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Unveiling the Dynamics: A Comprehensive Review of Biological Membrane Kinetics During Cell Division

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ABSTRACT

A cell division is a series of events that occur tandemly during cell growth. Most of the cell cycle is involved in the phase known as interphase, during which it matures, copies its chromosomes and prepares to divide. Following interphase, the cell completes mitosis and divides to complete offspring lineage. As a result, each of the daughter cells that form enters its interphase to begin a new cycle of the cell. During the cell division, eukaryotic cells undergo complicated and well-coordinated changes to their membranes and cytoskeleton. Organelles such as the nuclear envelope, endoplasmic reticulum and Golgi apparatus must be destroyed or altered, distributed and then rebuilt to complete the cell cycle. The precise distribution of cytoplasmic material and an appropriate number of organelles was also required between daughter cells. As cell division progresses, the plasma membrane that covers this mixture of cytoplasm and organelles is prone to alter. Resident lipids in these distinct membrane compartments exert their considerable influence on the division process. The cell splits into two daughter cells along with the two copies of the genetic material. During cell division, cell-cycle proteins have a role in both the regulation and maintenance of the cell cycle in eukaryotic cells. They regulate transitions between interphase, mitosis and cytokinesis, the three phases of the cell cycle that result in cell division and replication. Therefore, cells have developed sophisticated organelle inheritance mechanisms to permit efficient intracellular organelle sharing. To completely understand how the remodeling of various organelles is interconnected and how changes in organelle shape and function affect the process of cell division, it will be obvious to combine this information in the current context. The study aimed to review and synthesize current knowledge on the kinetics of biological membranes during cell division.

KEYWORDS

Biological membrane, cell division, cell organelle, mitosis, meiosis

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INTRODUCTION

All living organisms are made up of cells, which are the smallest units capable of independent life. Cell division is a dynamic process in which the parent cell is divided into daughter cells and the cellular components of the parent cell duplicate properly[1]. All eukaryotes share common unicellular predecessors and many parts of the cell cycle mechanisms are conserved[2]. Multicellularity requires the emergence of mechanisms that coordinate cell growth and proliferation across growing organs with the adoption of specialized cell fates[3]. There are two different types of cell division, the first one being vegetative division known as mitosis, in which each daughter cell replicates the parent cell. Meiosis is the second one to generate four haploid daughter cells which are also known as reductional division. Prophase is the first step of mitosis in which rearrangement of microtubules occurs and the DNA molecules are condensed gradually to form chromosomes[4]. The disassembly of the nuclear envelope is the feature of prometaphase and the alignment of chromosomes in the metaphase plate is observed during the metaphase stage in which the kinetochores are attached to the chromosome arms and are connected to opposite poles of the spindle fiber through the microtubules[5]. After completion of the metaphase stage when the spindle assembly checkpoint arrived, segregation of chromatids started in the anaphase stage[6]. Chromosomes are separated from each other and move towards the opposite pole. In telophase, chromosome segregation is mediated by the mitotic spindle[7]. In cytokinesis, the midbody is a structure formed in mammalian cells by the condensation of mitotic spindles[8]. According to Mierzwa and Gerlich[9], this process is called cytokinetic abscission where two daughter cells are separated by cleaving of the midbody. Cell division is completed by cytokinetic abscission, which results in the physical cutting of the intercellular bridge (ICB) that connects the daughter cells[10]. At the time of division, to accurately segregate the genetic material from the parent to the daughter cells, a high level of both spatial and temporal control is needed inside the cell.

After completion of the cell division, the newly formed daughter cells have to remain functionally active as they need to process the nutrients for pursuing their metabolism so they can produce proteins, DNA and other components as per their requirements for their survival and growth. Most of the cellular organelles are newly modified during the cell division procedure. In daughter cells, *de novo* synthesis of the cell organelles is not possible due to the requirement of time and resources and it also causes inequality of organelles in the daughter cells[11]. The division of cell organelles is a complex issue for a dividing cell as there are so many criteria like size, numbers, shape and locations. The organelles are also following different methods for division. Some of them are segregated into pieces to the cytoplasm and then reaggregate after the division of the nuclear membrane and Golgi bodies[12]. Some organelles like mitochondria may change their external structure and retain some of their stochastic inheritance but they never segregate and reaggregate[12]. Not even all the time the partitioning is correct, in some cases, such issues need to be solved by different methods and those are also different from organelle to organelle[13]. Therefore, it is clear that organelles require their mechanisms to maintain their characteristics. A number of proteins are involved in both cell division and organelle biology. Membrane-bound organelles contain the proteins that are recognized by the midbody for proteomic analyses and mitotic failure screened by RNA interference[14-19]. Scientists made a whole catalog for different types of proteins of the membrane compartment that have marked effects in the cell division process and also in organelle biology. Several proteins in this catalog are known to scientists but they are not deeply studied in this cell division context and some of the proteins are very less known[20-23]. In this review, several examples are taken from different organisms mainly highlighting the mammalian cell division steps to lipid dynamics. The specific objectives of this review are to review and synthesize current knowledge on the kinetics of biological membranes during cell division, focusing on the role of lipid composition and membrane compartmentalization; explore the mechanisms underlying the remodeling, distribution and inheritance of organelles during cell division, emphasizing their interconnection with membrane dynamics and analyze the regulatory roles of

cell-cycle proteins in coordinating membrane and cytoskeletal changes throughout the phases of cell division, including interphase, mitosis and cytokinesis. Here, the key mechanisms and processes that regulate membrane kinetics during cell division, such as membrane trafficking, lipid and protein rearrangements and the involvement of cytoskeletal elements are highlighted. In essence, the review would aim to provide a comprehensive summary of how biological membranes are involved in and affected by cell division, integrating existing research and identifying directions for future work.

How do lipids affect the division of cells? Lipids are essential for cell function in processes like organ morphogenesis, cell differentiation and organ proliferation, all of which are closely related to cell cycle development[24]. Lipids are involved in numerous biological activities and makeup a major portion of membranes. Dramatic changes in cell structure trigger cell division. The cytoskeleton and lipid remodeling, as well as local protein complex recruitment and membrane trafficking, are required for each phase of cell division[25]. Except for sterols, lipids are significantly smaller than proteins and typically consist of a polar head group facing the membrane exterior and one or more hydrophobic carbon side chains facing the membrane lipid bilayer interior[26]. As per head membrane protein, nearly 50-100 of them are present in the cellular context[27]. The length and saturation of hydrocarbon side chains might vary, resulting in complex and diverse lipidomes when combined with different head groups[28]. Lipids play a variety of activities in the cell including structural and signaling functions. The considerable energy requirements for the synthesis and maintenance of the wide variety of lipid molecules have been retained throughout species, demonstrating the crucial functions of lipids in cellular physiology[27]. At the molecular level, it is not clear why the cells devote their most vitality to manufacturing and keeping the lipid substances and how they restrain them. However, it is well known that cells communicate through complicated and different lipidomes[28]. Different percentages of lipids are present in the different organelle[29], which causes a difference in the interactions between protein and lipid which affects the fluidity thickness and other characteristics of the membranes[30]. As mentioned by Scorrano *et al*.[31] the contact sites of the membranes are known as the main functional sites of the cells in which the lipids can alter between organelles. According to Muro *et al*.[32], lipids are difficult to analyze since they are not generated iteratively like proteins or nucleic acids and hence cannot be systematically deleted or tagged.

