



HAL
open science

Septin Organization and Dynamics for Budding Yeast Cytokinesis

Maritzaida Varela Salgado, Simonetta Piatti

► **To cite this version:**

Maritzaida Varela Salgado, Simonetta Piatti. Septin Organization and Dynamics for Budding Yeast Cytokinesis. *Journal of Fungi*, 2024, 10, pp.642. 10.3390/jof10090642 . hal-04732704

HAL Id: hal-04732704

<https://hal.science/hal-04732704v1>

Submitted on 11 Oct 2024

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Distributed under a Creative Commons Attribution 4.0 International License

Review

Septin Organization and Dynamics for Budding Yeast Cytokinesis

Maritzaida Varela Salgado [†]  and Simonetta Piatti ^{*} 

CRBM (Centre de Recherche en Biologie cellulaire de Montpellier), University of Montpellier, CNRS UMR 5237, 34293 Montpellier, France; maritzaida.varela@ijm.fr

^{*} Correspondence: simonetta.piatti@crbm.cnrs.fr

[†] Current address: Institut Jacques Monod, 15 rue Héléne Brion, 75013 Paris, France.

Abstract: Cytokinesis, the process by which the cytoplasm divides to generate two daughter cells after mitosis, is a crucial stage of the cell cycle. Successful cytokinesis must be coordinated with chromosome segregation and requires the fine orchestration of several processes, such as constriction of the actomyosin ring, membrane reorganization, and, in fungi, cell wall deposition. In *Saccharomyces cerevisiae*, commonly known as budding yeast, septins play a pivotal role in the control of cytokinesis by assisting the assembly of the cytokinetic machinery at the division site and controlling its activity. Yeast septins form a collar at the division site that undergoes major dynamic transitions during the cell cycle. This review discusses the functions of septins in yeast cytokinesis, their regulation and the implications of their dynamic remodelling for cell division.

Keywords: *S. cerevisiae*; septins; cytokinesis



Citation: Varela Salgado, M.; Piatti, S. Septin Organization and Dynamics for Budding Yeast Cytokinesis. *J. Fungi* **2024**, *10*, 642. <https://doi.org/10.3390/jof10090642>

Academic Editors: Jian-Qiu Wu and Erfei Bi

Received: 26 July 2024

Revised: 30 August 2024

Accepted: 31 August 2024

Published: 9 September 2024



Copyright: © 2024 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

1. Introduction

Septins form an evolutionarily conserved family of cytoskeletal proteins expressed in many eukaryotic organisms but absent in land plants [1,2]. Septins were initially discovered in budding yeast through mutagenesis screens aimed at identifying genes involved in the cell division cycle (*CDC* [3]). Temperature-sensitive mutations affecting the genes *CDC3*, *CDC10*, *CDC11*, and *CDC12* cause hyperpolarized growth and cytokinesis failure at high temperatures, suggesting a role in cell morphogenesis and cell division [4,5]. Electron and immunofluorescence microscopy studies revealed that the products of these genes localize at the bud neck, forming a filamentous collar [6–8]. Given their role in septation, these proteins were named “septins”. Subsequently, three additional septin genes were identified in budding yeast based on sequence homology: *SHS1*, expressed like the aforementioned septins in vegetative cells, and *SPR3* and *SPR28*, which are expressed exclusively during meiosis [9–11].

Yeast septins participate in a wide range of cellular functions, including cell polarity [12], cell cycle progression [13–15], spindle positioning [16], cytokinesis and septum formation [4,17–19]. Since their discovery in budding yeast, septin orthologs have been identified in animals, fungi and protists, where they are involved in numerous cellular processes, such as cytokinesis [20–25], exocytosis [26], phagocytosis [27], morphogenesis [28–32], ciliogenesis [33–35], cell motility [36,37], spermatogenesis [38], lipid metabolism [39,40], synaptic activity [41–43] and bacterial entrapment during cell infection [44,45].

Given their pivotal roles in cellular organization and function, it is not surprising that septins are implicated in a range of pathological conditions. Indeed, septin dysfunction has been associated with cancer, neurodegenerative disorders, infectious diseases and infertility (reviewed in [46]).

Septins are GTP-binding proteins characterized by a conserved GTPase domain and variable N-terminal and C-terminal regions. A septin-unique element is a distinctive feature of all septins, although its precise function remains unknown [47]. Septins invariably assemble

into rod-shaped hetero-complexes composed of two copies of each septin monomer organized in a palindromic fashion (reviewed in [48]). Septin hetero-complexes then interact to form non-polar filaments and/or higher-order structures, such as bundles, rings or gauzes, as observed both in vitro and in vivo across various organisms [6,22,24,31,38,49–60].

Membranes play a crucial role in septin organization. Septins associate with lipid membranes containing negatively charged phospholipids, particularly phosphatidylinositol 4,5-bisphosphate (PI(4,5)P₂) [61–65]. In budding yeast, PI(4,5)P₂ is enriched at the presumptive bud site in G1 [66], thus accounting for septin recruitment at this specific cellular location. Additionally, perturbing the amount of PI(4,5)P₂ at the plasma membrane causes the septin collar to be disassembled at the bud neck and/or septin filaments to form aberrant structures cytoplasm, such as small rings and arcs [64,67], indicating that PI(4,5)P₂ is crucial for septin organization in vivo.

How septins bind to membranes is unclear. A protein motif rich in basic residues at the N-terminus of septins has been implicated in membrane binding via electrostatic interactions with the negative charges of phospholipids [63,68–70]. However, whether this polybasic motif is actually exposed on the surface of septins or buried, thus precluding a prominent role in membrane binding, is still debated [48].

Septin binding to membranes is also affected by membrane curvature, showing a strong preference for micrometric curvatures [71–73]. Consequently, septins are frequently located at curved membranes, such as the hyphal branch sites of filamentous fungi, the base of primary cilia, and the annulus of sperm tails [31–33,50,74,75].

Finally, multiple septin-associated proteins and post-translational modifications are likely involved in septin organization in different physiological contexts, highlighting the complexity and dynamic nature of septin regulation.

2. Budding Yeast Septins and Their Localization

In vegetative cells, budding yeast septins form two hetero-octamers with a common core composed of Cdc12-Cdc3-Cdc10-Cdc10-Cdc3-Cdc12 and comprising either Cdc11 or Shs1 at both extremities [58,59,76]. Surprisingly, septin octamers always have symmetric termini, while asymmetric complexes capped by Cdc11 and Shs1 at each end cannot be formed, at least in vitro [76]. The linear septin complexes are often referred to as septin “rods” and measure 32–35 nm in length and 4–5 nm in diameter [58,59]. In vitro reconstitution assays using recombinant septins have shown a striking difference between Cdc11-capped and Shs1-capped yeast octamers, where the former can spontaneously polymerize end-to-end to form long, paired filaments in solution, while the latter appear unable to polymerize [50,58,59,77,78]. Shs1-capped octamers can nevertheless interact laterally and stagger on top of each other to form curved bundles, rings and spirals in vitro [59]. Additionally, in the presence of lipids or upon an *SHS1* mutation mimicking constitutive phosphorylation of Ser259, Shs1-capped octamers form gauze-like structures in addition to rings, suggesting that the cellular context and post-translational modifications influence the supramolecular organization of septins [59,60]. Despite being unable to polymerize into linear filaments, Shs1-capped rods can form heterotypic interactions with the termini of Cdc11-capped octamers [78], suggesting that they may be interspersed in septin filaments and higher-order structures. Consistently, the five mitotic septins display a completely superimposable localization in vegetative cells throughout the cell cycle.

Formation of septin filaments is essential for cell division, as shown by the lethality of mutants incapable of septin polymerization [79]. This may explain why Cdc11 is required for viability in most yeast backgrounds, while Shs1 is dispensable [10,47,79]. During sporulation, the aforementioned septin hetero-octamers are replaced by the Spr28-Spr3-Cdc3-Cdc10-Cdc10-Cdc3-Spr3-Spr28 meiotic-specific complex, where Spr28 and Spr3 replace the terminal septin and Cdc12, respectively [80]. Unlike its mitotic Cdc11-capped counterpart, the Spr28-capped meiotic complex is unable to polymerize in solution, but it readily forms filaments on PI(4,5)P₂-containing lipid monolayers [80], suggesting that its polymerization requires binding to the plasma membrane. Its function during meiosis

is linked to proper biogenesis of the prospore membrane and cell wall assembly, thereby ensuring high sporulation efficiency [81].

The functional organization of septins is based on their assembly into higher-ordered structures. In vegetative cells, septins undergo dynamic changes in their structural organization during the cell cycle. First, in late G1, septins form a dynamic cortical ring at the presumptive bud site. Then, as the bud emerges, the septin ring expands into an hourglass-shaped structure, referred to as septin collar, around the bud neck. Finally, at the onset of cytokinesis, the septin collar splits into a double ring that sandwiches the AMR (Figure 1). This dramatic septin remodelling, which is essential for cell division (see below), is often referred to as septin ring splitting. Each split ring then persists in the mother and daughter cell, respectively, until the next cell cycle [7,82,83]. Florescence recovery after photobleaching (FRAP) experiments have shown that the newly forming septin ring is dynamic, as shown by its ability to exchange septins from an unassembled pool. In contrast, the septin collar is a stable and immobile structure. At cytokinesis, split septin rings become again relatively dynamic [84,85].

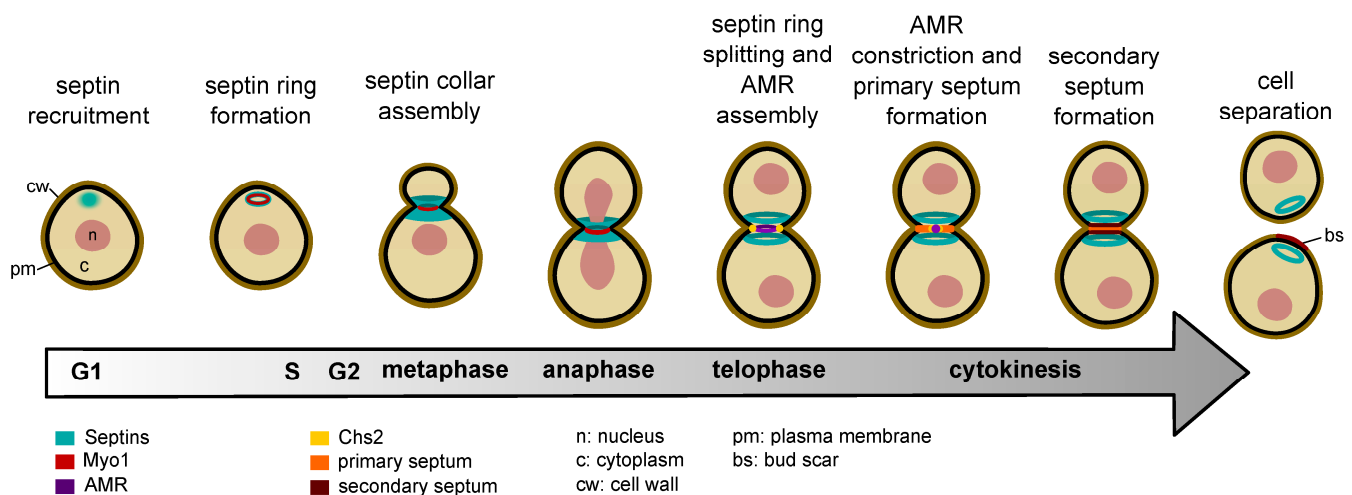


Figure 1. Main cell cycle events relevant for budding yeast cytokinesis and cell division. See text for details.

In cells exposed to mating pheromones, septins form a fuzzy band or a set of parallel bars at the base of the polarized projection (i.e. the shmoo) [7,83,86]. Upon cell fusion during mating, septins form an annulus at the midzone of the zygote that seems to affect the redistribution of supramolecular complexes and organelles [87].

During meiosis I, septins first form ring-like structures at the leading edge of membrane sacs, also known as prospore membranes, that originate in close apposition to the cytoplasmic face of the spindle pole bodies and that will later extend to engulf each of the four haploid nuclei. At the end of meiosis II, septins seem to localize quite uniformly at the plasma membrane surrounding the developing spore [9,88] (Figure 2). Interestingly, the septin Cdc10 accumulates in each mature spore to form a cortical cluster opposite to its neighboring sister spores in the ascus. This septin cluster serves as a prepolarity marker to direct the later polarised growth to penetrate the ascus wall during germination [89,90] (Figure 2). Additionally, upon germination but before bud emergence, Cdc10 localises as a band at the boundary between the two unequal halves of the germinating spore, suggesting that septins may form a cortical barrier between the growing and non-growing parts of the germinating spore [90].

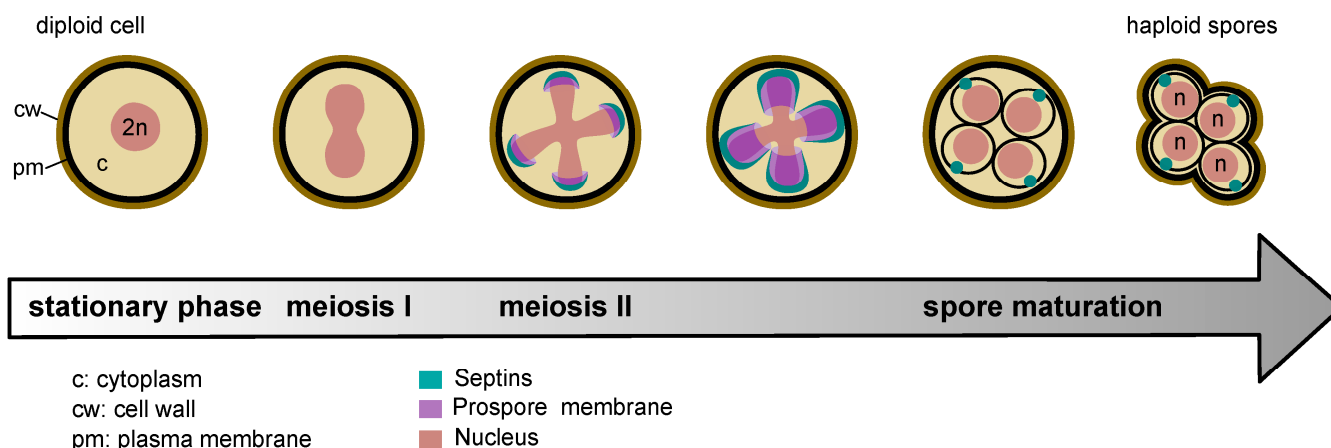


Figure 2. Septin localization during meiosis in budding yeast. Upon nitrogen deprivation and in the presence of poor carbon sources, budding yeast diploid cells stop dividing (stationary phase) and undergo meiosis. See text for details.

