proc Natl Acad Sci U S A 2008; 105:12076-8. PMID:18762572. http://dx.doi.org/10.1073/pnas.0807880106.

12. Gabbia LR, Nagai S, Shimada K, Motie F, Taddei A, Gauer SM. Nuclear geometry and rapid division. Cell 2011; 145:3-4. PMID:2194955. http://dx.doi.org/10.1016/j.cell.2011.06.033.

13. Manabe E, Alexander J, Ye JH, Chromyova P, Edgell DL, et al. The NIDOLIS family of CRM1-directed transcriptional accessory proteins. Mol Biol Cell 2012; 23:3569-82. PMID:22615052. http://dx.doi.org/10.1091/mbc.E11-12-1179.

14. Baiker A, Garcia M, Mikes I, Szabo-S, Vajna de Almada CR, Spiess M. Conserved regulators of the cell separation process in fission yeast. Fungal Genet Biol 2012; 49:235-49. PMID:22349816. http://dx.doi.org/10.1016/j.fgb.2012.05.005.

15. Kelly MT, MacCannell DM, Clency SD, OkI P, Brown AP, Barer G. The C. elegans Cbk1 and Cbk2 genes: morphogenetic, adhesion and neurite. Mol Biol Cell 2004; 15:565-90. PMID:15258946. http://dx.doi.org/10.1091/mbc.E03-07-0545.

16. Prent C, Li, others, 670. PMID:17826800. http://dx.doi.org/10.1038/jcb.2005047.

17. Stead DA, Walker J, Holmbacka L, Gilks SYR, V. Selby L, et al. Impact of the transcriptional regulator, Ascl, on the C. elegans germline mitotic division. 2010; 10:212-15. PMID:19943077.

18. Simpson AGB, et al. Phylogenomic analyses support the mitochondrial genome of the fission yeast Schizosaccharomyces pombe. Proc Natl Acad Sci U S A 2009; 106:3859-64. PMID:19237557. http://dx.doi.org/10.1073/pnas.0807880106.

19. Vázquez de Aldana CR, del Rey F. The endosomes of Schizosaccharomyces pombe. J Cell Biol 2008; 182:897-910. PMID:18762579. http://dx.doi.org/10.1083/jcb.200801015.

20. Gilchrist JD. The Diversity of Eukaryotes. Am Nat 2010; 176:5731-42. PMID:16317047. http://dx.doi.org/10.1086/524680.

21. Boeck JD. The molecular basis of nuclear shape in fission yeast. PLoS Biol 2007; 5:e170; PMID:17579515. http://dx.doi.org/10.1371/journal.pbio.0050170.

22. Grue BS, Park K, Mitchell P, Mcginley JR, harvJ: a gene required for cell-cycle-dependent spindle pole body anchoring in the nuclear envelope and spindle formation. Mol Biol Cell 2011; 22:10634-51. PMID:21825530. http://dx.doi.org/10.1091/mbc.E11-04-0312.

23. Doherty SR, Biker A, Anker JK, Bernard D, Amory C, et al. synaptic complex mediates asymmetric spore pole body morphogenesis and restricts Msp2p. Mol Biol Cell 2011; 22:10634-51. PMID:21825530. http://dx.doi.org/10.1091/mbc.E11-04-0312.

24. Shimizu T, Tashiro K, Henci KL, Kishpelk A, Oliferenko S. The spindle pole body facilitates nuclear envelope division during cell cycle progression. PLoS ONE 2007; 2: e13052. PMID:17979515. http://dx.doi.org/10.1371/journal.pone.0001305.

25. Fong M, Citi A, Dixit GP, Liang CL, et al. Microtubule remodeling in the fission yeast Schizosaccharomyces pombe. Proc Natl Acad Sci U S A 2009; 106:3859-64. PMID:19237557. http://dx.doi.org/10.1073/pnas.0807880106.

26. King MC, Lask CB, Biboiah KG. Synaptonema-mediated import of integral inner nuclear membrane proteins. Nature 2006; 442:810-13. PMID:16929305. http://dx.doi.org/10.1038/nature05075.

27. Rodriguez-Otero A, RC, Poirier-Ortín J-E. SBRG, an interacting complex that is required for cell cycle progression. Yeast 2012; 19:45-54. PMID:21774482. http://dx.doi.org/10.1002/yea.2509.

28. Huang J, Biele EL, Ville J, Gigi SF, Ann M, et al. Hologenomic recombination of the centromere ensures optimal meiosis. PLoS Genet 2011; 7:1002015. PMID:21825530. http://dx.doi.org/10.1371/journal.pgen.1002015.

29. Khadka K, Shykap S, Jigchi S, et al. cut11 such as a new gene with a novel function in yeast. Nature 2004; 441:567-70. PMID:15387972. http://dx.doi.org/10.1038/nature04987.

30. Grand SE, Fustes P, Calaf G, et al. Cut11 such as a new gene with a novel function in yeast. Nature 2004; 441:567-70. PMID:15387972. http://dx.doi.org/10.1038/nature04987.
44. Yan C, He Y, Zhang D, Chiou KH, Ohnishi S. Distinct strategies for controlling the nuclear envelope satisfy geometric constraints during nuclear division. Cell Biol 2011; 21:3160-3169; PMID:21624224; http://dx.doi.org/10.1083/jcb.201106.052.

45. Ando K, Hongo M, Funae K, Yato M, Takanai T, Osmani M, et al. Breakdown of the nuclear envelope by an extending mitotic nucleus occurs during amitosis in Schizosaccharomyces pombe. Genes Cells 2011; 16:911-20; PMID:21753694; http://dx.doi.org/10.1111/j.1365-2443.2011.01949.x.