Different lipids confined to the structures related to the division like the midbody and also the cleavage furrow can imply cell division[33-35]. In biological membranes, phospholipids are distributed asymmetrically between the inner and outer leaflets of the lipid bilayer[36]. Membrane lipids are hydrolyzed by phospholipases to produce a variety of cellular mediators. Several phospholipase-derived substances, including diacylglycerol, phosphatidic acid, inositol phosphates, lysophopholipids and free fatty acids are essential for signal transmission throughout plant growth, maturity and stress responses[37]. The faithful duplication of cellular components depends on the synchronization of metabolism and cell cycle development. A misaligned regulation would cause cells to have an excessive or insufficient amount of membrane surface, which would cause anomalies in cell size. Prior to cell division throughout the regular cell cycle, cells must manufacture more membrane phospholipids[38]. Membranes are interrelated with lots of cell division proteins[27]. Few membrane proteins are integral and they can expand the lipid bilayer. Whereas others are linked peripherally with the membranes by the domains attached with lipids like phosphoinositide are linked or attached with pleckstrin homology (PH) domains. Lastly, a few proteins of cell division are attached to the lipid moiety after translation, so these are mentioned as lipidated. Cells that are in dividing mode can control their lipid constitution and placement in them properly[39]. Echard[40] suggested that the role of phosphoinositide in cell division is well understood in directing the dynamics of actin and the development of the midbody. There is a complicated interconnection between the lipids and proteins of the cell during cell division. In this process, how both of these molecules are connected and interact together is the main thing to decipher.

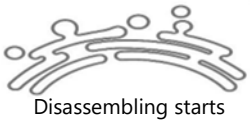
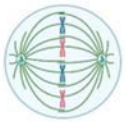
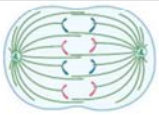
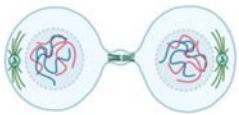
Phase of cell division	Whole cell dynamics	Endoplasmic reticulum and nuclear envelope dynamics	Mitochondria dynamics	Golgi body dynamics
Interphase		 ER and NE is properly assembled	 Highly fused	 Compact and highly connected structure
Prophase		 Disassembling starts during prophase	 Fission	 Connection breaks and disassembled
Metaphase		 Disassembled and mainly observed at cell periphery	 Fragmented	 Disassembled and distributed in the cytoplasm
Anaphase		 Disassembled and observed at cell periphery	 Fragmented	 Distributed in the cytoplasm
Telophase		 Reassembled in both the daughter cells around the nucleus	 Fusion	 Compactly reassembled into the daughter cells

Fig. 1: Changes in endoplasmic reticulum, nuclear envelope, mitochondria and Golgi body during cell division

Organelles as the building constituent of the cell: The cells give any organism shape and structure as well as perform a variety of other duties. For conducting different functions, a cell has several structures inside it named cell organelles which have individual functions to perform as well as either work singly or some of them work together[41]. If any one of them is not performing its function properly then definitely the whole body will be disturbed[41]. Each organelle has its own structural and functional changes during the cell division process. There are so many cell organelles bounded by the selectively permeable plasmalemma. In the cell cytoplasm, the cell organelles are present to perform their specific function. Some of them are membrane-less, some are single-membraned and some are double-membraned[42]. The nucleus is the largest double-membraned organelle of the cell, which mainly has all the genetic information of the cell. A network of fluid-filled membranous channels constitutes the endoplasmic reticulum of the cell[43]. They are the cell transportation system that is responsible for the movement of materials around the cell. Next is the mitochondria, which is the membraned structure known as the powerhouse of the cell[44]. Plastids are large membrane-bound organelles that contain pigments that are present in the plant kingdom[45]. Ribosomes are membrane-less cytoplasmic organelles found close to the endoplasmic reticulum[46]. Its primary function is to synthesize protein. Another important cell organelle is the Golgi apparatus, which is membrane-bound and helps in protein sorting[47]. Centrosomes are organelles comprised of centrioles and found only in animal cells[48]. Another tiny organelle is the lysosome which helps in cellular digestion[49]. The peroxisome is the membrane-bound organelle having several reducing enzymes[50]. These are the major cell organelles that have different roles in cell functioning and undergo several changes during cell division (Fig. 1).

Control of the cell cycle involves organelles: A crucial component of the regulatory network that controls the progression of each phase in the proper order is the cyclin and Cyclin-Dependent Kinases (CDKs). Additionally, both the CDK-cyclin complex's targets and the transcriptional events that follow are evolutionarily conserved[51]. Stimulation of the main cell division kinase CDK1 is the first step of eukaryotes

cell division. Then, auxiliary kinases such as Aurora A, Aurora B and Polo-Like Kinase 1 (PLK1) help in the division phase entry as well as perform a vital role in the division. It is generally believed that the functional and structural aspects of the cell cycle are primarily associated and connected with the cell nucleus[52]. Plant cyclins are unique in that they are diverse, across over 50 homologs in arabidopsis[53]. This diversity is thought to be a reflection of the various environmental factors that influence the course of the plant cell cycle as well as the specific cyclin functions carried out by various cell types[3]. The cell cycle's one-directional character is retained by targeted proteolysis of the major proteins. Phosphatases return cells from the dividing stage to the interphase stage once they have completed cell division[54,55]. Recent research suggests that synchronized alterations in the structure and positioning of intracellular organelles occur during mitosis.

Spatiotemporal localization of kinases and phosphatases during division: At the time of division, the CDK1 scattered throughout and Aurora A, Aurora B and Polo-Like Kinase 1 (PLK1) localize vigorously which helps in the error-free spatiotemporal control of cellular renovation. One of the master kinases in mitosis, PLK1, is involved in a variety of processes, including chromosomal segregation, spindle assembly checkpoint, cytokinesis and mitotic entry. Understanding how PLK1 localizes in a particular area of cells is crucial to analyzing the function of PLK1 in mitosis[56]. Some supplementary regulator kinases help to control particular incidents in kinetochores, like-MAST-L, MPS1 and serine/threonine-protein kinases haspin. The PLK1 is a kinetochore-localized protein that is crucial for the spindle assembly checkpoint and chromosomal segregation. However, still it is unclear how PLK1 localizes at the kinetochore[56]. Kinases are very important as they build the mitotic state by changing different protein-protein relationships and actions. Combes *et al*.[57] stated that the accuracy of cell division can be increased by the site-specific events of phosphorylation and dephosphorylation. In the spindle midzone, the placement of Aurora B makes an inclination toward its activity and influences the substrates to detach when the spindle extends[58]. There is a 'ruler-based' model of mitotic exit which explains that on lagging chromosomes Aurora B-dependent remembrance of condensins permits continuing chromosome loosening and nuclear rebuilding[59]. According to Wang *et al*.[60], the same inclination to Aurora B is there around the centromeres, from which it may conclude that diffusion gradients of a place may be a usual donor to the spatiotemporal controller of mitotic events. Mitosis-specific phosphorylations are separated by PP1 and PP2A, which are proteins that phosphatases in the time way out from the division of the cell[54,55,61]. The CDK1 phosphorylation inhibits PP1 activity, as does MAST-L-dependent phosphorylation, which is followed by the triggering of endosulfin, as indicated by ENSA, a PP2A inactivating protein[51,62-64]. The number of regulatory subunits is much more than those works like adaptors for the enzymes that obstruct our knowledge about biological matters[65]. The Repo-man and Ki67 are examples of PP1 adaptors that set up PP1 holoenzymes which create circumstances that help the cell to exit the cell division process like the reappearance of the nuclear envelope and reformation of chromatin[66-69]. The B55 and B56 are PP2A governmental B-type components with specific features for the mitotic exit, such as assisting in the reconstruction of nuclei, nuclear envelope and Golgi bodies[61,64,70].