Thus, budding yeast septins display a wide range of high order architectures depending on the physiological context, but they seem to be invariably associated with membranes, while in other eukaryotes, septins clearly also associate with the actin and microtubule cytoskeleton (reviewed in [91]).

3. Septin Organization and Cytokinesis in Budding Yeast

Our current knowledge of septin architecture at the bud neck of mitotic yeast cells comes mainly from ultrastructural studies. Freeze-fracture and platinum-replica electron microscopy on unroofed spheroplasts or cryo-electron tomography on intact cells have shown that the mature septin collar at the bud neck is made by a network of axial filaments lying parallel to the mother bud axis and circumferential filaments that are orthogonally oriented, thus forming a gauze-like meshwork (Figure 3) [49,52,92]. Conversely, the septin double ring at cytokinesis is made exclusively of circumferential filaments [92], in line with polarized fluorescence microscopy studies showing that the average orientation of septin filaments in the split septin rings is perpendicular relative to the polarity axis [93–95]. Since fluorescently labelled septins show a marked decrease in fluorescence at the bud neck at the moment of septin ring splitting [18,85,94,96], current models envision that at the onset of cytokinesis the axial septin filaments disassemble and possibly partially reassemble into two arrays of circumferential filaments, forming the double ring (reviewed in [97]).

Cytokinesis is a delicate process that must precisely partition an equal complement of the replicated genomes and organelles to each daughter cell. It relies on assembly and contraction of an actomyosin ring (AMR) and must be tightly controlled and coordinated with other cell cycle processes, such as chromosome segregation. Typically, yeast cytokinesis can be divided into four steps: selection of the division site, assembly of the AMR, cleavage furrow ingression and primary septum formation powered by the AMR, and localized membrane remodelling. In budding yeast, septins are involved in most of these cytokinetic steps by adopting different structural organizations.

The first steps of cytokinesis occur very early during the cell cycle. The selection of the bud site, which is also the division site, is determined in late G1 by the cell polarization machinery controlled by the master polarity GTPase Cdc42 that also recruits septins (reviewed in [98]; see below). Bud site selection follows a very specific pattern depending on whether cells are haploid or diploid. In haploid cells, the emergence of a new bud occurs proximally to the division site (axial budding pattern), while in diploid cells, it occurs distally (bipolar budding pattern). Such a stereotypical pattern depends on polarity landmark proteins that precisely mark the future bud site (reviewed in [98]). Despite their recruitment before bud emergence, septins are not required for bud emergence [4,99], but they contribute to the

axial budding pattern by recruiting to the bud neck landmark proteins, such as Bud3 and Bud4 [100–103]. In addition, they facilitate the formation of a narrow cluster of Cdc42 at the presumptive bud site by recruiting GTPase-activating proteins (GAPs) that in turn inhibit Cdc42 in the membrane area surrounding its activated cluster (Figure 3) [104].

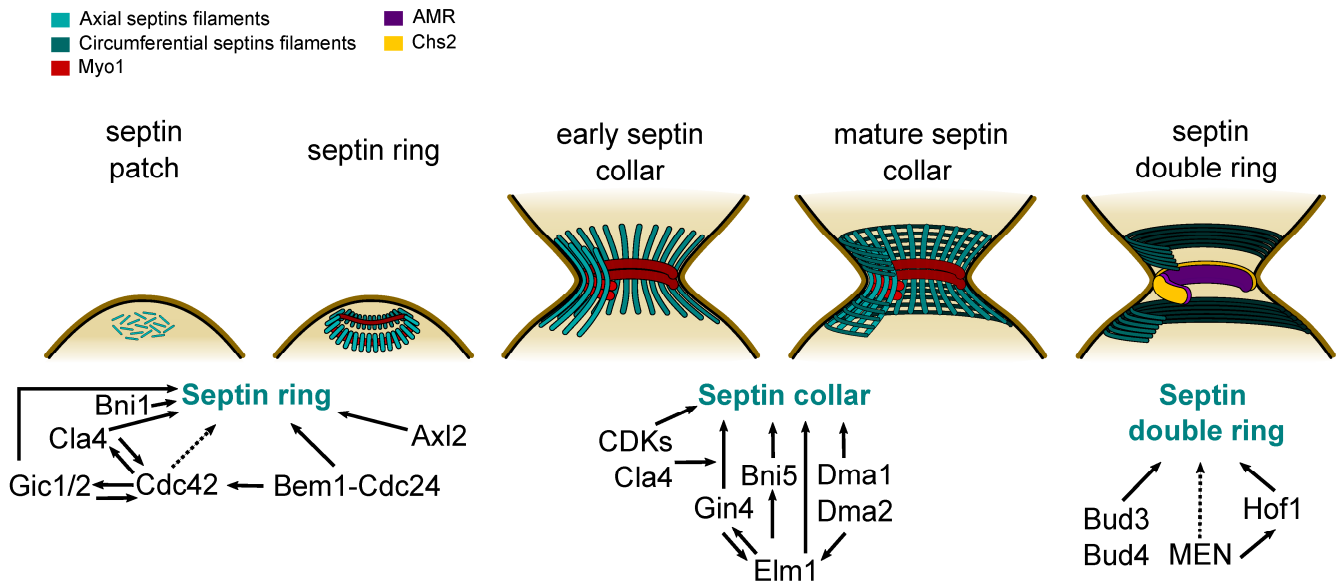


Figure 3. Septin architecture during different cell cycle phases. Septins are first recruited in G1 to the presumptive bud side as an unorganized patch that is rapidly converted into a cortical ring formed by radial septin filaments. Septin recruitment and/or ring formation involve the Cdc42 GTPase, in turn activated by the scaffold Bem1 and the GEF Cdc24, the Cdc42 effectors Cla4 and Gic1-2, the formin Bni1 and the axial landmark protein Axl2. At S phase entry, the septin ring expands into a septin collar that spans the bud neck and then matures into an hourglass-shaped structure formed by axial and circumferential septin filaments. Septin collar assembly involves the kinases Cla4, Gin4 and Elm1, CDKs, the septin-interacting protein Bni5 and the ubiquitin ligases Dma1 and Dma2. At the onset of cytokinesis, the axial septin filaments are depolymerized and possibly repolymerized into two arrays of circumferential filaments that make a double ring sandwiching the constricting AMR. Septin reorganization requires the MEN (mitotic exit network), its phosphorylation target Hof1 and other unidentified effectors. The septin double ring is patterned and stabilised by the Bud3 protein and the anillin-like Bud4. Solid arrows indicate established regulatory relationships; dashed arrows indicate hypothetical regulatory interactions. See text for further details.

Before bud emergence, Myo1, the sole myosin-II heavy chain in budding yeast, is recruited along with its regulatory light-chain Mlc2 in a septin-dependent manner, forming a ring (Figure 1) [19,105–107]. At the time of the bud emergence, the septin ring expands around the bud neck, forming an hourglass-shaped structure that remains stable during mitosis. AMR assembly proceeds until the end of anaphase (Figure 1), when the IQGAP Iqg1 appears at the bud neck to recruit actin [19,105,108,109]. Around the same time, the essential myosin light chain Mlc1 arrives at the bud neck to interact with Myo1 and to enhance Myo1 targeting to the neck, thus further increasing its local levels [106,110]. Concomitantly, Mlc1 also promotes Iqg1 recruitment to the bud neck by binding to its IQ motifs [111]. Two formins, Bni1 and Bnr1, are redundantly essential for actin polymerization at the AMR [112,113]. Furthermore, formins have been shown to contribute to the accumulation of Mlc1 at the bud neck during cytokinesis [114], suggesting an additional mechanism by which they could participate in AMR assembly. After mitotic exit, the chitin synthase Chs2 is recruited to the AMR to promote formation of the primary septum along with AMR constriction [115–119]. Additionally, a protein complex made by Hof1, Inn1 and Cyk3 also joins the AMR and partially constricts alongside it to boost Chs2 recruitment and

activity [113,116,120–128]. Several of the aforementioned AMR components and regulators, namely Myo1 [19,105], Mlc1 [106,108], Bnr1 [129,130] and Hof1 [128], require septins for their bud neck localization, suggesting that the septin collar orchestrates AMR assembly.

Constriction of the AMR marks the beginning of cytokinesis (Figure 1). AMR constriction may occur partly through sliding of bipolar myosin filaments along actin filaments that are tethered to the plasma membrane, similar to the way by which actomyosin generates force in the striated muscle [131,132]. However, actin depolymerization by cofilin plays a predominant role in budding yeast AMR constriction [133]. Iqg1 degradation by the APC/C complex might also contribute to AMR disassembly [134,135].

AMR constriction drives membrane invagination inward and is also coupled to targeted deposition of post-Golgi vesicles to the division site to increase surface area and deliver the chitin synthase-II Chs2 to drive primary septum formation (PS) [118,136,137]. Thus, AMR constriction guides PS formation, which in turn stabilises the AMR during constriction [138–140].

At the onset of cytokinesis, the septin hourglass is split into two rings that sandwich the AMR (Figure 1) [82,96,141]. This septin reorganization is an essential prerequisite for cytokinesis [96,142]. Indeed, lack of septin ring splitting prevents AMR constriction and PS formation. Thus, the septin collar has both a positive and a negative role in cytokinesis: during mitosis, it organises the cytokinetic machinery by recruiting several cytokinetic proteins to the bud neck, but at cytokinesis, it must be displaced from the bud neck (through septin ring splitting or clearance altogether) to allow AMR constriction [96,142]. Consistently, septins are not required for AMR constriction once the AMR has been assembled [96,143]. The septin double ring could nevertheless facilitate plasma membrane closure by acting as a diffusion barrier that concentrates membrane remodelling factors, such as the polarisome and the exocyst complex, to the cleavage site [143].

Once the primary septum has been laid down, a secondary septum (SS) is assembled by glucan synthases and the chitin synthase Chs3 on both the mother and daughter sides of the PS, thus leading to abscission (Figure 1) [125,136]. By recruiting Hof1 to the bud neck, which in turn inhibits precocious activation of Chs3, septins indirectly set the timing of SS assembly so that it occurs only after the PS has been deposited [121,144].

The final step of yeast cytokinesis is cell separation (Figure 1), which involves PS digestion and SS remodeling by chitinase and endoglucanases, respectively (reviewed in [145]). If and how septins are involved in the control of cell separation is not known. Intriguingly, however, in fission yeast glucanases are localized in between the two split septin rings and are unable to form a proper ring at the cortex in septin mutants [146], suggesting that the septin double ring may form a diffusion barrier for the enzymes involved in cell separation.

4. The Control of Septin Dynamics during the Cell Cycle

We can distinguish three main steps in the dynamics of septin architecture during the budding yeast cell cycle: (1) septin recruitment and ring assembly; (2) maturation into the septin collar; and (3) transition into the septin double ring.

4.1. Septin Recruitment and Ring Assembly

Septins accumulate at the presumptive bud site initially as a cloud or patch (Figure 3). This process depends on the Rho GTPase Cdc42, which is a conserved master regulator of cell polarity [82,84,147]. Indeed, Cdc42 depletion leads to an unbudded cell phenotype lacking cortical septin structures [148], and *cdc42* mutants are defective in septin recruitment to the presumptive bud site or display septin mislocalization [103,147,149]. However, in some of these mutants Cdc42 cannot hydrolyze GTP and is locked in a GTP-bound state, while overexpression of Cdc42 GTPase-activating proteins (GAPs) can restore proper septin localization and function in septin mutants [147,148]. These observations have led to the proposal that Cdc42 must continuously cycle between a GTP- and a GDP-bound state in order to gather septins at the future bud neck [149]. Alternatively, active GTP-bound Cdc42

may be required to establish the first polarity cue necessary for septin recruitment (e.g., to define a membrane domain permissive for septin accumulation), while the active GTPase actually prevents septins from piling up.

Once at the plasma membrane, septins rapidly organise into a cortical ring (Figure 3). Polarized exocytosis and insertion of membrane vesicles by Cdc42 and the exocyst complex are critical to this process by inhibiting septin accumulation in the center of the septin cap at the incipient bud site, thus creating the ring [104]. Thus, it is possible that the septin mislocalization phenotype of some GTP-bound *cdc42* mutants is accounted for by excessive or untargeted membrane vesicles tethering that disrupts septin ring assembly.

Several studies have attempted to link the function of Cdc42 in septin ring assembly to specific Cdc42 effectors (Figure 3). The p21-activated kinases (PAK) Cla4 and Ste20 are turned on by Cdc42 and Cla4 has been implicated in septin recruitment to the presumptive bud site through phosphorylation of several septins [99,150–152]. On the other hand, Cla4 binds the Cdc42 membrane scaffold Bem1 and recruits and phosphorylates Cdc24, the GEF (guanine-nucleotide exchange factor) for Cdc42, thus promoting its GTP-bound state [153–155]. Such a positive feedback loop suggests that the role of PAK kinases in septin recruitment may be partly mediated by a global enhancement in Cdc42 activity. In parallel, the Cdc24-Bem1 complex binds Cdc11, helping to bring septins to the bud site [156]. Two additional Cdc42 effectors, i.e. the paralogous membrane proteins Gic1 and Gic2, interact directly with septins and are involved in septin recruitment and organization in vivo and in vitro [104,148,157–159]. Accordingly, septin deposition and budding mostly fail in *gic1 gic2* double mutants at high temperatures [148]. Gic1 colocalizes with septins at the presumptive bud site at early stages of the cell cycle and at the bud neck later on [148], and it has been shown to bundle and cross-link septin filaments in vitro, thereby stabilizing them [159]. However, similar to PAK kinases, Gic1 and Gic2 are also involved in a positive feedback loop for Cdc42 activation, and overexpression of *CDC42* suppresses the lethality of cells lacking Gic1 and Gic2 at high temperatures [160,161], suggesting that septin recruitment to the future bud site may be empowered by Cdc42 itself or by effectors other than Gic proteins.

PAK kinases possess a pleckstrin-homology domain that binds preferentially to PI(4,5)P₂ [162]. Similarly, Gic2 (and possibly Gic1) bears a cluster of basic residues that binds to PI(4,5)P₂ [158]. Thus, the PI(4,5)P₂ binding properties of these Cdc42 effectors might influence their interaction with and organization of septin into a ring.

The formin Bni1 contributes to septin ring formation along with Cla4 (Figure 3), as *bni1 cla4* double mutants form a cap of septins at the presumptive bud site that is not converted into a ring [151]. This could be related to the function of Cdc42 in polarized exocytosis along actin cables generated by Bni1 and in creating the hole in the middle of the septin ring [104].