46. Strader A, Wiber I, Szabad G. A novel mechanism of nuclear envelope break-down in a fungus: nuclear migration strips off the envelope. EMBO J 2005; 24:1074-85; PMID:15846140; http://dx.doi.org/10.1038/sj.emboj.7600644.

47. Tho宴 U, Stroka A, Szabad G. Dynamic rearrangement of nucleoporins during fungal "open" mitosis. Mol Biol Cell 2008; 19:1230-40; PMID:18172026; http://dx.doi.org/10.1091/mbc.E07-02-0130.

48. Gonzalez J, Muthuk K, Cheng J, Turi Y, Pabst NN, inertia S. Nuclear shape, growth and integrity in the closed mitosis of fission yeast depend on the Rts1-GTPase system, the spindle pole body and the endoplasmic reticulum. J Cell Sci 2009; 122:2464-72; PMID:19775705; http://dx.doi.org/10.1242/jcb.048199.

49. Liu HWP, Hahn G, Turi Y, Heinzel A, Miller JA, Jaster V. Yeast-like Biomechanics Governs Important Aspects of Nuclear Geometry in Fission Yeast. Science 2007; 317:940; http://dx.doi.org/10.1126/science.1145486.

50. Wray M, Inza D, Gillingham T Jr, Martensson DSN. Nuclear pore complex number and distribution throughout the Saccharomyces cerevisiae cell cycle by three-dimensional reconstruction from electron micrographs of nuclear envelopes. Mol Biol Cell 1997; 8:2119-32; PMID:9362057.

51. Ingrouillon P, Edgerson NP, Schneider RL, Rupes L, Tyeys M, Fuchtbauer E. The size of the nuclear envelope in yeast cells grows. Mol Biol Cell 2007; 18:3925-30; PMID:17938521; http://dx.doi.org/10.1091/mbc.E07-02-0130.

52. Normann HR, Tan Y, Yarar D, Giddings TH Jr., Mastronarde DN, Martinez-Fernandez E, et al. Orchestrating nuclear envelope disassembly and reassembly during mitosis in fission yeast. J Cell Biol 2007; 179:593-600; PMID:17938401; http://dx.doi.org/10.1083/jcb.200702074.

53. Gareau S, Lansell A, Kanzy U. Orchestrating nuclear envelope disassembly and reassembly during mitosis. Nat Rev Mol Cell Biol 2007; 8:2119-32; PMID:17938401; http://dx.doi.org/10.1083/jcb.200702074.

54. Rose A. Open mitosis: nuclear envelope dynamics. Cell Division Control in Flies 2008; 207-30.

55. Leinert P, Baber G, Dugle N, Hard AR, Tanasaki M, Ellingsen I. Nuclear envelope breakdown in starfish eggs occurs preceded by partial NPC disassembly followed by a rapidly spreading fenestration of nuclear membranes. J Cell Biol 2005; 169:1095-108; PMID:16265092; http://dx.doi.org/10.1083/jcb.200501107.

56. Tanasaki M. Dynamics of the endoplasmic reticulum and pellicle appearance during early sea urchin development. Mol Biol Cell 2000; 11:897-914; PMID:10712508.

57. Kanamori K, Kanawee KE, Daigle N, Hand AR, Terasaki M, Ellingsen I. Nuclear envelope breakdown in starfish eggs proceeds by partial NPC disassembly followed by a rapidly spreading fenestration of nuclear membranes. J Cell Biol 2005; 169:1095-108; PMID:16265092; http://dx.doi.org/10.1083/jcb.200501107.

58. Wagner N, Kagemann B, Lauter S, Kühne G. The Drosophila melanogaster LEM-domain protein MAN1. Eur J Cell Biol 2006; 85:16-25; PMID:16419904; http://dx.doi.org/10.1016/j.ejcb.2005.10.002.

59. Hazai A, Zlokovic E, Naimark-Epstein S, Fuenteis N, Faber PA, Gruenbaum Y. Persistence of major nuclear envelope antigens in an envelope-like structure during mitosis in Drosophila melanogaster embryos. J Cell Biol 1985; 99:665-70; PMID:2517242.

60. Lee KK, Gruenbaum Y, Spahn T, Liu J, Wilson KL. C. elegans nuclear envelope proteins, MAN1, lamin, and nucleoporins reveal unique timing of nuclear envelope breakdown during mitosis. Mol Cell Biol 2006; 26:1689-99; PMID:16902632.

61. Lee KK, Stan D, Cobet M, Liu J, Wilson KL, et al. Lamin-dependent localization of UNC-84, a protein required for nuclear migration in Caenorhabditis elegans. Mol Cell Biol 2002; 13:902-910; PMID:11907270; http://dx.doi.org/10.1091/mbc.01-06-0294.

62. Anderson DJ, Heuser MJ. Redocking of the endoplasmic reticulum limits the size for nuclear envelope formation. J Cell Biol 2008; 180:911-24; PMID:18779370; http://dx.doi.org/10.1083/jcb.200805146.

63. Anderson DJ, Heuser MJ. Nuclear envelope formation by chromatin-mediated reorganization of the endoplasmic reticulum. Nat Cell Biol 2007; 9:1560-1566; PMID:17882649; http://dx.doi.org/10.1038/ncll0630.

64. Amstislav W, Ellingsen I, Stedli D. Nuclear pore complex assembly through the cell cycle: regulation and membrane organization. FEBS Lett 2008; 582:2064-2069; PMID:18520825; http://dx.doi.org/10.1016/j.febslet.2008.02.047.