Controlled proteolysis during cell division: In the case of the cell division process, the regulated proteolysis mechanism plays a significant role. The CDK regulates the progression of the cell cycle at distinct stages but regulates proteolysis in the case of cyclins[71], sister chromatid cohesion proteins[72] and major mitotic kinases[73-74] to secure the unidirectional progression. Recent research indicates that there may be numerous different ways in which the cell cycle-independent metabolic oscillations connect with the machinery involved in the cell cycle and certain biosynthetic pathways have temporally varying activity patterns throughout the cell cycle[75]. The cell cycle regulators are knocked down in proper time by ubiquitin ligases which keep forward progress. So, without these ligases, the progression in the right direction is not possible. For example, at the time of division, the anaphase conducting complex/cyclosome (APC/C) is present and the left of the process follows. The SKP, cullin and the

F-box-carrying complex are all present to keep the process in check[76]. Separation of chromatid and exit from the division state is carried out by regulated proteolysis. Direct proof for the value of proteolysis in the inheritance of organelles is very limited but it is proved that proteolysis indirectly controls the organelle dynamics due to the difference in the kinases and phosphatases levels. According to Sivakumar and Gorbsky[77], the role of APC/C is important as it suggests a spatiotemporal activity that ensures the proper settlement of cellular reorganization during mitosis. Horn *et al.*[78] mentioned that mitotic levels of DRP1 are controlled by APC/C. This is the main ubiquitin ligase that is needed for the fission of mitochondria and organellar proteostasis those combined with cell cycle progress.

Dynamic behaviour of nuclear envelope and endoplasmic reticulum during cell division: The endoplasmic reticulum (ER) creates a complex connected network of membrane layers and tubules. The endoplasmic reticulum is a polygonal network of linked tubules and layers that spans the whole eukaryotic cell and is next to the nuclear membrane[79]. The nucleus is surrounded and enclosed by ER membranes so there is a formation of a bi-membraned nuclear envelope (NE). The main role of this nucleus membrane is to give protection to the chromatin. Throughout the luminal and membrane sections of the ER, numerous nascent proteins are folded and matured. Despite the fact that the ER contains a number of elements that encourage protein folding, many proteins end up being destroyed because they are unable to fold and assemble correctly[80]. As reported by Güttinger *et al.*[81] and Champion *et al.*[23] after the NE, a series of intermediate filaments known as lamin like lamin A/C and lamin B create the nuclear lamina. This nuclear lamina gives mechanical rigidity to the nucleus and also helps the NE to organize the chromatins properly[82]. In this bi-membraned NE, massive macromolecular structures called nuclear pore complexes (NPCs) aid in transit throughout the nuclear membrane[83]. It joins the nucleoskeleton and cytoskeleton complexes to operate and connects the cell's nucleus and cytoskeleton[84]. The ER is a vital organelle that cannot be generated from scratch thus, it must be split into daughter cells before cell division[79]. The exterior nuclear membrane is always in contact with the ER, but the interior nuclear membrane (INM) is biochemically different from the outer one because it has its own set of proteins that interact with the lamina, histones and the proteins that are held together with chromatin[85,86]. The NE disintegrates in higher eukaryotes during the open cell division process and the destiny of the NE along with its components has piqued researchers' curiosity, especially in metazoans[87]. The INM is a highly specific membrane segment with several native intermembrane proteins that take an essential part in nuclear engineering, such as coupling the INM to chromatin and the nuclear lamina's intermediary filament network, transcription control and signal transduction[80,88]. It can be observed distinct morphologies in ER and NE during the interphase. The NE is a disjointed and flexible platform that affects gene expression and chromatin organization. In plant mitosis, NE offers a structural and regulatory framework. However, it must be rebuilt after each cell division[89]. The cisternal and tubular-reticular architecture of the external ER is created by membranous sheets and numerous morphometric proteins that make up the NE. The Reticulon (RTN1-RTN4) and Receptor expression-increasing proteins (REEP1-REEP6) are proteins that stabilize majorly curved ER tubules and the curved edges of sheets[90,91]. As mentioned by Nixon-Abell *et al.*[92] and Schroeder *et al.*[93] super-resolution microscopy reveals more morphological differences, such as ER matrices and reactive nanoholes. Microtubule cytoskeleton forms the interphase ER and during division, many modifications take place to allow the remodeling of ER[94,95].

Nuclear envelope breakdown is brought at the commencement of mitosis: Nuclear envelope breakdown (NEBD), which entails the loss and repositioning of the NE's structures and components, is essential for open mitosis. In the late prophase, NEBD in plants occurs relatively early than in animals[96]. The CDK1 relying on phosphorylation of INM-chromatin and INM-lamina binds the nuclear membrane to chromatin and aids in the redistribution of nuclear membrane proteins to the mitotic ER at the time of a cell's mitotic entrance[23]. The CDK1 and PKC phosphorylate the lamins which disintegrate the nuclear lamina and help to free the NE from the nucleus[97,98]. As reported by Bahmanyar *et al.*[99] phospholipid

metabolism is spatially controlled when phosphatidic acid is inactivated and phosphatase lipin restricts *de novo* ER membrane formation to aid in the breakdown of the nuclear membrane. After the NE is disassembled, each NE and ER characteristics are destroyed. The envelope protein molecules are split into daughter cells using ER elements. Audhya *et al.*[100] reported in the case of *Caenorhabditis elegans* that nuclear membrane disassociation is carried out by the reticulons and also suggested that a more tubular morphology is formed from the sheet-like NE which aided in the demembranation. Dynein creates pulling forces that aid in demembranation by the generation of NE invaginations in prophase[101-104]. As observed by Mori *et al.*[105] rapid polymerization of an F-actin layer at the nuclear border aids physical breakdown of the NE in starfish oocytes. Cyclin-Dependent Kinases (CDKs) have primary control over cycle progression, making them important conserved regulators. Animal CDK1, *Saccharomyces cerevisiae* Cdc28 and *Schizosaccharomyces pombe* Cdc2 are all closely related to plant CDKA, which is active throughout the cycle. Plants only have a second form of CDK, named CDKB1 and CDKB2, which is active from the S to M phase[96]. But in mammalian cells, the contribution of actin in mediating NE disassociation is not clear and this will be a major area of study in the future[106]. At the start of mitosis, the rapid disassembly of NPCs is carried out by the phosphorylation of nucleoporins (NUPs)[107]. The transmembrane NUPs enter the mitotic ER and the other NUPs are liberated into the cytoplasm[108,109]. Current studies show that in early mitosis, the nuclear pore knockdown and the subsequent disassociation of the nuclear membrane are the results of the transient localization of PLK1 to NPCs. The PLK1 and CDK1 act together and hyper-phosphorylate NUP98 and NUP53. These take the main step toward the separation of the key interconnection of NUPs and the breakdown of the nuclear pore[56,107,110-112]. At the start of mitosis, this kind of phosphorylation-induced separation of inter-NUP interactions is the major cause of the disintegration of nuclear categorization and disassociation of the NE.