Finally, Axl2 is a bud site landmark protein that also participates in septin recruitment (Figure 3) [163,164]. Recent two-hybrid and bimolecular fluorescence complementation (BiFC) assays indicated that Axl2 interacts with the septin Cdc10, as well as with Cdc42-GTP and Bud3, and contributes to the efficient gathering of septins to the cell division site [103].

In summary, while Cdc42 effectors, activators and cell polarity proteins have been implicated in septin organization, the precise mechanistic processes underlying septin recruitment and septin ring formation need to be further elucidated.

Another Rho GTPase, Rho1, has been implicated in timely and efficient septin recruitment and septin collar stability through activation of one of its downstream effectors, the kinase Pkc1 that in turn phosphorylates the septin-interacting protein Syp1 and promotes its turnover at the bud neck [165,166]. Syp1 appears at the presumptive bud site concomitant with septins and in vitro is able to align laterally and bundle septin filaments, thus offering mechanical stability to the new-forming septin ring/collar [167].

4.2. Maturation into the Septin Collar

As the bud emerges, the septin ring at the bud neck expands into a rigid hourglass-shaped septin collar. Whether this septin remodeling occurs suddenly at the G1/S transition or progressively throughout mitotic progression is not known. As mentioned above, the mature septin hourglass is a gauze-like, very stable septin assembly comprising axial and circumferential filaments (Figure 3) [49,84,85,92]. Likely, the transition from the initial septin ring, which is mainly composed by radial septin filaments converging towards a central hole [92], into the septin collar is controlled by the cell cycle machinery.

One of the first septin-interacting proteins that was found to be involved in septin organization is the Nim-related kinase Gin4. Mutations in *GIN4* are synthetically lethal with *cdc12* and cause septins to form a fuzzy band or parallel axial “bars” at the bud neck instead of the hourglass [86]. A similar phenotype was observed in cells lacking the Gin4-binding protein Nap1 [14] or the septin Shs1 [13] and in cells exposed to mating pheromones [7,83,86]. These observations suggest that Gin4 may be involved in stabilising the circumferential septin filaments, which in turn require Shs1 for the overall gauze-like organization in a septin collar [92,94]. Consistently, Gin4 phosphorylates directly Shs1, thus contributing to the robustness of the hourglass [168,169]. Gin4 colocalises with septins from septin appearance throughout mitosis, but is displaced from the bud neck before septin ring splitting and is absent in septin structures of mating cells [86]. Its association with septins requires Nap1, Shs1, the PAK kinase Cla4 and cyclin-dependent kinases (CDKs) [168], thus partly explaining the links between septin collar organization and cell cycle progression. Accordingly, Shs1 phosphorylation by G1 CDKs stabilises the interaction of septins with Gin4 [170]. Two additional Nim-related kinases, Hsl1 and Kcc4, associate with septins in yeast cells [171–177]. Although the lack of Hsl1 or Kcc4 does not cause any obvious defect in septin organization [14], these kinases may share with Gin4 partially overlapping functions in building up the architecture of the septin collar [174].

During bud emergence, Gin4 targets to the bud neck the LKB1-like kinase Elm1, which contributes to septin collar architecture and stability until cytokinesis (Figure 3) [178–181]. In turn, Elm1 phosphorylates and activates Cla4, Gin4 and Hsl1 [14,168,169,180–182]. Its efficient recruitment to the bud neck requires the Dma1 and Dma2 ubiquitin ligases, which in turn are involved in proper septin stability at the bud neck through an unknown molecular mechanism [183,184]. The role of Elm1 in septin organization also involves the phosphorylation and functionality of Bni5 [179,185], a septin-interacting protein that was identified as a dosage suppressor of septin mutants [186]. Bni5 can dimerize and in vitro is able to crosslink septin filaments into networks by bridging pairs or multiple filaments [185,187]. Additionally, Bni5 interacts with the C-terminal extension of Cdc11 and Shs1 and recruits Myo1 to the septin ring and collar throughout most of the cell cycle except during cytokinesis [110,188]. The exact role of Myo1 at the septin collar is unclear, but myosin II filaments may assemble on the membrane-distal side of the septin hourglass perpendicular to the axial septin filaments and somehow contribute to the appropriate septin architecture [92].

Post-translational modifications (PTMs) likely play an important role in the organization and stability of the septin hourglass. Besides the aforementioned septin phosphorylations by Cla4, Gin4 and CDKs, septins may be phosphorylated by other septin- and bud neck-associated kinases (e.g. Hsl1, Kcc4, polo kinase Cdc5, Mob1-Dbf2, etc.). Plenty of septin phosphorylation sites have been identified by phosphoproteomics, and some of them are regulated across the cell cycle [189–198], suggesting that they may play an important role in the control of septin dynamics. However, their elevated number has so far hampered their systematic characterization. Septin SUMOylation occurs mainly during mitosis and could potentially promote the stability of the septin hourglass or septin interactions [199]. However, non-SUMOylatable septin mutants do not show any obvious defects in septin architecture and experience only a mild delay in septin disassembly after cell division [199]. Septin acetylation may also impact the overall architecture of the septin collar, as septin mutants with reduced acetylation levels have defects in septin localization [200]. Finally,

some septins have also been found to be ubiquitylated [191], but the functional significance of this PTM has not been explored so far.

Since the septin collar is necessary for the recruitment of several cytokinetic proteins to the bud neck (see above), it is often considered as a “scaffold” for cytokinesis. However, septins actually inhibit AMR constriction and are no longer required for cytokinesis after AMR assembly [96,143]. Thus, whether the term “scaffold” is appropriate may be questionable. Why certain cytokinetic factors require to bind the septin collar in order to be incorporated into the AMR remains to be further investigated. Possibly, the septin collar congregates these proteins at the bud neck to reach threshold concentrations necessary for AMR assembly, thus acting as a molecular crowder. A non-mutually exclusive hypothesis is that binding to septins may induce specific protein conformations that are compatible with AMR interactions.

4.3. Transition into the Septin Double Ring

During cytokinesis, the septin collar undergoes a sudden and dramatic reorganization, resulting in its splitting into two distinct rings that sandwich the AMR (Figure 3) [105,141]. This septin displacement is crucial for AMR constriction and cytokinesis, indicating that building a force-generating AMR requires at least two septin-related steps. Initially, the septin collar spurs the assembly of the cytokinetic machinery at the right place while preventing AMR-driven membrane ingression; subsequently, the confined eviction of septins from the division site during septin ring splitting initiates AMR constriction [96,142].

Formation and maintenance of the double septin ring depend on the anillin homologue Bud4 and the Rho-GEF Bud3 (Figure 3) [18,94,201–203], which have been previously implicated in the axial budding pattern of haploid yeast cells [101,202,204]. In the absence of Bud3 and Bud4 the septin collar disassembles completely at mitotic exit, without any obvious consequence on the kinetics of AMR constriction and cytokinesis [201], in line with the notion that septin clearance after AMR assembly is not detrimental for cytokinesis. Both Bud3 and Bud4 localise as a double ring at the edges of the septin collar during mitosis and then remain associated with the split septin rings, suggesting that these proteins act as spatial cues to pre-pattern the septin double ring [100,101,201,205–207].

Septin ring splitting requires the activity of the mitotic exit network (MEN; Figure 3), a Hippo-like pathway triggered by a top GTPase (Tem1) that turns on two sequentially acting protein kinases (Cdc15 and Mob1-Dbf2) to ultimately promote the activation of the Cdc14 phosphatase (reviewed in [208]). In turn, Cdc14 dephosphorylates and activates its upstream kinases Cdc15 and Mob1-Dbf2 in a positive feedback loop [209,210]. Cdc14 is the main CDK-counteracting phosphatase in budding yeast, and its function, together with that of its upstream MEN regulators, is essential for mitotic exit and cytokinesis [211–216]. Mitotic exit elicits several processes, such as spindle disassembly and licensing of replication origins, and it is an essential prerequisite for cytokinesis through dephosphorylation of specific mitotic CDK substrates [217]. However, MEN also promotes septin ring splitting independently of its role in mitotic exit [96], suggesting that one or more MEN targets prompt septin remodelling at cytokinesis. We have recently identified the cytokinetic protein Hof1 as one of such septin ring splitting regulators (Figure 3) [218]. Hof1 is involved in cytokinesis and is an established target of MEN [113,128,219,220]. It plays a major role in the control of actin polymerization and bundling, partly by modulating formin activity [221–226]. Consistently, it has been implicated in polarised growth [224,227]. However, the cytokinetic function of Hof1 was mainly linked to activation of the chitin synthase Chs2, and *CHS2* overexpression or gain of function mutations can suppress the lethality of *hof1Δ* mutants at high temperatures [113,127]. Our recent data indicate that *hof1* mutants also display defects in septin remodeling at cytokinesis [218]. During mitosis, Hof1 associates with septins, forming two closely spaced rings at the edges of the septin collar [128,218,219,228]. In vitro, it can induce the formation of intertwined septin bundles, suggesting that during this cell cycle stage it may contribute to the robustness of the septin hourglass [218,225]. However, shortly before septin ring splitting, Hof1 is displaced from the septin collar and relocates to the AMR, where it partially constricts alongside

it [128,218,219,228]. This rapid translocation from septins to the AMR is triggered by Hof1 phosphorylation, primarily by the MEN kinase Dbf2, which disengages Hof1 from the septins, allowing it to join the AMR [218–220]. Phospho-mimicking *HOF1* mutant alleles can bypass the septin reorganization defects seen in MEN mutants by displacing Hof1 from septins and enhancing its translocation to the AMR. Importantly, septin remodeling by Hof1 depends on its membrane-binding F-BAR domain, suggesting that a local membrane reorganization could underlie septin disassembly from the cleavage site and remodelling into the double ring [218]. Since BAR domains can induce membrane curvature [229,230], Hof1 may bend the membrane at the division site to a radius that causes septin disassembly. Another possibility, stemming from the ability of BAR domains to cluster phosphoinositides [229,230], is that Hof1 may promote septin ring splitting by modifying the local composition of the plasma membrane. The non-essentiality of *HOF1*, especially in some strain background, suggests that additional, as-yet unidentified proteins participate in septin ring splitting alongside Hof1.

5. Conclusions and Perspectives

Cytokinesis is a fundamental yet vulnerable process that requires the orchestration of many players. Given the prominent role of septins in budding yeast cytokinesis, understanding how septin remodeling is controlled during the yeast cell cycle is key in order to gain insights into how cytokinesis is regulated in time and space. Unresolved questions that deserve further investigation regard the precise mechanism underlying septin membrane interactions, the impact of septin-binding proteins and post-translational modifications on septin structural organization, and the possible interplay between septins and other cytoskeletal elements. Unravelling how the yeast septin collar inhibits AMR constriction is another critical question for future research. As we continue to tease apart the complexity of septin biology, their integral role in cellular organization and function becomes increasingly apparent, opening exciting avenues for our comprehension of septin functions in physiological and pathological conditions.

Funding: Work in Piatti's lab is supported by the Agence Nationale pour la Recherche (ANR project CONSTRUCT) and Fondation pour la Recherche Médicale (FRM project EQU202303016309) to S.P.; M.V.S has been supported by Fondation pour la Recherche Médicale (FRM project FDT202106012881).

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Nishihama, R.; Onishi, M.; Pringle, J.R. New Insights into the Phylogenetic Distribution and Evolutionary Origins of the Septins. *Biol. Chem.* **2011**, *392*, 681–687. [[CrossRef](#)] [[PubMed](#)]
2. Shuman, B.; Momany, M. Septins From Protists to People. *Front. Cell Dev. Biol.* **2022**, *9*, 824850. [[CrossRef](#)] [[PubMed](#)]
3. Hartwell, L.H.; Culotti, J.; Reid, B. Genetic Control of the Cell-Division Cycle in Yeast, I. Detection of Mutants. *Proc. Natl. Acad. Sci. USA* **1970**, *66*, 352–359. [[CrossRef](#)] [[PubMed](#)]
4. Hartwell, L.H. Genetic Control of the Cell Division Cycle in Yeast. IV. Genes Controlling Bud Emergence and Cytokinesis. *Exp. Cell Res.* **1971**, *69*, 265–276. [[CrossRef](#)]
5. Hartwell, L.H.; Mortimer, R.K.; Culotti, J.; Culotti, M. Genetic Control of the Cell Division Cycle in Yeast: V. Genetic Analysis of Cdc Mutants. *Genetics* **1973**, *74*, 267–286. [[CrossRef](#)]
6. Byers, B.; Goetsch, L. A Highly Ordered Ring of Membrane-Associated Filaments in Budding Yeast. *J. Cell Biol.* **1976**, *69*, 717–721. [[CrossRef](#)]
7. Ford, S.K.; Pringle, J.R. Cellular Morphogenesis in the *Saccharomyces Cerevisiae* Cell Cycle: Localization of the CDC11 Gene Product and the Timing of Events at the Budding Site. *Dev. Genet.* **1991**, *12*, 281–292. [[CrossRef](#)]
8. Haarer, B.K.; Pringle, J.R. Immunofluorescence Localization of the *Saccharomyces Cerevisiae* CDC12 Gene Product to the Vicinity of the 10-Nm Filaments in the Mother-Bud Neck. *Mol. Cell. Biol.* **1987**, *7*, 3678–3687.
9. De Virgilio, C.; DeMarini, D.J.; Pringle, J.R. SPR28, a Sixth Member of the Septin Gene Family in *Saccharomyces Cerevisiae* That Is Expressed Specifically in Sporulating Cells. *Microbiology* **1996**, *142 Pt 10*, 2897–2905. [[CrossRef](#)]
10. Mino, A.; Tanaka, K.; Kamei, T.; Umikawa, M.; Fujiwara, T.; Takai, Y. Shs1p: A Novel Member of Septin That Interacts with Spa2p, Involved in Polarized Growth in *Saccharomyces Cerevisiae*. *Biochem. Biophys. Res. Commun.* **1998**, *251*, 732–736. [[CrossRef](#)]
11. Ozsarac, N.; Bhattacharyya, M.; Dawes, I.W.; Clancy, M.J. The SPR3 Gene Encodes a Sporulation-Specific Homologue of the Yeast CDC3/10/11/12 Family of Bud Neck Microfilaments and Is Regulated by ABFI. *Gene* **1995**, *164*, 157–162. [[CrossRef](#)] [[PubMed](#)]