Division results in morphometric changes to the ER: The morphology of the ER in the mitotic phase is not clearly known but in interphase ER shows various tubulo-reticular and cisternal subdomains. The outer nuclear membrane (ONM) as well as the inner nuclear membrane (INM), which creates the nuclear envelope (NE) is continuous with the ER[80]. Live cell imaging of mitotic cells reveals that extended curvilinear membranes are present in the ER membrane with a cisternal arrangement[113,114]. Volume electron microscopy approaches reveal the tubular-reticular and peppered sheet structure formation of ER[115-116]. During the division, the ER-forming proteins REEP3 and REEP4 form the tubular architecture. The cell type determines how much each morphology predominates[116]. The nucleoplasm and cytoplasmic proteins that interact with the envelope are essential to its completion, as are timing and location[96]. Schweizer and Maiato[117] reported that in dividing cells the peripheral ER is not attached to the spindle and it forms a barrier to make possible the exit of other organelles. The ER morphology can be affected by mitotic phosphorylation of ER morphometric proteins by stopping LNP multimerization[118-119]. Because of these events, the connection between the ER and microtubules is affected to stop the ER from assembling with spindle assembly[120]. As observed by Schlaitz *et al.*[121] REEP3 and REEP4 are also critical for concentrating the ER at spindle poles, preventing them from forming early associations with chromatin and resulting in cell division error. The attachment of ER with spindle poles and astral microtubules controls the ER inheritance[122]. Therefore, it is important to understand definitely through which process the spindle controls the inheritance of ER without letting these organelles hinder chromosome segregation[123].

Regeneration of nuclear enclosures: The NE reappears in late anaphase and telophase when the daughter nuclei are connected and surrounded by ER membranes[23,124,125]. Unlike animal cells, the multiplying plant cells build a new cell wall to finish their division. It is necessary to synchronize the division plane because the wall inhibits later correction. The wall inhibits the swelling that happens during animal cell division. As a result, the membrane plays a significant role in the formation of the two plasma membranes as well as the movement of vesicles that carry the building blocks for the new wall[96]. The

switch from tubular to sheet-like architecture is a frequency-limiting phase in NE rebuilding as evidenced by the fact that overexpression of ER tubule-shaping proteins hindered the prior process[126]. The chromatin-associated protein molecule barrier-to-autointegration factor (BAF) forms a crosslinked chromatin interface where the ER membranes are assembled[127]. Reversible disintegration of the interphase lamina may govern NE deconstruction and rebuilding during mitosis, based on the physical features of the interphase lamins and their activities throughout the mitosis. The NE reappearance requires reversal of the mitotic state and PP1 and PP2A play crucial roles in linking nuclear reorganization and chromatin decondensation. Thus, the absence of these PP1 and PP2A affects the reorientation of NE. The PP2A governing component B55 is necessary for NE reconstruction during mitotic departure[61]. In timely nuclear membrane reorientation, lamin rearrangement, NPC reorganization and the re-employment of INM-chromatin need PP1 by its targeting subunits PNUTS[128], AKAP149[129], Repo-Man[67] and Ki67[130,131]. The NE reappearance in late-segregating chromosomes can be delayed by Aurora B which suggests that re-memorization is controlled spatially by the midzone Aurora B gradient[132]. This type of Aurora B-dependent spatial regulation of nuclear membrane reassembly may happen even in human cells. The NE governs macromolecule interchange between the nucleus and the cytoplasm and may have a role in the interphase chromosomal architectural organization[133]. However, modern research suggests that rather than membrane generation, it regulates the presence of NPCs and the nature of the NE[134]. The NPCs that have been disassembled at the end of mitosis are reassembled by incorporation into NE which has previously been secured by the formation of the membrane around a pre-existing pore structure. The nuclear envelope (NE), which serves as both a physical barrier partitioning the nucleus from the cytoplasm and a gatekeeper for selective transport between the two, is essential to the structure of eukaryotic cells. In contrast, the NE breaks down during open mitosis to enable spindle formation and chromosomal segregation, which causes the nuclear and cytoplasmic soluble portions to mix[135]. The nuclear lamina, a fibrillar protein meshwork (typically 10-20 nm thick) that lines the nucleoplasmic face of the inner nuclear membrane appears to be connected to pore complexes. Relating live-cell electron tomography illustrates that as anaphase continues, small gaps in the aperture of NE sheets expand to gain the morphological features of NPCs[136]. Transient intracellular calcium elevations may influence the advancement of mitotic cells into anaphase. It is concluded that the NE is assembled around one pre-existing aperture at this moment. The NPCs can also be incorporated into the centre of the nuclear membrane when the nucleus increases, simulating NPC integration in the interphase. According to Otsuka *et al*.[137] through the INM and extrusion of a pre-existing body NPCs can form at the time of mitotic exit as revealed by tomographic approaches. In fact, at the time of mitotic exit, the lamina and the INM proteins are formed by nucleoporins[138]. The leftover holes in the NE are sealed by membrane fusion which is the last event in nuclear regeneration. Point fusion in ER membranes allows NE to expand to accommodate decondensed chromatin and plug holes in the two-layered NE, splitting the internal and external nuclear membranes associated at these places where a specialized fusion event is required. The ESCRT-III mediates this process (annular fusion), which is responsible for ontologically identical membrane filtration events in several other cellular activities, for the assignment of the endosomal sorting complex[10,139,140]. In light of the interphase lamins' physicochemical features and simultaneous disintegration of their behaviour during mitosis, the NE deconstruction could be regulated by the lamina and the process of mitotic rebuilding. The CHMP7 is an ESCRT-III component found in the ER that transiently crosses target places during annular fusion by interacting with the INM protein LEM2[141,142]. It is well known that the ESCRT-III complex causes the intercellular bridge to tighten and scissor in several animal cells[10]. Elements of the ESCRT-III are allocated to close the leftover holes in the NE, allowing nuclear fragmentation[139,140]. Gaps in the NE are formed when membranes enclose spindle microtubules and the microtubule-severing enzyme spastin is allocated by ESCRT-III to associate microtubule disassembly with membrane sealing[140]. However, the connection between the mitotic reassembly of NPCs and this process is still unknown[143-145]. The discovery that this pathway exists in archaea shows that this mechanism has evolved to play a function in cellular division[146].

Mitochondria during cell division: Mitochondria are bi-membranous organelles that serve as the cell's primary energy producers. Plant mitochondria are pleomorphic, active organelles. The higher plant chondroma (all mitochondria in a cell) is usually made up of a large number of morphologically distinct mitochondria[147]. It is suggested by Labbé *et al*.[148] that continuous fusion and fission happen inside the mitochondria for adaptation to the energy requirements of the cell. Mitochondria contain their independent genomes that are duplicated in a separate growth and division cycle from the cell cycle, something that the other organelles lack. The genes that regulate mitochondrial fusion in plants are unknown; most of the genes reported to be associated with fusion in other organisms have no plant homologs[147]. Therefore, a dividing cell must finish two tasks at a time including mitochondria division, which firstly optimizes energy production at the time of division and secondly relocate mitochondria between the daughter cells. Mitochondria are extremely dynamic organelles that experience fission, fusion and transport events during various cell cycle stages. Notably, many kinases phosphorylate the essential mitochondrial components and promote mitochondrial fragmentation during mitosis to enable efficient mitochondrial dispersion and transmission to two daughter cells[149]. As proposed by Mishra and Chan[13] mitochondria are united throughout the G1 phase, stretched during the S phase, divided during G2-M transition and shattered during division. Dynamin-related GTPase proteins (DRPs) control mitochondrial fission-fusion cycles. Labbé *et al*.[148] proposed that mainly DRP1 controls the fission and the distribution of mitochondrial DNA at ER-mitochondria contact sites. Mitofusin 1 and Mitofusin 2 (MFN1 and MFN2) are DRPs that control mitochondrial outer membrane merging, while DRP OPA1 controls mitochondrial inner membrane merging. According to Fenton *et al*.[150], the morphology of the mitochondria can change depending on a number of cellular signals. Increased intracellular calcium and mitochondrial depolarization are two stimuli that are frequently employed to modify mitochondrial network architecture. The Aurora A through the small GTPase RalA and CDK1 activates and phosphorylates DRP1 during G2-M, which results in higher mitochondrial fission[151,152]. The mitochondrial disintegration mechanism, on the other hand, is highly preserved[147]. This may be coming together by the ubiquitylation and breakdown of MFN1 and MFN2[153]. Higher plants' mitochondria are reported to differ significantly from other eukaryotes in a number of ways[154]. The DRP1 is sumoylated and ubiquitylated in mitotic exit, causing its disintegration and restoration of mitochondrial fusion following cytokinesis[78,152]. According to Adachi *et al*.[155], the activity of DRP1 is restricted by attaching to phospholipids and this incident suggested that other ways may also take part in controlling mitochondrial position during cell division. Dynamic mitochondrial partitioning may potentially contribute to cell fate determination. Mitochondria are involved in the production of various substances, including phospholipids, nucleotides and many amino acids, in addition to their critical roles in respiration and photorespiration[147,149]. As a result of the uneven splitting of stem cells, the more stem-like daughter cell maintains newer and more efficient mitochondria[156]. During the cell dividing process, Mitochondrial disintegration may aid in the stochastic transmission of mitochondria as suggested by Jajoo *et al*.[157]. Mitochondria are pushed and placed to the ends of dividing cells in *Schizosaccharomyces pombe* and re-disseminated soon before splitting in according to the partitioned cytoplasmic content. Mitotic kinesin KIF5B, coupled with MIRO1, attaches astral microtubules to transport mitochondria to the trench during the mitotic departure[158]. The MIRO1 and MIRO2 appear to operate as a connector between the mitochondria, microtubules and the actin cytoskeleton. The other of these connections can be made via the non-processive myosin drive unconventional myosin-XIX, which is transcribed by MYO19 and found in mitochondria's outer layer[159]. During mitotic entry and exit, the control of mitochondrial-cytoskeletal interactions permits future studies to know how cytoskeletal targeting aids accurate inheritance patterns. The activity of particular cyclin-Cdk complexes determines cell-cycle progression. These complexes control several structural changes essential for cell division, including changes in mitochondria shape that allow for mitochondrial segregation across daughter cells[21,160-162]. Major avenues of cellular processes, including crucial roles in the regulation of immunity and mitochondrial quality control, appear to be influenced by mitochondrial division[163]. How cells regulate the

availability of energy throughout the cell division mechanism is a vast and fascinating subject. Mitochondria are the main energy makers of the cell and they could aid cells to satisfy the high energy needs during the whole division process[164]. In addition to offering power for life, mitochondria can serve as a death trigger[147]. At the activation by Aurora A, the small GTPase RalA comes to the mitochondria. The degradation of RalA and RALBP1 demonstrates a regulatory link between the division mechanism and the cell's power supply[151].

Golgi apparatus disassembly and suppression of transport activity: To continue the equal inheritance at the time of cell division, the Golgi body must be disorganized and again reorganized in daughter cells as this is a single-copy organelle. Several extended piled cisternae formed an interconnected ribbon-like pattern in Golgi[165]. However, certain protozoa only have a solitary Golgi mini-stack within the cell and cytokinesis is purely in charge of controlling the mini-stack's division[166]. Outer Golgi reassembly and piling proteins (GRASPs) such as GRASP55 and GRASP65, play an important role in Golgi structure[167,168]. The Golgi apparatus in mammalian cells is made up of a highly organized parallel network of cisternae that form a layered architecture in the cell's perinuclear zone[166]. Golgins are a class of coiled-coil proteins that interact with anchoring factors such as p115 to grab vesicles[169,170]. Golgins can be linked with GRASPs for making the structure of the Golgi. In proper controlling of biosynthetic and endocytic trafficking pathways, the Golgi has the main role at the time of cell division to regulate the remodeling program that certifies its disassembly and restoration. Golgi dispersion begins in the late G2 phase of the cell cycle. As the C-terminal-attaching protein, 1/brefeldin A ADP ribosylated substrate (CtBP1/BARS) breaks links between cisternae layers, the Golgi ribbon splits into solitary ministacks[171,172]. At the time of mitotic entry, GRASPs can be phosphorylated by CDK1, PLK1 and MAP kinases which conceal the trans-oligomerization and help in the Golgi ribbon unattached[173-178]. The Golgi apparatus is hypothesized to have a role in the production of complex carbohydrate frameworks that are coupled to a variety of cellular and secretory proteins and lipids, as well as the filtering of these proteins and lipids to their proper subcellular locations. The GRASP65 also stabilizes microtubules, which are required for interphase Golgi aggregation. Ribbon unlinking is caused by its phosphorylation and deactivation after mitotic entry, which subdues its attachment to the cytoskeleton[179]. The shifting of Aurora A to centrosomes is halted by blocking the unlinking of the Golgi ribbon, which also stops the mitotic entrance[180]. At the beginning of mitosis, the GC is rearranged to form an entirely new ribbon in each of its daughter cells. During mitosis, the GC is fragmented into vesicular/tubular clumps that are scattered in the cytoplasm until the infamous "Golgi haze" is created[178]. Elements that contribute to the fusing of Golgi membranes to generate cisternae and the following stacking of these cisternae to form stacks have been found in a number of investigations in recent years[168]. Cells keep monitoring the stability of this organelle to allow mitosis to continue if the correct lineage is feasible[174,181]. After the unlinking of the Golgi stack, the coatomer COPI helps the Golgi cisternae to vesiculate through persistent budding. Phosphorylation of golgins such as GM130 paused the vesicular fusion to inhibit the relation with p115[182-184]. Membrane fusion at the Golgi is also controlled by the Ubiquitin dynamics. For instance, Tang *et al.*[185] and Huang *et al.*[186] reported that HACE1, an E3 ubiquitin ligase, ubiquitylates the Golgi SNARE syntaxin 5 to halt its association with its corresponding SNARE BET1 and disrupts integration activities at the Golgi when mitosis begins. The VCIP135-dependent deubiquitination of this SNARE restores union, whereas phosphorylation of VCIP135 blocks this action in mitotic cell division[187,188]. The GRASP55, a GRASP65-related mammalian protein, is involved in the development of Golgi bundles[168]. The ATPases NSF and valosin-containing protein allow the disassociation of trans-SNARE complexes to maintain continuous cycles of vesicular integration processes. Mitotic phosphorylation of the VCP-adaptor p47 removes this protein from walls to stop VCP-dependent membrane addition in mitosis when mitotic phosphorylation of GM130 obstructs NSF-mediated union[184,189]. Small tubulovesicular clusters are developed through the inhibition of membrane fusion to form cytoplasmic Golgi 'haze' and this haze may be divisible into daughter cells by stochastic