12. Barral, Y.; Mermall, V.; Mooseker, M.S.; Snyder, M. Compartmentalization of the Cell Cortex by Septins Is Required for Maintenance of Cell Polarity in Yeast. *Mol. Cell* **2000**, *5*, 841–851. [[CrossRef](#)]
13. Castillon, G.A.; Adames, N.R.; Rosello, C.H.; Seidel, H.S.; Longtine, M.S.; Cooper, J.A.; Heil-Chapdelaine, R.A. Septins Have a Dual Role in Controlling Mitotic Exit in Budding Yeast. *Curr. Biol.* **2003**, *13*, 654–658. [[CrossRef](#)]
14. Longtine, M.S.; Theesfeld, C.L.; McMillan, J.N.; Weaver, E.; Pringle, J.R.; Lew, D.J. Septin-Dependent Assembly of a Cell Cycle-Regulatory Module in *Saccharomyces Cerevisiae*. *Mol. Cell Biol.* **2000**, *20*, 4049–4061. [[CrossRef](#)] [[PubMed](#)]
15. Sakchaisri, K.; Asano, S.; Yu, L.-R.; Shulewitz, M.J.; Park, C.J.; Park, J.-E.; Cho, Y.-W.; Veenstra, T.D.; Thorner, J.; Lee, K.S. Coupling Morphogenesis to Mitotic Entry. *Proc. Natl. Acad. Sci. USA* **2004**, *101*, 4124–4129. [[CrossRef](#)] [[PubMed](#)]
16. Kusch, J.; Meyer, A.; Snyder, M.P.; Barral, Y. Microtubule Capture by the Cleavage Apparatus Is Required for Proper Spindle Positioning in Yeast. *Genes. Dev.* **2002**, *16*, 1627–1639. [[CrossRef](#)] [[PubMed](#)]
17. DeMarini, D.J.; Adams, A.E.M.; Fares, H.; Virgilio, C.D.; Valle, G.; Chuang, J.S.; Pringle, J.R. A Septin-Based Hierarchy of Proteins Required for Localized Deposition of Chitin in the *Saccharomyces Cerevisiae* Cell Wall. *J. Cell Biol.* **1997**, *139*, 75. [[CrossRef](#)]
18. Wloka, C.; Nishihama, R.; Onishi, M.; Oh, Y.; Hanna, J.; Pringle, J.R.; Krauss, M.; Bi, E. Evidence That a Septin Diffusion Barrier Is Dispensable for Cytokinesis in Budding Yeast. *Biol. Chem.* **2011**, *392*, 813–829. [[CrossRef](#)]
19. Estey, M.P.; Di Ciano-Oliveira, C.; Froese, C.D.; Bejide, M.T.; Trimble, W.S. Distinct Roles of Septins in Cytokinesis: SEPT9 Mediates Midbody Abscission. *J. Cell Biol.* **2010**, *191*, 741–749. [[CrossRef](#)]
20. Joo, E.; Surka, M.C.; Trimble, W.S. Mammalian SEPT2 Is Required for Scaffolding Nonmuscle Myosin II and Its Kinases. *Dev. Cell* **2007**, *13*, 677–690. [[CrossRef](#)]
21. Karasmanis, E.P.; Hwang, D.; Nakos, K.; Bowen, J.R.; Angelis, D.; Spiliotis, E.T. A Septin Double Ring Controls the Spatiotemporal Organization of the ESCRT Machinery in Cytokinetic Abscission. *Curr. Biol.* **2019**, *29*, 2174–2182.e7. [[CrossRef](#)] [[PubMed](#)]
22. Kim, M.S.; Froese, C.D.; Estey, M.P.; Trimble, W.S. SEPT9 Occupies the Terminal Positions in Septin Octamers and Mediates Polymerization-Dependent Functions in Abscission. *J. Cell Biol.* **2011**, *195*, 815–826. [[CrossRef](#)] [[PubMed](#)]
23. Renshaw, M.J.; Liu, J.; Lavoie, B.D.; Wilde, A. Anillin-Dependent Organization of Septin Filaments Promotes Intercellular Bridge Elongation and Chmp4B Targeting to the Abscission Site. *Open Biol.* **2014**, *4*, 130190. [[CrossRef](#)]
24. Surka, M.C.; Tsang, C.W.; Trimble, W.S. The Mammalian Septin MSF Localizes with Microtubules and Is Required for Completion of Cytokinesis. *Mol. Biol. Cell* **2002**, *13*, 3532–3545. [[CrossRef](#)] [[PubMed](#)]
25. Tokhtaeva, E.; Capri, J.; Marcus, E.A.; Whitelegge, J.P.; Khuzakhmetova, V.; Bukharaeva, E.; Deiss-Yehiely, N.; Dada, L.A.; Sachs, G.; Fernandez-Salas, E.; et al. Septin Dynamics Are Essential for Exocytosis. *J. Biol. Chem.* **2015**, *290*, 5280–5297. [[CrossRef](#)]
26. Huang, Y.-W.; Yan, M.; Collins, R.F.; DiCiccio, J.E.; Grinstein, S.; Trimble, W.S. Mammalian Septins Are Required for Phagosome Formation. *Mol. Biol. Cell* **2008**, *19*, 1717–1726. [[CrossRef](#)]
27. Hu, J.; Bai, X.; Bowen, J.R.; Dolat, L.; Korobova, F.; Yu, W.; Baas, P.W.; Svitkina, T.; Gallo, G.; Spiliotis, E.T. Septin-Driven Coordination of Actin and Microtubule Remodeling Regulates the Collateral Branching of Axons. *Curr. Biol.* **2012**, *22*, 1109–1115. [[CrossRef](#)]
28. Li, C.-R.; Lee, R.T.-H.; Wang, Y.-M.; Zheng, X.-D.; Wang, Y. *Candida Albicans* Hyphal Morphogenesis Occurs in Sec3p-Independent and Sec3p-Dependent Phases Separated by Septin Ring Formation. *J. Cell Sci.* **2007**, *120*, 1898–1907. [[CrossRef](#)]
29. Nguyen, P.A.; Field, C.M.; Groen, A.C.; Mitchison, T.J.; Loose, M. Using Supported Bilayers to Study the Spatiotemporal Organization of Membrane Bound Proteins. *Methods Cell Biol.* **2015**, *128*, 223–241. [[CrossRef](#)]
30. Tada, T.; Simonetta, A.; Batterton, M.; Kinoshita, M.; Edbauer, D.; Sheng, M. Role of Septin Cytoskeleton in Spine Morphogenesis and Dendrite Development in Neurons. *Curr. Biol.* **2007**, *17*, 1752–1758. [[CrossRef](#)]
31. Xie, Y.; Vessey, J.P.; Konecna, A.; Dahm, R.; Macchi, P.; Kiebler, M.A. The GTP-Binding Protein Septin 7 Is Critical for Dendrite Branching and Dendritic-Spine Morphology. *Curr. Biol.* **2007**, *17*, 1746–1751. [[CrossRef](#)]
32. Hu, Q.; Milenkovic, L.; Jin, H.; Scott, M.P.; Nachury, M.V.; Spiliotis, E.T.; Nelson, W.J. A Septin Diffusion Barrier at the Base of the Primary Cilium Maintains Ciliary Membrane Protein Distribution. *Science* **2010**, *329*, 436–439. [[CrossRef](#)] [[PubMed](#)]
33. Kim, S.K.; Shindo, A.; Park, T.J.; Oh, E.C.; Ghosh, S.; Gray, R.S.; Lewis, R.A.; Johnson, C.A.; Attie-Bittach, T.; Katsanis, N.; et al. Planar Cell Polarity Acts through Septins to Control Collective Cell Movement and Ciliogenesis. *Science* **2010**, *329*, 1337–1340. [[CrossRef](#)] [[PubMed](#)]
34. Palander, O.; El-Zeir, M.; Trimble, W.S. Uncovering the Roles of Septins in Cilia. *Front. Cell Dev. Biol.* **2017**, *5*, 36. [[CrossRef](#)] [[PubMed](#)]
35. Tooley, A.J.; Gilden, J.; Jacobelli, J.; Beemiller, P.; Trimble, W.S.; Kinoshita, M.; Krummel, M.F. Amoeboid T Lymphocytes Require the Septin Cytoskeleton for Cortical Integrity and Persistent Motility. *Nat. Cell Biol.* **2009**, *11*, 17–26. [[CrossRef](#)]
36. Dolat, L.; Hunyara, J.L.; Bowen, J.R.; Karasmanis, E.P.; Elgawly, M.; Galkin, V.E.; Spiliotis, E.T. Septins Promote Stress Fiber-Mediated Maturation of Focal Adhesions and Renal Epithelial Motility. *J. Cell Biol.* **2014**, *207*, 225–235. [[CrossRef](#)]
37. Ihara, M.; Kinoshita, A.; Yamada, S.; Tanaka, H.; Tanigaki, A.; Kitano, A.; Goto, M.; Okubo, K.; Nishiyama, H.; Ogawa, O.; et al. Cortical Organization by the Septin Cytoskeleton Is Essential for Structural and Mechanical Integrity of Mammalian Spermatozoa. *Dev. Cell* **2005**, *8*, 343–352. [[CrossRef](#)]
38. Chen, F.; Yan, B.; Ren, J.; Lyu, R.; Wu, Y.; Guo, Y.; Li, D.; Zhang, H.; Hu, J. FIT2 Organizes Lipid Droplet Biogenesis with ER Tubule-Forming Proteins and Septins. *J. Cell Biol.* **2021**, *220*, e201907183. [[CrossRef](#)]

39. Moreno-Castellanos, N.; Rodríguez, A.; Rabanal-Ruiz, Y.; Fernández-Vega, A.; López-Miranda, J.; Vázquez-Martínez, R.; Frühbeck, G.; Malagón, M.M. The Cytoskeletal Protein Septin 11 Is Associated with Human Obesity and Is Involved in Adipocyte Lipid Storage and Metabolism. *Diabetologia* **2017**, *60*, 324–335. [[CrossRef](#)]
40. Nurullin, L.F.; Khuzakhmetova, V.F.; Khaziev, E.F.; Samigullin, D.V.; Tsentsevitsky, A.N.; Skorinkin, A.I.; Bukharaeva, E.A.; Vagin, O. Reorganization of Septins Modulates Synaptic Transmission at Neuromuscular Junctions. *Neuroscience* **2019**, *404*, 91–101. [[CrossRef](#)]
41. Tsang, C.W.; Estey, M.P.; DiCiccio, J.E.; Xie, H.; Patterson, D.; Trimble, W.S. Characterization of Presynaptic Septin Complexes in Mammalian Hippocampal Neurons. *Biol. Chem.* **2011**, *392*, 739–749. [[CrossRef](#)] [[PubMed](#)]
42. Yang, Y.-M.; Fedchyshyn, M.J.; Grande, G.; Aitoubah, J.; Tsang, C.W.; Xie, H.; Ackerley, C.A.; Trimble, W.S.; Wang, L.-Y. Septins Regulate Developmental Switching from Microdomain to Nanodomain Coupling of Ca²⁺ Influx to Neurotransmitter Release at a Central Synapse. *Neuron* **2010**, *67*, 100–115. [[CrossRef](#)]
43. Mostowy, S.; Bonazzi, M.; Hamon, M.A.; Tham, T.N.; Mallet, A.; Lelek, M.; Gouin, E.; Demangel, C.; Brosch, R.; Zimmer, C.; et al. Entrapment of Intracytosolic Bacteria by Septin Cage-like Structures. *Cell Host Microbe* **2010**, *8*, 433–444. [[CrossRef](#)] [[PubMed](#)]
44. Krokowski, S.; Lobato-Márquez, D.; Chastanet, A.; Pereira, P.M.; Angelis, D.; Galea, D.; Larrouy-Maumus, G.; Henriques, R.; Spiliotis, E.T.; Carballido-López, R.; et al. Septins Recognize and Entrap Dividing Bacterial Cells for Delivery to Lysosomes. *Cell Host Microbe* **2018**, *24*, 866–874.e4. [[CrossRef](#)] [[PubMed](#)]
45. Dolat, L.; Hu, Q.; Spiliotis, E.T. Septin Functions in Organ System Physiology and Pathology. *Biol. Chem.* **2014**, *395*, 123–141. [[CrossRef](#)]
46. Versele, M.; Gullbrand, B.; Shulewitz, M.J.; Cid, V.J.; Bahmanyar, S.; Chen, R.E.; Barth, P.; Alber, T.; Thorner, J. Protein-Protein Interactions Governing Septin Heteropentamer Assembly and Septin Filament Organization in *Saccharomyces Cerevisiae*. *Mol. Biol. Cell* **2004**, *15*, 4568–4583. [[CrossRef](#)]
47. Cavini, I.A.; Leonardo, D.A.; Rosa, H.V.D.; Castro, D.K.S.V.; D’Muniz Pereira, H.; Valadares, N.F.; Araujo, A.P.U.; Garratt, R.C. The Structural Biology of Septins and Their Filaments: An Update. *Front. Cell Dev. Biol.* **2021**, *9*, 765085. [[CrossRef](#)]
48. Bertin, A.; McMurray, M.A.; Pierson, J.; Thai, L.; McDonald, K.L.; Zehr, E.A.; Garcia, G.; Peters, P.; Thorner, J.; Nogales, E. Three-Dimensional Ultrastructure of the Septin Filament Network in *Saccharomyces Cerevisiae*. *Mol. Biol. Cell* **2012**, *23*, 423–432. [[CrossRef](#)]
49. Bridges, A.A.; Zhang, H.; Mehta, S.B.; Occhipinti, P.; Tani, T.; Gladfelter, A.S. Septin Assemblies Form by Diffusion-Driven Annealing on Membranes. *Proc. Natl. Acad. Sci. USA* **2014**, *111*, 2146–2151. [[CrossRef](#)]
50. John, C.M.; Hite, R.K.; Weirich, C.S.; Fitzgerald, D.J.; Jawhari, H.; Faty, M.; Schläpfer, D.; Kroschewski, R.; Winkler, F.K.; Walz, T.; et al. The *Caenorhabditis Elegans* Septin Complex Is Nonpolar. *EMBO J.* **2007**, *26*, 3296–3307. [[CrossRef](#)]
51. Rodal, A.A.; Kozubowski, L.; Goode, B.L.; Drubin, D.G.; Hartwig, J.H. Actin and Septin Ultrastructures at the Budding Yeast Cell Cortex. *MBoC* **2005**, *16*, 372–384. [[CrossRef](#)] [[PubMed](#)]
52. Sirajuddin, M.; Farkasovsky, M.; Hauer, F.; Kühlmann, D.; Macara, I.G.; Weyand, M.; Stark, H.; Wittinghofer, A. Structural Insight into Filament Formation by Mammalian Septins. *Nature* **2007**, *449*, 311–315. [[CrossRef](#)] [[PubMed](#)]
53. Field, C.M.; al-Awar, O.; Rosenblatt, J.; Wong, M.L.; Alberts, B.; Mitchison, T.J. A Purified *Drosophila* Septin Complex Forms Filaments and Exhibits GTPase Activity. *J. Cell Biol.* **1996**, *133*, 605–616. [[CrossRef](#)] [[PubMed](#)]
54. Arbizzani, F.; Mavrikis, M.; Hoya, M.; Ribas, J.C.; Brasselet, S.; Paoletti, A.; Rincon, S.A. Septin Filament Compaction into Rings Requires the Anillin Mid2 and Contractile Ring Constriction. *Cell Rep.* **2022**, *39*, 110722. [[CrossRef](#)] [[PubMed](#)]
55. An, H.; Morrell, J.L.; Jennings, J.L.; Link, A.J.; Gould, K.L. Requirements of Fission Yeast Septins for Complex Formation, Localization, and Function. *Mol. Biol. Cell* **2004**, *15*, 5551–5564. [[CrossRef](#)]
56. Wu, J.Q.; Kuhn, J.R.; Kovar, D.R.; Pollard, T.D. Spatial and Temporal Pathway for Assembly and Constriction of the Contractile Ring in Fission Yeast Cytokinesis. *Dev. Cell* **2003**, *5*, 723–734. [[CrossRef](#)]
57. Bertin, A.; McMurray, M.A.; Grob, P.; Park, S.S.; Garcia, G., 3rd; Patanwala, I.; Ng, H.L.; Alber, T.; Thorner, J.; Nogales, E. *Saccharomyces Cerevisiae* Septins: Supramolecular Organization of Heterooligomers and the Mechanism of Filament Assembly. *Proc. Natl. Acad. Sci. USA* **2008**, *105*, 8274–8279. [[CrossRef](#)]
58. Garcia, G., 3rd; Bertin, A.; Li, Z.; Song, Y.; McMurray, M.A.; Thorner, J.; Nogales, E. Subunit-Dependent Modulation of Septin Assembly: Budding Yeast Septin Shs1 Promotes Ring and Gauze Formation. *J. Cell Biol.* **2011**, *195*, 993–1004. [[CrossRef](#)]
59. Taveneau, C.; Blanc, R.; Péhau-Arnaudet, G.; Di Cicco, A.; Bertin, A. Synergistic Role of Nucleotides and Lipids for the Self-Assembly of Shs1 Septin Oligomers. *Biochem. J.* **2020**, *477*, 2697–2714. [[CrossRef](#)]
60. Beber, A.; Alqabandi, M.; Prévost, C.; Viars, F.; Lévy, D.; Bassereau, P.; Bertin, A.; Mangenot, S. Septin-Based Readout of PI(4,5)P₂ Incorporation into Membranes of Giant Unilamellar Vesicles. *Cytoskeleton* **2019**, *76*, 92–103. [[CrossRef](#)]
61. Szuba, A.; Bano, F.; Castro-Linares, G.; Iv, F.; Mavrikis, M.; Richter, R.P.; Bertin, A.; Koenderink, G.H. Membrane Binding Controls Ordered Self-Assembly of Animal Septins. *eLife* **2021**, *10*, e63349. [[CrossRef](#)] [[PubMed](#)]
62. Zhang, J.; Kong, C.; Xie, H.; McPherson, P.S.; Grinstein, S.; Trimble, W.S. Phosphatidylinositol Polyphosphate Binding to the Mammalian Septin H5 Is Modulated by GTP. *Curr. Biol.* **1999**, *9*, 1458–1467. [[CrossRef](#)] [[PubMed](#)]
63. Bertin, A.; McMurray, M.A.; Thai, L.; Garcia, G.; Votin, V.; Grob, P.; Allyn, T.; Thorner, J.; Nogales, E. Phosphatidylinositol-4,5-Bisphosphate Promotes Budding Yeast Septin Filament Assembly and Organization. *J. Mol. Biol.* **2010**, *404*, 711–731. [[CrossRef](#)] [[PubMed](#)]