mechanisms[178]. The inheritance of these membranes is supported by a mitotic spindle and GM130-microtubule interlinkage[190]. Another theory proposes that the reorganization of Golgi parts into the ER is caused by a halt in transport[191,192]. During the G2–M transition, Golgi separation occurs in tandem with global cellular reintegration activities such as spindle formation. Following the reassembly of the Golgi complex, there is an issue with the mitotic breakdown in a cell-free system. In the first phase, cisternae are reconstructed from the ground up MGFs[193]. During mitotic entry, CDK1 phosphorylates GM130[184] which detaches its connection with p115 and a nuclear localization signal is produced within GM130. Importin-alters its location and is deposited in Golgi membranes as a result of this nuclear localization signal, releasing it to inhibit TPX2-dependent spindle formation[190]. The Golgi restoration takes place mostly in between the anaphase and cytokinesis stages. The reformation of the Golgi cisterna, restacking of the cisterna, congression and fusion of piles to produce ribbons are all required for Golgi rejuvenation. The Golgi apparatus's organized architecture is hypothesized to represent the need for these enzymes and the protein filtering mechanism to be partitioned to carry out a particular set of post-translational alterations and sorting activities. Metal ions have recently been shown to be essential for the Golgi complex to continue functioning properly[194]. The PP2A-dependent dephosphorylation of GM130 reconstitutes p115-dependent vesicular tethering and fusion[195]. With this, cisternal re-stacking is permitted by PP2A-mediated dephosphorylation of GRASP65[196,197]. Both radial and tangential microtubule-dependent mobility help to rebuild a Golgi string[198]. 'Twin' Golgi are formed in each daughter cell in telophase[199]. A shorter twin faces the cleavage trench, while the bigger twin faces the centrosome. In cytokinesis, the shorter twin moves around the nuclei to join its twin and recreate a single Golgi. The Golgi apparatus disperses into the farthest portions of the cell-cytoplasm in highly differentiated cells like the oocyte or nerve ganglion cell and so lacks its link to the centrosome in most situations. Goss and Toomre[200] revealed that the smaller twin may deliver Golgi-derived particles to the midbody for abscission, although the rationale for this placement is unknown[201]. The ARF1 guanine nucleotide transfer factor GBF1 was recently found to be required for the movement and attachment of these two Golgi twins.

Actin and phosphoinositide clearance from the midbody: The reshaping of actin, change from a contractile band to a midbody band and the formation of the abscission region are all required for cortical entering. Many organelles in a wide range of plant cells acquire non-random orientations during cellular division. Wong *et al.*[202] mentioned that the lipid phosphatidylinositol 4,5-bisphosphate (PtdIns(4,5)P2) regulates actin movements and is present in the cleavage furrow. As cytokinesis progresses, this PtdIns(4,5)P2 is used, which is important for furrow durability. At the midbody, Rab35[203] and 27 kDa inositol polyphosphate phosphatase-interacting protein A (IPIP27)[204] coordinate the PtdIns(4,5)P2 phosphatase OCRL. Through hydrolysis of PtdIns(4,5)P2, OCRL allows midbody actin to be removed for effective abscission[203]. Phosphatidylinositol 4-phosphate (PtdIns(4)P), which is the product of PtdIns(4,5)P2 hydrolysis, can maintain the PtdIns(4)P-binding proteins required for cytokinesis[205]. As a result, a phosphoinositide transformation cascade might be used to modulate cytokinesis spatially and temporally. To understand the mechanism underlying this placement, more research will be required. The Rab35 interacts with recruits and activates the oxidoreductase MICAL1, which depolymerizes and decondenses actin filaments and facilitates actin cleanup[206]. Other members of the MICAL family help to orient vesicles within the midbody. The Rab11-positive endosomes are transported by MICAL-L1[207] and Rab8-positive vesicles are tethered by MICAL3[208]. These suggest that this family of proteins has an important role in synchronizing cytokinetic consequences.

Membrane dynamics in small organelles: Cells also contain many smaller membrane-bound structures whose biological roles are still under research. The inheritances of these small organelles are less compactly regulated than that of huge single-copy organelles previously described as they are usually numerous and dynamic[209]. The membrane of pre-existing peroxisome lengthens to create a tubule during the mitotic division, which subsequently constricts and eventually undergoes scission to produce new peroxisomes[210]. Fate-determining endosomes are dynamically separated at the midzone microtubules with the support of kinesin motors in unevenly dividing sensory organs in *D. melanogaster* with asymmetric

spindle disarrangement directing endosomal transmission[211]. Recent researches suggest that endocytic membrane transit may play a role in cell plate development. In the near future, one main area of study can be revealed by dealing with the mechanisms of symmetric versus asymmetric inheritance of endosomes[212]. During the time of mitotic exit, Endosomes are also needed[213,214]. Cytokinesis failure is the result of the shutting down of endosomal sorting due to the minute molecule prazosin[215]. Exocyst-dependent vesicular union has been proposed as a mechanism for total abscission in previous investigations[216]. However, more recent proof reports that endosomes are positive for Rab11 family-interconnecting protein 3 (Rab11-FIP3) or Rab35 convey proteins needed for the finishing of cytokinesis[217-219]. The prevalence of intra-midbody vesicles decreases as midbodies mature, indicating that the need for these union events decreases as cells attain abscission[213]. Near the future abscission site, both Rab11-FIP3-positive and Rab35-positive endosomes are stored to produce secondary ingression which is an intra-midbody construction that specifies the final cut site[206,220]. According to Pohl and Jentsch[221], it must eliminate and/or reuse midbody remains of post-abscission at the end of cell division. This could be attributable to autophagy, although other evidence suggests that phagocytosis completes the process[222]. However, more research in the future is needed to confirm the actual roles of autophagy and the division of autophagosomes at the time of cell division. With a few exceptions, each cell in the G1 phase contains just one centrosome, which is made up of two centrioles and pericentriolar materials (PCM) and which need to be replicated before cell division occurs so that the two newly created cells both inherit a single centrosome[223]. During interphase, many organelles in plant cells have a more or less random distribution, but during mitosis and/or cytokinesis, they assume fairly definite positions[224]. The synthesis and turnover of lipid droplets, which serve as repositories for membrane extension and energy generation, are tightly regulated[225]. A recent report by Cruz *et al*.[226] proposed that the cell cycle regulates the lipid droplet number and localization at the time of division. Peroxisomes can be replicated by division or can also be formed *de novo* from the ER. At the time of mitosis, these peroxisomes are oriented together around spindle poles to maintain the symmetric inheritance[227,228]. The deletion of the peroxisomal protein PEX11B causes uneven peroxisome transmission, mismatched mitotic spindles and aberrant division in epidermal progenitor cells[229]. Midbodies help in abscission without which cell division is incomplete and it needs the help of membrane and cytoskeletal reorganizing activities[230].

Dynamics of plasma membranes: The plasma membrane of the cells at the time of division takes various curve-like structures as they react to the mechanical stress due to their close attachment to the cortex and their rearrangement that supports the enhancement of the surface area of the cells. According to Ramkumar and Baum[231], cell volume, as well as surface topology, changes gradually at the time of mitosis. Mitosis begins with a dramatic series of changes in cell shape, motility and polarity for many animal cells[232-234]. Cells go through the shaping of the membrane, delivery and removal as there is very little time for d*e novo* membrane synthesis. The membrane's architecture and functional activity vary not just between specimens but also within cell populations[235]. Newly formed membranes emerge from internal parts through cell trafficking methods like exocytosis or endocytosis recycling[236] or through plasma membrane restructuring[237]. During diffuse development, enzymes, carbohydrates and other elements of the cell wall are continuously and actively transferred to and from the plasma membrane. Vesicles carrying cell wall components travel along actin and microtubule-based cytoskeletal pathways. During cytokinesis, many of these elements as well as other proteins, vesicles and lipids are transported to and from the cell plate[238]. At the anaphase stage, the plasma membrane expands and this process helps the spindle to take a proper position for maintaining symmetry in the division. The architecture of the plasma membrane and the location of the spindle has an inverse relationship. The spindle position perturbation relocates the apparatus during the asymmetrical expansion of the cortex membrane through membrane blebbing[237]. The cleavage furrow location, which is controlled by the polar nature of membrane blebbing through rhythmic motion along the cortex, relieves membrane tension created by repeated changes in membrane shape throughout the division phase[239]. Expansion of the polar membrane is the cause of a driving force to help the furrow ingression in the embryos of sea urchins[240]. Different internal matters are acting as a helping hand in membrane expansion that is not yet known but all the above-mentioned examples prove

that different cells have different pathways for membrane expansion. There is an intense relationship between the endocytic network and the plasma membrane. The plasma membrane serves as the entry point into the endocytic network. There are two different opinions about the endocytosis is continued or not during the time of cell division. Some proposed that there is no endocytosis at the time of cell division[241] and according to others, endocytic recycling can be the means of plasma membrane expansion[236]. But the most followed opinion says that some amount of endocytosis is carried out during the division. Growth factor receptors and other important endocytosis cargoes have been given the first chance for their partitioning during division[242].