64. Tanaka-Takiguchi, Y.; Kinoshita, M.; Takiguchi, K. Septin-Mediated Uniform Bracing of Phospholipid Membranes. *Curr. Biol.* **2009**, *19*, 140–145. [[CrossRef](#)] [[PubMed](#)]
65. Garrenton, L.S.; Stefan, C.J.; McMurray, M.A.; Emr, S.D.; Thorner, J. Pheromone-Induced Anisotropy in Yeast Plasma Membrane Phosphatidylinositol-4,5-Bisphosphate Distribution Is Required for MAPK Signaling. *Proc. Natl. Acad. Sci. USA* **2010**, *107*, 11805–11810. [[CrossRef](#)]
66. Rodríguez-Escudero, I.; Roelants, F.M.; Thorner, J.; Nombela, C.; Molina, M.; Cid, V.J. Reconstitution of the Mammalian PI3K/PTEN/Akt Pathway in Yeast. *Biochem. J.* **2005**, *390*, 613–623. [[CrossRef](#)]
67. Casamayor, A.; Snyder, M. Molecular Dissection of a Yeast Septin: Distinct Domains Are Required for Septin Interaction, Localization, and Function. *Mol. Cell. Biol.* **2003**, *23*, 2762–2777. [[CrossRef](#)]
68. Omrane, M.; Camara, A.S.; Taveneau, C.; Benzoubir, N.; Tubiana, T.; Yu, J.; Guérois, R.; Samuel, D.; Goud, B.; Poüs, C.; et al. Septin 9 Has Two Polybasic Domains Critical to Septin Filament Assembly and Golgi Integrity. *iScience* **2019**, *13*, 138–153. [[CrossRef](#)]
69. Alaoui, F.E.; Al-Akiki, I.; Ibanes, S.; Lyonais, S.; Sanchez-Fuentes, D.; Desgarceaux, R.; Cazevielle, C.; Blanchard, M.-P.; Parmeggiani, A.; Carretero-Genevri, A.; et al. Septin Filament Assembly Assist the Lateral Organization of Membranes. *bioRxiv* **2024**, preprint.
70. Beber, A.; Taveneau, C.; Nania, M.; Tsai, F.-C.; Di Cicco, A.; Bassereau, P.; Lévy, D.; Cabral, J.T.; Isambert, H.; Mangenot, S.; et al. Membrane Reshaping by Micrometric Curvature Sensitive Septin Filaments. *Nat. Commun.* **2019**, *10*, 420. [[CrossRef](#)]
71. Bridges, A.A.; Jentsch, M.S.; Oakes, P.W.; Occhipinti, P.; Gladfelter, A.S. Micron-Scale Plasma Membrane Curvature Is Recognized by the Septin Cytoskeleton. *J. Cell Biol.* **2016**, *213*, 23–32. [[CrossRef](#)]
72. Cannon, K.S.; Woods, B.L.; Crutchley, J.M.; Gladfelter, A.S. An Amphipathic Helix Enables Septins to Sense Micrometer-Scale Membrane Curvature. *J. Cell Biol.* **2019**, *218*, 1128–1137. [[CrossRef](#)]
73. Ewers, H.; Tada, T.; Petersen, J.D.; Racz, B.; Sheng, M.; Choquet, D. A Septin-Dependent Diffusion Barrier at Dendritic Spine Necks. *PLoS ONE* **2014**, *9*, e113916. [[CrossRef](#)] [[PubMed](#)]
74. Kuo, Y.C.; Lin, Y.H.; Chen, H.I.; Wang, Y.Y.; Chiou, Y.W.; Lin, H.H.; Pan, H.A.; Wu, C.M.; Su, S.M.; Hsu, C.C.; et al. SEPT12 Mutations Cause Male Infertility with Defective Sperm Annulus. *Hum. Mutat.* **2012**, *33*, 710–719. [[CrossRef](#)]
75. Khan, A.; Newby, J.; Gladfelter, A.S. Control of Septin Filament Flexibility and Bundling by Subunit Composition and Nucleotide Interactions. *MBoC* **2018**, *29*, 702–712. [[CrossRef](#)] [[PubMed](#)]
76. Frazier, J.A.; Wong, M.L.; Longtine, M.S.; Pringle, J.R.; Mann, M.; Mitchison, T.J.; Field, C. Polymerization of Purified Yeast Septins: Evidence That Organized Filament Arrays May Not Be Required for Septin Function. *J. Cell Biol.* **1998**, *143*, 737–749. [[CrossRef](#)] [[PubMed](#)]
77. Booth, E.A.; Vane, E.W.; Dovala, D.; Thorner, J. A Förster Resonance Energy Transfer (FRET)-Based System Provides Insight into the Ordered Assembly of Yeast Septin Hetero-Octamers*. *J. Biol. Chem.* **2015**, *290*, 28388–28401. [[CrossRef](#)] [[PubMed](#)]
78. McMurray, M.A.; Bertin, A.; Garcia, G.; Lam, L.; Nogales, E.; Thorner, J. Septin Filament Formation Is Essential in Budding Yeast. *Dev. Cell* **2011**, *20*, 540–549. [[CrossRef](#)]
79. Garcia, G.; Finnigan, G.C.; Heasley, L.R.; Sterling, S.M.; Aggarwal, A.; Pearson, C.G.; Nogales, E.; McMurray, M.A.; Thorner, J. Assembly, Molecular Organization, and Membrane-Binding Properties of Development-Specific Septins. *J. Cell Biol.* **2016**, *212*, 515–529. [[CrossRef](#)]
80. Heasley, L.R.; McMurray, M.A. Roles of Septins in Prospore Membrane Morphogenesis and Spore Wall Assembly in *Saccharomyces Cerevisiae*. *MBoC* **2016**, *27*, 442–450. [[CrossRef](#)]
81. Cid, V.J.; Adamikova, L.; Sanchez, M.; Molina, M.; Nombela, C. Cell Cycle Control of Septin Ring Dynamics in the Budding Yeast. *Microbiology* **2001**, *147*, 1437–1450. [[CrossRef](#)] [[PubMed](#)]
82. Kim, H.B.; Haarer, B.K.; Pringle, J.R. Cellular Morphogenesis in the *Saccharomyces cerevisiae* Cell Cycle: Localization of the CDC3 Gene Product and the Timing of Events at the Budding Site. *J. Cell Biol.* **1991**, *112*, 535–544. [[CrossRef](#)]
83. Caviston, J.P.; Longtine, M.; Pringle, J.R.; Bi, E. The Role of Cdc42p GTPase-Activating Proteins in Assembly of the Septin Ring in Yeast. *Mol. Biol. Cell* **2003**, *14*, 4051–4066. [[CrossRef](#)] [[PubMed](#)]
84. Dobbelaere, J.; Gentry, M.S.; Hallberg, R.L.; Barral, Y. Phosphorylation-Dependent Regulation of Septin Dynamics during the Cell Cycle. *Dev. Cell* **2003**, *4*, 345–357. [[CrossRef](#)] [[PubMed](#)]
85. Longtine, M.S.; Fares, H.; Pringle, J.R. Role of the Yeast Gin4p Protein Kinase in Septin Assembly and the Relationship between Septin Assembly and Septin Function. *J. Cell Biol.* **1998**, *143*, 719–736. [[CrossRef](#)]
86. Tartakoff, A.M.; Aylyarov, I.; Jaiswal, P. Septin-Containing Barriers Control the Differential Inheritance of Cytoplasmic Elements. *Cell Rep.* **2013**, *3*, 223–236. [[CrossRef](#)]
87. Fares, H.; Goetsch, L.; Pringle, J.R. Identification of a Developmentally Regulated Septin and Involvement of the Septins in Spore Formation in *Saccharomyces Cerevisiae*. *J. Cell Biol.* **1996**, *132*, 399–411. [[CrossRef](#)]
88. Heasley, L.R.; Singer, E.; Cooperman, B.J.; McMurray, M.A. *Saccharomyces* Spores Are Born Prepolarized to Outgrow Away from Spore–Spore Connections and Penetrate the Ascus Wall. *Yeast* **2021**, *38*, 90–101. [[CrossRef](#)]
89. Joseph-Strauss, D.; Zenvirth, D.; Simchen, G.; Barkai, N. Spore Germination in *Saccharomyces Cerevisiae*: Global Gene Expression Patterns and Cell Cycle Landmarks. *Genome Biol.* **2007**, *8*, R241. [[CrossRef](#)]
90. Spiliotis, E.T.; Nakos, K. Cellular Functions of Actin- and Microtubule-Associated Septins. *Curr. Biol.* **2021**, *31*, R651–R666. [[CrossRef](#)]