Plasma membrane is connected to the cell cortex: The plasma membrane consists of some lipids and proteins that interact with the cortex in a highly structured, organized and constrained manner[243-245]. These physical limits are crucial in terms of functionality. Schmidt and Nichols[246] reported that in the cleavage furrow, a diffusion barrier is present. It is attached to both the membranes and also with the cytoskeleton through the help of filament-forming septin proteins. Measurements of lipid and protein dispersion rates in remanufactured synthetic bilayers surpass cellular membranes with similar lipid content by more than a factor of ten[247]. Various additional membrane-binding adaptors help the plasma membrane of a dividing cell to make a connection with the cell cortex[27]. It includes anillin[248], ezrin, radixin and moesin (ERM) proteins[249]. The Rho, a small GTPase and its deformers are important controllers of cytoskeletal mobility that work in conjunction with the plasma membrane[249-252]. Some other proteins that control the actin cytoskeleton have lipid-binding domains in them such as capping protein[253], profilin[254], cofilin[255] and myosin II[208]. Actin filament polymerization against membranes generates force for several cellular activities, including organelle dynamics, endocytosis, phagocytosis, migration and morphogenesis[256]. A synchronized pattern of cell shape modification characterizes both cell division and cell migration. Despite their apparent functional differences, these two processes may have identical mechanisms and signaling pathways when it comes to cell shape[257]. These crucial linkers or responder proteins are not insider membrane proteins; instead, they are linked to the membrane by lipid alterations or lipid-attaching regions. The reproducing cell can control and perform membrane modifications with high spatial and temporal accuracy due to bidirectional interconnections between the membrane as well as the cortex. Throughout cytokinesis, daughter cells are irrevocably separated by furrow build-up and plasma membrane fission[258]. Knowing the processes underlying the regulated restructuring of the plasma membrane and its interaction with the cortex in cell cultures and 3D tissue settings will be a huge task in the coming years[259].

Cytokinetic abscission: Our current understanding of how cytokinesis is completed has been fueled by the midbody location and functional requirements for ESCRT proteins in cytokinetic abscission[10]. Although the intercellular tract holding the midbody is one of the most visible structures in dividing animal cells, its role in the abscission phase of cytokinesis is mostly unclear[260]. As reported by Fabbro *et al.*[261] a portion of the central pindling complex, MKLP1 involves the centrosomal protein CEP55 in the midbody. In turn CEP55, by direct connection with upstream ESCRT elements TSG101 and ALIX engages the ESCRT-III machinery[262-266]. During anaphase, cytokinesis in animal cells begins with the formation of a central spindle. Active ESCRT-III filament polymerization is believed to operate cytokinetic abscission[213,266,267]. By super-resolution approaches, it has recently become known that spiral filaments of ESCRT-III broaden the abscission area[268]. In various cell culture mechanisms and certain tissues, ESCRT-III is crucial for cytokinesis. For instance, it can be mentioned about the germline stem cells in *D. melanogaster*[269,270], in mice[271] and some crenarchaea[146]. This shows that as animals' sophistication has grown, this machinery's ancestral job in cell replication has been co-opted for additional membrane restructuring processes. It is recently shown by Karasmanis *et al.*[266] that at the midbody bi-ringed membrane-bound septins direct orderly ESCRT assembly during cytokinesis. As a result, imaging-based experiments have been the primary research technique for uncovering many elements of the molecular signaling pathways that govern mitosis and cytokinesis[160]. In yeast cytokinesis, septins have important roles as connections between septins and controllers of cytokinesis in metazoans such as Rab35[219] and GOLPH3[205] were recognized.

Table 1: Biophysical techniques for studying the membrane dynamics

Techniques	Advantages	References
Confocal microscopy	For following membrane dynamics on a time scale (flat membranes can easily studied in the focused thin sections)	Minsky[282]
Fluorescence recovery after Photo-bleaching (FRAP)	Gives data about the dynamic nature of the sample fluorophores	Snapp *et al.*[290]
Total internal reflection Fluorescence (TIRF) microscopy	Detection of single molecules, high temporal resolution for observing the dynamic phenomena of the surface	Groves *et al.*[289]
Single particle tracking (SPT)	Very useful method for distinguishing different motion types of the particles	García-Sáez and Schwille[286]
Fluorescence correlation Spectroscopy (FCS)	Powerful in characterization of the fluidity and the state of lipid bilayers	Haustein and Schwille[291]
Fluorescence interference Contrast microscopy (FLIC)	More topographically quantifiable, greater range than TIRF	Groves *et al.*[289]
Image correlation spectroscopy (ICS)	Spatial correlation studies of the fluctuations in fluorescence intensity of an image	Petersen *et al.*[292]
Spatio-temporal image correlation spectroscopy (STICS)	Whole spatial and temporal correlation function analysis of a stack of images is possible	Hebert *et al.*[293]
Particle image correlation spectroscopy (PICS)	Combination of ICS and SPT	Semrau and Schmidt[294]
Secondary ion mass spectroscopy	Gives us a map of the chemical constitution of supported bilayers with phase distinction	Kraft *et al.*[295]
Stimulated emission depletion microscopy (STED)	Improve the resolution to a few 10 nm	Nägerl *et al.*[296]
Photoactivated localization microscopy (PALM)	Increases resolution	Sengupta *et al.*[297]
Near-field scanning optical microscopy (NSOM)	Estimates the fluorescence intensity on the surface of the membrane with a very sharp optical fiber	Ianoul and Johnston[298]

Interactions between cells affect cell division: The extracellular environment and the nearby cells of the dividing cell have a deep connection during the division stage[231,272]. According to Roubinet *et al.*[257] a force is required for all the changes that happen in mitotic rounding, ingression of the cleavage furrow and sealing after abscission[273]. If consider the monolayers of the epithelium at the time of mitotic entry, the tension between cells decreases, as well as the cytokinetic progress is put forward by the neighbouring cells. However, if the adhesive force is more than required then cytokinesis failure may happen[274,275]. All of these prove that every cell has a connection with the external environment as well as its nearby cells during division and there are also many mechanical signals and signaling cascades that are there for maintaining this connection with nearby cells. Changes to cellular mechanics are brought on by both exogenous (contact, wounding, dehydration, floods, pathogen invasion, gravity) and endogenous (expansion, cell mobility, division, morphogenesis) sources, which must be detected and responded to[276]. Plant development and its morphogenesis can be influenced by the cell cycle regulating and controlling mechanism. However, all the cell cycle regulation is more complicated than simply speeding up or lowering cell synthesis, which results in quicker or slower growth[277]. Depletion of cell plasma membrane inhabited proteins such as particular G protein-coupled sensors can inhibit cell division[278,279]. As proposed by Özlü *et al.*[280] mitotic and interphase cell surface proteomes are different. This opens up new possibilities for signals that connect the cell division process to the cell's external environment.