91. Ong, K.; Wloka, C.; Okada, S.; Svitkina, T.; Bi, E. Architecture and Dynamic Remodelling of the Septin Cytoskeleton during the Cell Cycle. *Nat. Commun.* **2014**, *5*, 5698. [[CrossRef](#)] [[PubMed](#)]
92. Vrabioiu, A.M.; Mitchison, T.J. Structural Insights into Yeast Septin Organization from Polarized Fluorescence Microscopy. *Nature* **2006**, *443*, 466–469. [[CrossRef](#)] [[PubMed](#)]
93. McQuilken, M.; Jentzsch, M.S.; Verma, A.; Mehta, S.B.; Oldenbourg, R.; Gladfelter, A.S. Analysis of Septin Reorganization at Cytokinesis Using Polarized Fluorescence Microscopy. *Front. Cell Dev. Biol.* **2017**, *5*, 42. [[CrossRef](#)] [[PubMed](#)]
94. Demay, B.S.; Bai, X.; Howard, L.; Occhipinti, P.; Meseroll, R.A.; Spiliotis, E.T.; Oldenbourg, R.; Gladfelter, A.S. Septin Filaments Exhibit a Dynamic, Paired Organization That Is Conserved from Yeast to Mammals. *J. Cell Biol.* **2011**, *193*, 1065–1081. [[CrossRef](#)]
95. Tamborrini, D.; Juanes, M.A.; Ibanes, S.; Rancati, G.; Piatti, S. Recruitment of the Mitotic Exit Network to Yeast Centrosomes Couples Septin Displacement to Actomyosin Constriction. *Nat. Commun.* **2018**, *9*, 4308. [[CrossRef](#)]
96. Marquardt, J.; Chen, X.; Bi, E. Architecture, Remodeling, and Functions of the Septin Cytoskeleton. *Cytoskeleton* **2019**, *76*, 7–14. [[CrossRef](#)]
97. Bi, E.; Park, H.O. Cell Polarization and Cytokinesis in Budding Yeast. *Genetics* **2012**, *191*, 347–387. [[CrossRef](#)]
98. Cvrckova, F.; De Virgilio, C.; Manser, E.; Pringle, J.R.; Nasmyth, K. Ste20-like Protein Kinases Are Required for Normal Localization of Cell Growth and for Cytokinesis in Budding Yeast. *Genes. Dev.* **1995**, *9*, 1817–1830. [[CrossRef](#)]
99. Kang, P.J.; Mullner, R.; Lian, K.; Park, H.-O. Cdc42 Couples Septin Recruitment to the Axial Landmark Assembly via Axl2 in Budding Yeast. *J. Cell Sci.* **2023**, *137*, jcs261080. [[CrossRef](#)]
100. Chant, J.; Mischke, M.; Mitchell, E.; Herskowitz, I.; Pringle, J.R. Role of Bud3p in Producing the Axial Budding Pattern of Yeast. *J. Cell Biol.* **1995**, *129*, 767–778. [[CrossRef](#)]
101. Sanders, S.L.; Herskowitz, I. The BUD4 Protein of Yeast, Required for Axial Budding, Is Localized to the Mother/BUD Neck in a Cell Cycle-Dependent Manner. *J. Cell Biol.* **1996**, *134*, 413–427. [[CrossRef](#)] [[PubMed](#)]
102. Kang, P.J.; Angerman, E.; Jung, C.H.; Park, H.O. Bud4 Mediates the Cell-Type-Specific Assembly of the Axial Landmark in Budding Yeast. *J. Cell Sci.* **2012**, *125*, 3840–3849. [[CrossRef](#)] [[PubMed](#)]
103. Okada, S.; Leda, M.; Hanna, J.; Savage, N.S.; Bi, E.; Goryachev, A.B. Daughter Cell Identity Emerges from the Interplay of Cdc42, Septins, and Exocytosis. *Dev. Cell* **2013**, *26*, 148–161. [[CrossRef](#)]
104. Lippincott, J.; Li, R. Sequential Assembly of Myosin II, an IQGAP-like Protein, and Filamentous Actin to a Ring Structure Involved in Budding Yeast Cytokinesis. *J. Cell Biol.* **1998**, *140*, 355–366. [[CrossRef](#)]
105. Luo, J.; Vallen, E.A.; Dravis, C.; Tcheperegine, S.E.; Drees, B.; Bi, E. Identification and Functional Analysis of the Essential and Regulatory Light Chains of the Only Type II Myosin Myo1p in *Saccharomyces Cerevisiae*. *J. Cell Biol.* **2004**, *165*, 843–855. [[CrossRef](#)] [[PubMed](#)]
106. Watts, F.Z.; Shiels, G.; Orr, E. The Yeast MYO1 Gene Encoding a Myosin-like Protein Required for Cell Division. *EMBO J.* **1987**, *6*, 3499–3505. [[CrossRef](#)]
107. Boyne, J.R.; Yusuf, H.M.; Bieganowski, P.; Brenner, C.; Price, C. Yeast Myosin Light Chain, Mlc1p, Interacts with Both IQGAP and Class II Myosin to Effect Cytokinesis. *J. Cell Sci.* **2000**, *113 Pt 24*, 4533–4543. [[CrossRef](#)]
108. Epp, J.A.; Chant, J. An IQGAP-Related Protein Controls Actin-Ring Formation and Cytokinesis in Yeast. *Curr. Biol.* **1997**, *7*, 921–929. [[CrossRef](#)]
109. Fang, X.; Luo, J.; Nishihama, R.; Wloka, C.; Dravis, C.; Travaglia, M.; Iwase, M.; Vallen, E.A.; Bi, E. Biphasic Targeting and Cleavage Furrow Ingression Directed by the Tail of a Myosin II. *J. Cell Biol.* **2010**, *191*, 1333–1350. [[CrossRef](#)]
110. Shannon, K.B.; Li, R. A Myosin Light Chain Mediates the Localization of the Budding Yeast IQGAP-like Protein during Contractile Ring Formation. *Curr. Biol.* **2000**, *10*, 727–730. [[CrossRef](#)]
111. Tolliday, N.; VerPlank, L.; Li, R. Rho1 Directs Formin-Mediated Actin Ring Assembly during Budding Yeast Cytokinesis. *Curr. Biol.* **2002**, *12*, 1864–1870. [[CrossRef](#)] [[PubMed](#)]
112. Vallen, E.A.; Caviston, J.; Bi, E. Roles of Hof1p, Bni1p, Bnr1p, and Myo1p in Cytokinesis in *Saccharomyces Cerevisiae*. *Mol. Biol. Cell* **2000**, *11*, 593–611. [[CrossRef](#)]
113. Feng, Z.; Okada, S.; Cai, G.; Zhou, B.; Bi, E. MyosinII Heavy Chain and Formin Mediate the Targeting of Myosin Essential Light Chain to the Division Site before and during Cytokinesis. *Mol. Biol. Cell* **2015**, *26*, 1211–1224. [[CrossRef](#)] [[PubMed](#)]
114. Chuang, J.S.; Schekman, R.W. Differential Trafficking and Timed Localization of Two Chitin Synthase Proteins, Chs2p and Chs3p. *J. Cell Biol.* **1996**, *135*, 597–610. [[CrossRef](#)]
115. Meitinger, F.; Petrova, B.; Lombardi, I.M.; Bertazzi, D.T.; Hub, B.; Zentgraf, H.; Pereira, G. Targeted Localization of Inn1, Cyk3 and Chs2 by the Mitotic-Exit Network Regulates Cytokinesis in Budding Yeast. *J. Cell Sci.* **2010**, *123*, 1851–1861. [[CrossRef](#)] [[PubMed](#)]
116. Foltman, M.; Filali-Mouneef, Y.; Crespo, D.; Sanchez-Diaz, A. Cell Polarity Protein Spa2 Coordinates Chs2 Incorporation at the Division Site in Budding Yeast. *PLoS Genet.* **2018**, *14*, e1007299. [[CrossRef](#)]
117. VerPlank, L.; Li, R. Cell Cycle-Regulated Trafficking of Chs2 Controls Actomyosin Ring Stability during Cytokinesis. *Mol. Biol. Cell* **2005**, *16*, 2529–2543. [[CrossRef](#)]
118. Chin, C.F.; Bennett, A.M.; Ma, W.K.; Hall, M.C.; Yeong, F.M. Dependence of Chs2 ER Export on Dephosphorylation by Cytoplasmic Cdc14 Ensures That Septum Formation Follows Mitosis. *Mol. Biol. Cell* **2012**, *23*, 45–58. [[CrossRef](#)]
119. Nishihama, R.; Schreiter, J.H.; Onishi, M.; Vallen, E.A.; Hanna, J.; Moravcevic, K.; Lippincott, M.F.; Han, H.; Lemmon, M.A.; Pringle, J.R.; et al. Role of Inn1 and Its Interactions with Hof1 and Cyk3 in Promoting Cleavage Furrow and Septum Formation in *S. Cerevisiae*. *J. Cell Biol.* **2009**, *185*, 995–1012. [[CrossRef](#)]

120. Oh, Y.; Schreiter, J.H.; Okada, H.; Wloka, C.; Okada, S.; Yan, D.; Duan, X.; Bi, E. Hof1 and Chs4 Interact via F-BAR Domain and Sel1-like Repeats to Control Extracellular Matrix Deposition during Cytokinesis. *Curr. Biol.* **2017**, *27*, 2878–2886.e5. [[CrossRef](#)]
121. Wang, M.; Nishihama, R.; Onishi, M.; Pringle, J.R. Role of the Hof1–Cyk3 Interaction in Cleavage-Furrow Ingression and Primary-Septum Formation during Yeast Cytokinesis. *Mol. Biol. Cell* **2018**, *29*, 597–609. [[CrossRef](#)]
122. Devrekanli, A.; Foltman, M.; Roncero, C.; Sanchez-Diaz, A.; Labib, K. Inn1 and Cyk3 Regulate Chitin Synthase during Cytokinesis in Budding Yeasts. *J. Cell Sci.* **2012**, *125*, 5453–5466. [[CrossRef](#)] [[PubMed](#)]
123. Jendretzki, A.; Ciklic, I.; Rodicio, R.; Schmitz, H.P.; Heinisch, J.J. Cyk3 Acts in Actomyosin Ring Independent Cytokinesis by Recruiting Inn1 to the Yeast Bud Neck. *Mol. Genet. Genom. MGG* **2009**, *282*, 437–451. [[CrossRef](#)] [[PubMed](#)]
124. Onishi, M.; Ko, N.; Nishihama, R.; Pringle, J.R. Distinct Roles of Rho1, Cdc42, and Cyk3 in Septum Formation and Abscission during Yeast Cytokinesis. *J. Cell Biol.* **2013**, *202*, 311–329. [[CrossRef](#)]
125. Sanchez-Diaz, A.; Marchesi, V.; Murray, S.; Jones, R.; Pereira, G.; Edmondson, R.; Allen, T.; Labib, K. Inn1 Couples Contraction of the Actomyosin Ring to Membrane Ingression during Cytokinesis in Budding Yeast. *Nat. Cell Biol.* **2008**, *10*, 395–406. [[CrossRef](#)] [[PubMed](#)]
126. Foltman, M.; Molist, I.; Arcones, I.; Sacristan, C.; Filali-Mounecef, Y.; Roncero, C.; Sanchez-Diaz, A. Ingression Progression Complexes Control Extracellular Matrix Remodelling during Cytokinesis in Budding Yeast. *PLoS Genet.* **2016**, *12*, e1005864. [[CrossRef](#)]
127. Lippincott, J.; Li, R. Dual Function of Cyk2, a Cdc15/PSTPIP Family Protein, in Regulating Actomyosin Ring Dynamics and Septin Distribution. *J. Cell Biol.* **1998**, *143*, 1947–1960. [[CrossRef](#)]
128. Gao, L.; Liu, W.; Bretscher, A. The Yeast Formin Bnr1p Has Two Localization Regions That Show Spatially and Temporally Distinct Association with Septin Structures. *Mol. Biol. Cell* **2010**, *21*, 1253–1262. [[CrossRef](#)]
129. Pruyne, D.; Gao, L.; Bi, E.; Bretscher, A. Stable and Dynamic Axes of Polarity Use Distinct Formin Isoforms in Budding Yeast. *Mol. Biol. Cell* **2004**, *15*, 4971–4989. [[CrossRef](#)]
130. Huxley, H.E. The Mechanism of Muscular Contraction. *Science* **1969**, *164*, 1356–1365. [[CrossRef](#)]
131. Huxley, H.; Hanson, J. Changes in the Cross-Striations of Muscle during Contraction and Stretch and Their Structural Interpretation. *Nature* **1954**, *173*, 973–976. [[CrossRef](#)] [[PubMed](#)]
132. Mendes Pinto, I.; Rubinstein, B.; Kucharavy, A.; Unruh, J.R.; Li, R. Actin Depolymerization Drives Actomyosin Ring Contraction during Budding Yeast Cytokinesis. *Dev. Cell* **2012**, *22*, 1247–1260. [[CrossRef](#)] [[PubMed](#)]
133. Ko, N.; Nishihama, R.; Tully, G.H.; Ostapenko, D.; Solomon, M.J.; Morgan, D.O.; Pringle, J.R. Identification of Yeast IQGAP (Iqg1p) as an Anaphase-Promoting-Complex Substrate and Its Role in Actomyosin-Ring-Independent Cytokinesis. *MBoC* **2007**, *18*, 5139–5153. [[CrossRef](#)] [[PubMed](#)]
134. Tully, G.H.; Nishihama, R.; Pringle, J.R.; Morgan, D.O. The Anaphase-Promoting Complex Promotes Actomyosin-Ring Disassembly during Cytokinesis in Yeast. *MBoC* **2009**, *20*, 1201–1212. [[CrossRef](#)]
135. Cabib, E. The Septation Apparatus, a Chitin-Requiring Machine in Budding Yeast. *Arch. Biochem. Biophys.* **2004**, *426*, 201–207. [[CrossRef](#)]
136. Zhang, G.; Kashimshetty, R.; Ng, K.E.; Tan, H.B.; Yeong, F.M. Exit from Mitosis Triggers Chs2p Transport from the Endoplasmic Reticulum to Mother-Daughter Neck via the Secretory Pathway in Budding Yeast. *J. Cell Biol.* **2006**, *174*, 207–220. [[CrossRef](#)]
137. Bi, E. Cytokinesis in Budding Yeast: The Relationship between Actomyosin Ring Function and Septum Formation. *Cell Struct. Funct.* **2001**, *26*, 529–537. [[CrossRef](#)]
138. Schmidt, M.; Bowers, B.; Varma, A.; Roh, D.H.; Cabib, E. In Budding Yeast, Contraction of the Actomyosin Ring and Formation of the Primary Septum at Cytokinesis Depend on Each Other. *J. Cell Sci.* **2002**, *115*, 293–302. [[CrossRef](#)]
139. Roh, D.-H.; Bowers, B.; Schmidt, M.; Cabib, E. The Septation Apparatus, an Autonomous System in Budding Yeast. *Mol. Biol. Cell* **2002**, *13*, 2747–2759. [[CrossRef](#)]
140. Lippincott, J.; Shannon, K.B.; Shou, W.; Deshaies, R.J.; Li, R. The Tem1 Small GTPase Controls Actomyosin and Septin Dynamics during Cytokinesis. *J. Cell Sci.* **2001**, *114*, 1379–1386. [[CrossRef](#)]
141. Tamborrini, D.; Piatti, S. Septin Clearance from the Division Site Triggers Cytokinesis in Budding Yeast. *Microb. Cell* **2019**, *6*, 296–298. [[CrossRef](#)]
142. Dobbelaere, J.; Barral, Y. Spatial Coordination of Cytokinetic Events by Compartmentalization of the Cell Cortex. *Science* **2004**, *305*, 393–396. [[CrossRef](#)]
143. Okada, H.; MacTaggart, B.; Ohya, Y.; Bi, E. The Kinetic Landscape and Interplay of Protein Networks in Cytokinesis. *iScience* **2021**, *24*, 101917. [[CrossRef](#)] [[PubMed](#)]
144. Weiss, E.L. Mitotic Exit and Separation of Mother and Daughter Cells. *Genetics* **2012**, *192*, 1165–1202. [[CrossRef](#)] [[PubMed](#)]
145. Martin-Cuadrado, A.B.; Morrell, J.L.; Konomi, M.; An, H.; Petit, C.; Osumi, M.; Balasubramanian, M.; Gould, K.L.; Del Rey, F.; de Aldana, C.R. Role of Septins and the Exocyst Complex in the Function of Hydrolytic Enzymes Responsible for Fission Yeast Cell Separation. *Mol. Biol. Cell* **2005**, *16*, 4867–4881. [[CrossRef](#)] [[PubMed](#)]
146. Gladfelter, A.S.; Moskow, J.J.; Zyla, T.R.; Lew, D.J. Isolation and Characterization of Effector-Loop Mutants of CDC42 in Yeast. *Mol. Biol. Cell* **2001**, *12*, 1239–1255. [[CrossRef](#)]
147. Iwase, M.; Luo, J.; Nagaraj, S.; Longtine, M.; Kim, H.B.; Haarer, B.K.; Caruso, C.; Tong, Z.; Pringle, J.R.; Bi, E. Role of a Cdc42p Effector Pathway in Recruitment of the Yeast Septins to the Presumptive Bud Site. *Mol. Biol. Cell* **2006**, *17*, 1110–1125. [[CrossRef](#)]

148. Gladfelder, A.S.; Bose, I.; Zyla, T.R.; Bardes, E.S.; Lew, D.J. Septin Ring Assembly Involves Cycles of GTP Loading and Hydrolysis by Cdc42p. *J. Cell Biol.* **2002**, *156*, 315–326. [[CrossRef](#)]
149. Benton, B.K.; Tinkelenberg, A.; Gonzalez, I.; Cross, F.R. Cla4p, a *Saccharomyces Cerevisiae* Cdc42p-Activated Kinase Involved in Cytokinesis, Is Activated at Mitosis. *Mol. Cell Biol.* **1997**, *17*, 5067–5076. [[CrossRef](#)]
150. Kadota, J.; Yamamoto, T.; Yoshiuchi, S.; Bi, E.; Tanaka, K. Septin Ring Assembly Requires Concerted Action of Polarisome Components, a PAK Kinase Cla4p, and the Actin Cytoskeleton in *Saccharomyces Cerevisiae*. *Mol. Biol. Cell* **2004**, *15*, 5329–5345. [[CrossRef](#)]
151. Versele, M.; Thorner, J. Septin Collar Formation in Budding Yeast Requires GTP Binding and Direct Phosphorylation by the PAK, Cla4. *J. Cell Biol.* **2004**, *164*, 701–715. [[CrossRef](#)]
152. Bose, I.; Irazoqui, J.E.; Moskow, J.J.; Bardes, E.S.; Zyla, T.R.; Lew, D.J. Assembly of Scaffold-Mediated Complexes Containing Cdc42p, the Exchange Factor Cdc24p, and the Effector Cla4p Required for Cell Cycle-Regulated Phosphorylation of Cdc24p. *J. Biol. Chem.* **2001**, *276*, 7176–7186. [[CrossRef](#)] [[PubMed](#)]
153. Gulli, M.-P.; Jaquenoud, M.; Shimada, Y.; Niederhäuser, G.; Wiget, P.; Peter, M. Phosphorylation of the Cdc42 Exchange Factor Cdc24 by the PAK-like Kinase Cla4 May Regulate Polarized Growth in Yeast. *Mol. Cell* **2000**, *6*, 1155–1167. [[CrossRef](#)] [[PubMed](#)]
154. Kozubowski, L.; Saito, K.; Johnson, J.M.; Howell, A.S.; Zyla, T.R.; Lew, D.J. Symmetry-Breaking Polarization Driven by a Cdc42p GEF-PAK Complex. *Curr. Biol.* **2008**, *18*, 1719–1726. [[CrossRef](#)]
155. Chollet, J.; Dünkler, A.; Bäuerle, A.; Vivero-Pol, L.; Mulaw, M.A.; Gronemeyer, T.; Johnsson, N. Cdc24 Interacts with the Septins to Create a Positive Feedback during Bud Site Assembly in Yeast. *J. Cell Sci.* **2020**, *133*, jcs240283. [[CrossRef](#)] [[PubMed](#)]
156. Brown, J.L.; Jaquenoud, M.; Gulli, M.P.; Chant, J.; Peter, M. Novel Cdc42-Binding Proteins Gic1 and Gic2 Control Cell Polarity in Yeast. *Genes. Dev.* **1997**, *11*, 2972–2982. [[CrossRef](#)] [[PubMed](#)]
157. Orlando, K.; Sun, X.; Zhang, J.; Lu, T.; Yokomizo, L.; Wang, P.; Guo, W. Exo-Endocytic Trafficking and the Septin-Based Diffusion Barrier Are Required for the Maintenance of Cdc42p Polarization during Budding Yeast Asymmetric Growth. *Mol. Biol. Cell* **2011**, *22*, 624–633. [[CrossRef](#)]
158. Sadian, Y.; Gatsogiannis, C.; Patasi, C.; Hofnagel, O.; Goody, R.S.; Farkasovsky, M.; Raunser, S. The Role of Cdc42 and Gic1 in the Regulation of Septin Filament Formation and Dissociation. *eLife* **2013**, *2*, e01085. [[CrossRef](#)]
159. Kang, P.J.; Miller, K.E.; Guegueniat, J.; Beven, L.; Park, H.-O. The Shared Role of the Rsr1 GTPase and Gic1/Gic2 in Cdc42 Polarization. *MBoC* **2018**, *29*, 2359–2369. [[CrossRef](#)]
160. Daniels, C.N.; Zyla, T.R.; Lew, D.J. A Role for Gic1 and Gic2 in Cdc42 Polarization at Elevated Temperature. *PLoS ONE* **2018**, *13*, e0200863. [[CrossRef](#)]
161. Lemmon, M.A.; Ferguson, K.M.; O'Brien, R.; Sigler, P.B.; Schlessinger, J. Specific and High-Affinity Binding of Inositol Phosphates to an Isolated Pleckstrin Homology Domain. *Proc. Natl. Acad. Sci. USA* **1995**, *92*, 10472–10476. [[CrossRef](#)]
162. Gao, X.-D.; Sperber, L.M.; Kane, S.A.; Tong, Z.; Tong, A.H.Y.; Boone, C.; Bi, E. Sequential and Distinct Roles of the Cadherin Domain-Containing Protein Axl2p in Cell Polarization in Yeast Cell Cycle. *MBoC* **2007**, *18*, 2542–2560. [[CrossRef](#)] [[PubMed](#)]
163. Lai, H.; Chiou, J.-G.; Zhurikhina, A.; Zyla, T.R.; Tsygankov, D.; Lew, D.J. Temporal Regulation of Morphogenetic Events in *Saccharomyces Cerevisiae*. *Mol. Biol. Cell* **2018**, *29*, 2069–2083. [[CrossRef](#)] [[PubMed](#)]
164. Merlini, L.; Bolognesi, A.; Juanes, M.A.; Vandermoere, F.; Courtellemont, T.; Pascolutti, R.; Seveno, M.; Barral, Y.; Piatti, S. Rho1- and Pkc1-Dependent Phosphorylation of the F-BAR Protein Syp1 Contributes to Septin Ring Assembly. *Mol. Biol. Cell* **2015**, *26*, 3245–3262. [[CrossRef](#)] [[PubMed](#)]
165. Qiu, W.; Neo, S.P.; Yu, X.; Cai, M. A Novel Septin-Associated Protein, Syp1p, Is Required for Normal Cell Cycle-Dependent Septin Cytoskeleton Dynamics in Yeast. *Genetics* **2008**, *180*, 1445–1457. [[CrossRef](#)]
166. Ibanes, S.; El-Alaoui, F.; Lai-Kee-Him, J.; Cazevieille, C.; Hoh, F.; Lyonnais, S.; Bron, P.; Cipelletti, L.; Picas, L.; Piatti, S. The Syp1/FCHo2 Protein Induces Septin Filament Bundling through Its Intrinsically Disordered Domain. *Cell Rep.* **2022**, *41*, 111765. [[CrossRef](#)]
167. Mortensen, E.M.; McDonald, H.; Yates, J., 3rd; Kellogg, D.R. Cell Cycle-Dependent Assembly of a Gin4-Septin Complex. *Mol. Biol. Cell* **2002**, *13*, 2091–2105. [[CrossRef](#)]
168. Asano, S.; Park, J.E.; Yu, L.R.; Zhou, M.; Sakchaisri, K.; Park, C.J.; Kang, Y.H.; Thorner, J.; Veenstra, T.D.; Lee, K.S. Direct Phosphorylation and Activation of a Nim1-Related Kinase Gin4 by Elm1 in Budding Yeast. *J. Biol. Chem.* **2006**, *281*, 27090–27098. [[CrossRef](#)]
169. Egelhofer, T.A.; Villen, J.; McCusker, D.; Gygi, S.P.; Kellogg, D.R. The Septins Function in G1 Pathways That Influence the Pattern of Cell Growth in Budding Yeast. *PLoS ONE* **2008**, *3*, e2022. [[CrossRef](#)]
170. Shulewitz, M.J.; Inouye, C.J.; Thorner, J. Hsl7 Localizes to a Septin Ring and Serves as an Adapter in a Regulatory Pathway That Relieves Tyrosine Phosphorylation of Cdc28 Protein Kinase in *Saccharomyces Cerevisiae*. *Mol. Cell Biol.* **1999**, *19*, 7123–7137. [[CrossRef](#)]
171. Thomas, C.L.; Blacketer, M.J.; Edgington, N.P.; Myers, A.M. Assembly Interdependence among the *S. Cerevisiae* Bud Neck Ring Proteins Elm1p, Hsl1p and Cdc12p. *Yeast* **2003**, *20*, 813–826. [[CrossRef](#)] [[PubMed](#)]
172. Finnigan, G.C.; Duvalyan, A.; Liao, E.N.; Sargsyan, A.; Thorner, J. Detection of Protein-Protein Interactions at the Septin Collar in *Saccharomyces Cerevisiae* Using a Tripartite Split-GFP System. *Mol. Biol. Cell* **2016**, *27*, 2708–2725. [[CrossRef](#)] [[PubMed](#)]
173. Barral, Y.; Parra, M.; Bidlingmaier, S.; Snyder, M. Nim1-Related Kinases Coordinate Cell Cycle Progression with the Organization of the Peripheral Cytoskeleton in Yeast. *Genes. Dev.* **1999**, *13*, 176–187. [[CrossRef](#)] [[PubMed](#)]