Techniques to study biological membrane dynamics: During the previous few years, understanding of the molecular structure of biological membranes has substantially improved due to the explosion of new technical interventions that have made it possible to examine membrane surfaces in unimaginable ways[281]. There are several novel methods for studying the membrane dynamics available most of them are microscopy-based (Table 1). Due to these recent advances, membrane and organelle dynamics during cell division can easily be examined. These techniques help us to examine the physical, biochemical as well as biophysical changes of the cellular membranes during the whole cell division process[282]. Confocal microscopy is the most widely used technique in many laboratories for the routine study of biological

membranes. To maximize its spatial resolution power, it exclusively measures the fluorescence of thin sample slices[282]. In confocal microscopy, in front of the detector, a mini pinhole is kept for imaging only the fluorescence coming from the focus of the sample. A laser beam is used as a light source to facilitate the scanning of the sample. Fluorescence recovery after photo-bleaching (FRAP) is a technique or mechanism that is used in confocal microscopes to let us know about the dynamic nature of the fluorophores in the sample[281]. The foundation of FRAP is the study of the temporal rise in emission intensity in a sample area that has previously undergone photobleaching under intense laser illumination. But in this technique, high illumination power is required which often proves harmful to the sample and even to its dynamics[283]. Total Internal Reflection Fluorescence (TIRF) Microscopy uses the optically dense medium like glass and the less dense medium like an aqueous solution to its complete internal reflection to limit illumination to a range of 100-200 nm above the glass/water boundary[284]. In TIRF, data are assembled with cameras that have a huge resolution. The main advantage of this technique is it allows to detection of even the change of a single molecule[284]. In addition, single particle tracking (SPT) can be used to track the movement of individual lipids or proteins inside membranes[247,285,286]. In this technique, Nanoparticles coated with ligands (like antibodies) are used to label the samples. The SPT develops into a very potent technique for identifying different motion patterns such as directed flow, irregular diffusion, confined diffusion and Brownian diffusion[287]. Fluorescence correlation spectroscopy (FCS) is based on the statistical assessment of fluctuations in fluorescence over time, which are typically brought on by the diffusion of a single fluorophore into and out of the sensor container[288]. Characterizing the fluidity and state of lipid bilayers of various complexity has shown to be highly helpful[286]. In fluorescence interference contrast (FLIC) microscopy fluorescent probes are placed close to a reflective plane[289]. The fluorescence intensity is modulated and the collected data can be used to map topography at the nanoscale level. This has a greater range than TIRF as it allows more topographically quantifiable limits[289].

Technological intervention to achieve conceptual advances: Understanding the dynamic regulation of organelles and sub-organellar structures at the time of division would be difficult until we can anticipate and regulate them in high resolution non-invasively. During mitosis, several membrane compartments undergo rigorous spatiotemporal reorganization. Nowadays, several new technologies such as lattice light sheet microscopy with adaptive optics have evolved for high-resolution live imaging of cellular organelles at the time of their partition[134]. The 4D imaging data have permitted to making of a dynamic protein atlas of cell division[299]. The cell is made up of DNA along with complex elements that must be meticulously passed on to the offspring. Several new technologies are being developed that allow us to see several new insights into the division process. Modern imaging devices and organelle-specific probes are being created. The capacity to precisely alter division structures utilizing novel approaches like optogenetics catalyzes technological intervention to study membrane dynamics[300-302]. Distinct intracellular membrane groups with different roles appear to have discovered different methods to ensure proper transmission[21]. Although symmetrically dividing cultured cells have been researched extensively, mechanical conditions in 3D and uneven situations may also be important for organelle heredity and appropriate division. In the future, the knowledge about these processes will surely increase with the evolution of revised models to enquire about the division in more physiologically relevant ways. Combining data from several areas of inquiry will be a major conceptual issue. This is challenging and tough to undertake since the primary viewpoints will certainly be achieved by exploring different fields and dimensions to gather atomic relationships that determine lipid behaviour to organelle migration in dividing cells[303]. Life propagation necessitates adequate knowledge to duplicate life to understand the knowledge of cell division. For instance, it is not well understood how the specification and molecular interactions of membrane proteins and lipids involved in organelle remodeling disrupt organellar function. Intracellular membrane sections tasked with various activities seem to have devised several strategies for ensuring proper transmission[27]. During mitosis, several membrane sections undergo extensive spatiotemporal reorganization. These precisely orchestrated organizational shifts are critical to completing the division cycle to guarantee viable

offspring. The role of kinases in organellar dissociation and cellular rearrangements for mitotic entry is well understood, but how this phosphorylation is restored mechanistically is rarely documented in the journal. In upcoming years, a better knowledge of the regional roles of phosphatases will be a major challenge. The knowledge about the contribution and organization of splicing speckles, stress granules and centrioles on membrane-less organelles in cell biology at the time of division is very limited[304]. According to some current investigations, the protein kinase DYRK3 works as mitotic dissolves assisting in the solubilization of several of the above-mentioned structures. However, it is yet unknown why these structures must be disassembled during division[305-307]. Even though the knowledge of intracellular compartment inheritance is growing, our comprehension of the molecular pathways driving the equitable distribution of organelles among two daughter cells is still lacking to answer many critical and fascinating topics. It will be hard to define membrane and organelle movements in cell division without knowing how they associate with membrane-bound organelles that are similarly characterized by weak collective interactions within the membrane.

CONCLUSION AND FUTURE PROSPECTS

The cytoskeleton and different signaling cascades drive the cells carefully to strategize their genetic matter division and their physical partitioning. But still, there are enumerable questions that remain about this topic. The disassociation, separation and reassociation of ER, Golgi and nuclear envelope-like larger organelles are carefully controlled. But previously it was believed that mitochondria and an endo-lysosomal system like smaller organelles were partitioned stochastically. In recent studies, it is revealed that they are also partitioned actively. In the coming years, it will be extremely difficult to combine this knowledge to comprehend how the remodeling of various organelles is linked and how changes in organelle function and structure affect the cell division mechanism. Cells have developed complicated organelle inheritance systems to achieve productive intracellular organelle sharing. Below, we outline several significant areas and breakthroughs that will revolutionize our knowledge of this field and illuminate the theoretical advancements required to achieve these breakthroughs.

Understanding the kinetics of biological membranes during cell division is crucial for both plant and animal biologists. Membranes are crucial for preserving the integrity of cells and controlling molecular transport, both of which are necessary for cell division. Plant processes such as cytokinesis, in which the membrane's behaviour directs the development of a new cell wall, are influenced by membrane dynamics. Understanding membrane dynamics during cell division also provides insights into haploid induction in maize, where abnormality in membrane behavior can lead to the formation of haploid seeds. For animal cells to divide equally among their daughter cells during mitosis, membrane alterations are essential. Biologists can learn more about how aberrations in membrane behaviour may result in illnesses or defects in development by examining these kinetics. Furthermore, by using this knowledge, specific interventions may be developed to rectify these disturbances, which will ultimately increase our capacity to control cell division for a variety of purposes, including medical therapy and agricultural improvement.

SIGNIFICANCE STATEMENT

Since cell division is an essential process for the survival and reproduction of eukaryotic cells, this review is important because it offers a thorough synthesis of current information on the dynamics of biological membranes during this process. Reviewing the relationship between these components and their effects on cell division, the paper focuses on lipid composition, membrane compartmentalization and the processes behind organelle remodelling, distribution and inheritance. It also highlights how important cell-cycle proteins are in controlling and preserving the changes between mitosis, cytokinesis and interphase. Discovering the intricacies of cellular reproduction requires an understanding of these dynamics, which has ramifications for cell biology, genetics and related disciplines of study.

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