174. Breikreutz, A.; Choi, H.; Sharom, J.R.; Boucher, L.; Neduva, V.; Larsen, B.; Lin, Z.Y.; Breikreutz, B.J.; Stark, C.; Liu, G.; et al. A Global Protein Kinase and Phosphatase Interaction Network in Yeast. *Science* **2010**, *328*, 1043–1046. [[CrossRef](#)]
175. Finnigan, G.C.; Sterling, S.M.; Duvalyan, A.; Liao, E.N.; Sargsyan, A.; Garcia, G.; Nogales, E.; Thorner, J. Coordinate Action of Distinct Sequence Elements Localizes Checkpoint Kinase Hsl1 to the Septin Collar at the Bud Neck in *Saccharomyces Cerevisiae*. *Mol. Biol. Cell* **2016**, *27*, 2213–2233. [[CrossRef](#)]
176. Okuzaki, D.; Nojima, H. Kcc4 Associates with Septin Proteins of *Saccharomyces Cerevisiae*. *FEBS Lett.* **2001**, *489*, 197–201. [[CrossRef](#)]
177. Bouquin, N.; Barral, Y.; Courbeyrette, R.; Blondel, M.; Snyder, M.; Mann, C. Regulation of Cytokinesis by the Elm1 Protein Kinase in *Saccharomyces Cerevisiae*. *J. Cell Sci.* **2000**, *113 Pt 8*, 1435–1445. [[CrossRef](#)]
178. Marquardt, J.; Yao, L.-L.; Okada, H.; Svitkina, T.; Bi, E. The LKB1-like Kinase Elm1 Controls Septin Hourglass Assembly and Stability by Regulating Filament Pairing. *Curr. Biol.* **2020**, *30*, 2386–2394.e4. [[CrossRef](#)]
179. Sreenivasan, A.; Kellogg, D. The Elm1 Kinase Functions in a Mitotic Signaling Network in Budding Yeast. *Mol. Cell Biol.* **1999**, *19*, 7983–7994. [[CrossRef](#)]
180. Marquardt, J.; Chen, X.; Bi, E. Reciprocal Regulation by Elm1 and Gin4 Controls Septin Hourglass Assembly and Remodeling. *J. Cell Biol.* **2024**, *223*, e202308143. [[CrossRef](#)]
181. Szkotnicki, L.; Crutchley, J.M.; Zyla, T.R.; Bardes, E.S.; Lew, D.J. The Checkpoint Kinase Hsl1p Is Activated by Elm1p-Dependent Phosphorylation. *Mol. Biol. Cell* **2008**, *19*, 4675–4686. [[CrossRef](#)]
182. Fraschini, R.; Bilotta, D.; Lucchini, G.; Piatti, S. Functional Characterization of Dma1 and Dma2, the Budding Yeast Homologues of *Schizosaccharomyces Pombe* Dma1 and Human Chfr. *Mol. Biol. Cell* **2004**, *15*, 3796–3810. [[CrossRef](#)] [[PubMed](#)]
183. Merlini, L.; Fraschini, R.; Boettcher, B.; Barral, Y.; Lucchini, G.; Piatti, S. Budding Yeast Dma Proteins Control Septin Dynamics and the Spindle Position Checkpoint by Promoting the Recruitment of the Elm1 Kinase to the Bud Neck. *PLoS Genet.* **2012**, *8*, e1002670. [[CrossRef](#)]
184. Patasi, C.; Godocikova, J.; Michlikova, S.; Nie, Y.; Kacerikova, R.; Kvalova, K.; Raunser, S.; Farkasovsky, M. The Role of Bni5 in the Regulation of Septin Higher-Order Structure Formation. *Biol. Chem.* **2015**, *396*, 1325–1337. [[CrossRef](#)] [[PubMed](#)]
185. Lee, P.R.; Song, S.; Ro, H.S.; Park, C.J.; Lippincott, J.; Li, R.; Pringle, J.R.; De Virgilio, C.; Longtine, M.S.; Lee, K.S. Bni5p, a Septin-Interacting Protein, Is Required for Normal Septin Function and Cytokinesis in *Saccharomyces Cerevisiae*. *Mol. Cell Biol.* **2002**, *22*, 6906–6920. [[CrossRef](#)]
186. Booth, E.A.; Sterling, S.M.; Dovala, D.; Nogales, E.; Thorner, J. Effects of Bni5 Binding on Septin Filament Organization. *J. Mol. Biol.* **2016**, *428*, 4962–4980. [[CrossRef](#)]
187. Finnigan, G.C.; Booth, E.A.; Duvalyan, A.; Liao, E.N.; Thorner, J. The Carboxy-Terminal Tails of Septins Cdc11 and Shs1 Recruit Myosin-II Binding Factor Bni5 to the Bud Neck in *Saccharomyces Cerevisiae*. *Genetics* **2015**, *200*, 843–862. [[CrossRef](#)]
188. Touati, S.A.; Kataria, M.; Jones, A.W.; Snijders, A.P.; Uhlmann, F. Phosphoproteome Dynamics during Mitotic Exit in Budding Yeast. *EMBO J.* **2018**, *37*, e98745. [[CrossRef](#)] [[PubMed](#)]
189. Renz, C.; Oeljeklaus, S.; Grinhagens, S.; Warscheid, B.; Johnsson, N.; Gronemeyer, T. Identification of Cell Cycle Dependent Interaction Partners of the Septins by Quantitative Mass Spectrometry. *PLoS ONE* **2016**, *21*, e0148340. [[CrossRef](#)]
190. Swaney, D.L.; Beltrao, P.; Starita, L.; Guo, A.; Rush, J.; Fields, S.; Krogan, N.J.; Villen, J. Global Analysis of Phosphorylation and Ubiquitylation Cross-Talk in Protein Degradation. *Nat. Methods* **2013**, *10*, 676–682. [[CrossRef](#)]
191. Albuquerque, C.P.; Smolka, M.B.; Payne, S.H.; Bafna, V.; Eng, J.; Zhou, H. A Multidimensional Chromatography Technology for In-Depth Phosphoproteome Analysis. *Mol. Cell. Proteom. MCP* **2008**, *7*, 1389–1396. [[CrossRef](#)] [[PubMed](#)]
192. Holt, L.J.; Tuch, B.B.; Villen, J.; Johnson, A.D.; Gygi, S.P.; Morgan, D.O. Global Analysis of Cdk1 Substrate Phosphorylation Sites Provides Insights into Evolution. *Science* **2009**, *325*, 1682–1686. [[CrossRef](#)] [[PubMed](#)]
193. Smolka, M.B.; Albuquerque, C.P.; Chen, S.H.; Zhou, H. Proteome-Wide Identification of in Vivo Targets of DNA Damage Checkpoint Kinases. *Proc. Natl. Acad. Sci. USA* **2007**, *104*, 10364–10369. [[CrossRef](#)]
194. Bodenmiller, B.; Wanka, S.; Kraft, C.; Urban, J.; Campbell, D.; Pedrioli, P.G.; Gerrits, B.; Picotti, P.; Lam, H.; Vitek, O.; et al. Phosphoproteomic Analysis Reveals Interconnected System-Wide Responses to Perturbations of Kinases and Phosphatases in Yeast. *Sci. Signal.* **2010**, *3*, rs4. [[CrossRef](#)]
195. Soufi, B.; Kelstrup, C.D.; Stoehr, G.; Frohlich, F.; Walther, T.C.; Olsen, J.V. Global Analysis of the Yeast Osmotic Stress Response by Quantitative Proteomics. *Mol. BioSyst.* **2009**, *5*, 1337–1346. [[CrossRef](#)] [[PubMed](#)]
196. Li, J.; Paulo, J.A.; Nusinow, D.P.; Huttlin, E.L.; Gygi, S.P. Investigation of Proteomic and Phosphoproteomic Responses to Signaling Network Perturbations Reveals Functional Pathway Organizations in Yeast. *Cell Rep.* **2019**, *29*, 2092–2104.e4. [[CrossRef](#)]
197. Lanz, M.C.; Yugandhar, K.; Gupta, S.; Sanford, E.J.; Faça, V.M.; Vega, S.; Joiner, A.M.N.; Fromme, J.C.; Yu, H.; Smolka, M.B. In-Depth and 3-Dimensional Exploration of the Budding Yeast Phosphoproteome. *EMBO Rep.* **2021**, *22*, e51121. [[CrossRef](#)]
198. Johnson, E.S.; Blobel, G. Cell Cycle-Regulated Attachment of the Ubiquitin-Related Protein SUMO to the Yeast Septins. *J. Cell Biol.* **1999**, *147*, 981–994. [[CrossRef](#)]
199. Mitchell, L.; Lau, A.; Lambert, J.P.; Zhou, H.; Fong, Y.; Couture, J.F.; Figeys, D.; Baetz, K. Regulation of Septin Dynamics by the *Saccharomyces Cerevisiae* Lysine Acetyltransferase NuA4. *PLoS ONE* **2011**, *6*, e25336. [[CrossRef](#)]
200. Chen, X.; Wang, K.; Svitkina, T.; Bi, E. Critical Roles of a RhoGEF-Anillin Module in Septin Architectural Remodeling during Cytokinesis. *Curr. Biol.* **2020**, *30*, 1477–1490.e3. [[CrossRef](#)]

201. Kang, P.J.; Hood-DeGrenier, J.K.; Park, H.O. Coupling of Septins to the Axial Landmark by Bud4 in Budding Yeast. *J. Cell Sci.* **2013**, *126*, 1218–1226. [[CrossRef](#)] [[PubMed](#)]
202. Wu, H.; Guo, J.; Zhou, Y.T.; Gao, X.D. The Anillin-Related Region of Bud4 Is the Major Functional Determinant for Bud4's Function in Septin Organization during Bud Growth and Axial Bud Site Selection in Budding Yeast. *Eukaryot. Cell* **2015**, *14*, 241–251. [[CrossRef](#)] [[PubMed](#)]
203. Chant, J.; Herskowitz, I. Genetic Control of Bud Site Selection in Yeast by a Set of Gene Products That Constitute a Morphogenetic Pathway. *Cell* **1991**, *65*, 1203–1212. [[CrossRef](#)]
204. Guo, J.; Gong, T.; Gao, X.-D. Identification of an Amphipathic Helix Important for the Formation of Ectopic Septin Spirals and Axial Budding in Yeast Axial Landmark Protein Bud3p. *PLoS ONE* **2011**, *6*, e16744. [[CrossRef](#)]
205. Schrock, M.N.; Yan, Y.; Goeckel, M.E.; Basgall, E.M.; Lewis, I.C.; Leonard, K.G.; Halloran, M.; Finnigan, G.C. Characterization of Bud3 Domains Sufficient for Bud Neck Targeting in *S. Cerevisiae*. *Access Microbiol.* **2022**, *4*, 000341. [[CrossRef](#)]
206. Stegmeier, F.; Amon, A. Closing Mitosis: The Functions of the Cdc14 Phosphatase and Its Regulation. *Annu. Rev. Genet.* **2004**, *38*, 203–232. [[CrossRef](#)]
207. Jaspersen, S.L.; Morgan, D.O. Cdc14 Activates Cdc15 to Promote Mitotic Exit in Budding Yeast. *Curr. Biol.* **2000**, *10*, 615–618. [[CrossRef](#)]
208. König, C.; Maekawa, H.; Schiebel, E. Mutual Regulation of Cyclin-Dependent Kinase and the Mitotic Exit Network. *J. Cell Biol.* **2010**, *188*, 351–368. [[CrossRef](#)] [[PubMed](#)]
209. Visintin, R.; Craig, K.; Hwang, E.S.; Prinz, S.; Tyers, M.; Amon, A. The Phosphatase Cdc14 Triggers Mitotic Exit by Reversal of Cdk-Dependent Phosphorylation. *Mol. Cell* **1998**, *2*, 709–718. [[CrossRef](#)] [[PubMed](#)]
210. Gray, C.H.; Good, V.M.; Tonks, N.K.; Barford, D. The Structure of the Cell Cycle Protein Cdc14 Reveals a Proline-Directed Protein Phosphatase. *Embo J.* **2003**, *22*, 3524–3535. [[CrossRef](#)]
211. Miller, D.P.; Hall, H.; Chaparian, R.; Mara, M.; Mueller, A.; Hall, M.C.; Shannon, K.B. Dephosphorylation of Iqg1 by Cdc14 Regulates Cytokinesis in Budding Yeast. *Mol. Biol. Cell* **2015**, *26*, 2913–2926. [[CrossRef](#)] [[PubMed](#)]
212. Bloom, J.; Cristea, I.M.; Procko, A.L.; Lubkov, V.; Chait, B.T.; Snyder, M.; Cross, F.R. Global Analysis of Cdc14 Phosphatase Reveals Diverse Roles in Mitotic Processes*. *J. Biol. Chem.* **2011**, *286*, 5434–5445. [[CrossRef](#)] [[PubMed](#)]
213. Palani, S.; Meitinger, F.; Boehm, M.E.; Lehmann, W.D.; Pereira, G. Cdc14-Dependent Dephosphorylation of Inn1 Contributes to Inn1–Cyk3 Complex Formation. *J. Cell Sci.* **2012**, *125*, 3091–3096. [[CrossRef](#)] [[PubMed](#)]
214. Shou, W.; Seol, J.H.; Shevchenko, A.; Baskerville, C.; Moazed, D.; Chen, Z.W.; Jang, J.; Charbonneau, H.; Deshaies, R.J. Exit from Mitosis Is Triggered by Tem1-Dependent Release of the Protein Phosphatase Cdc14 from Nucleolar RENT Complex. *Cell* **1999**, *97*, 233–244. [[CrossRef](#)]
215. Kuilman, T.; Maiolica, A.; Godfrey, M.; Scheidel, N.; Aebersold, R.; Uhlmann, F. Identification of Cdk Targets That Control Cytokinesis. *EMBO J.* **2015**, *34*, 81–96. [[CrossRef](#)]
216. Varela Salgado, M.; Adriaans, I.E.; Touati, S.A.; Ibanes, S.; Lai-Kee-Him, J.; Ancelin, A.; Cipelletti, L.; Picas, L.; Piatti, S. Phosphorylation of the F-BAR Protein Hof1 Drives Septin Ring Splitting in Budding Yeast. *Nat. Commun.* **2024**, *15*, 3383. [[CrossRef](#)]
217. Meitinger, F.; Boehm, M.E.; Hofmann, A.; Hub, B.; Zentgraf, H.; Lehmann, W.D.; Pereira, G. Phosphorylation-Dependent Regulation of the F-BAR Protein Hof1 during Cytokinesis. *Genes. Dev.* **2011**, *25*, 875–888. [[CrossRef](#)]
218. Meitinger, F.; Palani, S.; Hub, B.; Pereira, G. Dual Function of the NDR-Kinase Dbf2 in the Regulation of the F-BAR Protein Hof1 during Cytokinesis. *Mol. Biol. Cell* **2013**, *24*, 1290–1304. [[CrossRef](#)]
219. Fankhauser, C.; Reymond, A.; Cerutti, L.; Utzig, S.; Hofmann, K.; Simanis, V. The *S. Pombe* Cdc15 Gene Is a Key Element in the Reorganization of F-Actin at Mitosis. *Cell* **1995**, *82*, 435–444. [[CrossRef](#)]
220. Willet, A.H.; McDonald, N.A.; Bohnert, K.A.; Baird, M.A.; Allen, J.R.; Davidson, M.W.; Gould, K.L. The F-BAR Cdc15 Promotes Contractile Ring Formation through the Direct Recruitment of the Formin Cdc12. *J. Cell Biol.* **2015**, *208*, 391–399. [[CrossRef](#)]
221. Carnahan, R.H.; Gould, K.L. The PCH Family Protein, Cdc15p, Recruits Two F-Actin Nucleation Pathways to Coordinate Cytokinetic Actin Ring Formation in *Schizosaccharomyces Pombe*. *J. Cell Biol.* **2003**, *162*, 851–862. [[CrossRef](#)] [[PubMed](#)]
222. Graziano, B.R.; Yu, H.Y.; Alioto, S.L.; Eskin, J.A.; Ydenberg, C.A.; Waterman, D.P.; Garabedian, M.; Goode, B.L. The F-BAR Protein Hof1 Tunes Formin Activity to Sculpt Actin Cables during Polarized Growth. *Mol. Biol. Cell* **2014**, *25*, 1730–1743. [[CrossRef](#)]
223. Garabedian, M.V.; Wirshing, A.; Vakhrusheva, A.; Turegun, B.; Sokolova, O.S.; Goode, B.L. A Septin-Hof1 Scaffold at the Yeast Bud Neck Binds and Organizes Actin Cables. *Mol. Biol. Cell* **2020**, *31*, 1988–2001. [[CrossRef](#)]
224. Garabedian, M.V.; Stanishneva-Konovalova, T.; Lou, C.; Rands, T.J.; Pollard, L.W.; Sokolova, O.S.; Goode, B.L. Integrated Control of Formin-Mediated Actin Assembly by a Stationary Inhibitor and a Mobile Activator. *J. Cell Biol.* **2018**, *217*, 3512–3530. [[CrossRef](#)]
225. Bohnert, K.A.; Gould, K.L. Cytokinesis-Based Constraints on Polarized Cell Growth in Fission Yeast. *PLoS Genet.* **2012**, *8*, e1003004. [[CrossRef](#)] [[PubMed](#)]
226. Oh, Y.; Schreiter, J.; Nishihama, R.; Wloka, C.; Bi, E. Targeting and Functional Mechanisms of the Cytokinesis-related F-BAR Protein Hof1 during the Cell Cycle. *Mol. Biol. Cell* **2013**, *24*, 1305–1320. [[CrossRef](#)] [[PubMed](#)]
227. Frost, A.; Unger, V.M.; De Camilli, P. The BAR Domain Superfamily: Membrane-Molding Macromolecules. *Cell* **2009**, *137*, 191–196. [[CrossRef](#)]
228. Suetsugu, S.; Toyooka, K.; Senju, Y. Subcellular Membrane Curvature Mediated by the BAR Domain Superfamily Proteins. *Semin. Cell Dev. Biol.* **2010**, *21*, 340–349. [[CrossRef](#)]

-
229. Zhao, H.; Michelot, A.; Koskela, E.V.; Tkach, V.; Stamou, D.; Drubin, D.G.; Lappalainen, P. Membrane-Sculpting BAR Domains Generate Stable Lipid Microdomains. *Cell Rep.* **2013**, *4*, 1213–1223. [[CrossRef](#)]
230. Picas, L.; Viaud, J.; Schauer, K.; Vanni, S.; Hnia, K.; Fraiser, V.; Roux, A.; Bassereau, P.; Gaits-Iacovoni, F.; Payrastre, B.; et al. BIN1/M-Amphiphysin2 Induces Clustering of Phosphoinositides to Recruit Its Downstream Partner Dynammin. *Nat. Commun.* **2014**, *5*, 5647. [[CrossRef](#)]

Disclaimer/Publisher’s Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.