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Review

Incomplete abscission and cytoplasmic bridges in the evolution of eukaryotic multicellularity

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SUMMARY

The textbook view of cell division terminates with the final separation of the two daughter cells in the process called abscission. However, in contrast to this classical view, a variety of cell types in multicellular organisms are connected through cytoplasmic bridges, which most often form by incomplete abscission or — more rarely — by local fusion of plasma membranes. In this review, we survey the distribution, function, and formation of cytoplasmic bridges across the eukaryotic tree of life. We find that cytoplasmic bridges are widespread, and were likely ancestrally present, in almost all lineages of eukaryotes with clonal multicellularity — including the five 'complex multicellular' lineages: animals, fungi, land plants, red algae, and brown algae. In animals, cytoplasmic bridges resulting from incomplete abscission are ubiquitous in the germline and common in pluripotent cell types. Although cytoplasmic bridges have been less studied than other structural mediators of multicellularity (such as adhesion proteins and extracellular matrix), we propose that they have played a pivotal role in the repeated evolution of eukaryotic clonal multicellularity — possibly by first performing a structural role and later by allowing exchange of nutrients and/or intercellular communication, which notably buffered cell–cell competition by averaging gene expression. Bridges were eventually lost from many animal tissues in concert with the evolution of spatial cell differentiation, cell motility within the organism, and other mechanisms for intercellular distribution of signals and metabolites. Finally, we discuss the molecular basis for the evolution of incomplete abscission and examine the alternative hypotheses of single or multiple origins.

Introduction

Animal cell division results from two parallel processes: the separation of nuclear components, called mitosis, and the separation of the membrane and cytoplasmic components, called cytokinesis[1]. Mitosis proceeds in four steps: prophase, metaphase, anaphase and telophase. In the first three steps, chromosomes condense, a microtubule spindle aligns them, and the two sets of sister chromatids are separated and pulled to opposite poles of the cell. During telophase, nuclear envelopes reform around both daughter nuclei. Cytokinesis temporally overlaps with mitosis: it canonically starts in anaphase and extends after telophase[1]. During early cytokinesis, the future daughter cells are separated by constriction of an actomyosin ring, or 'primary constriction'[2]. The bridge thus formed is further narrowed by a 'secondary constriction' accompanied and possibly mediated by anchoring of the midbody (an electron-dense complex of proteins traversed by microtubules in the spindle remnant) to the plasma membrane[2]. Finally, in the last phase of cytokinesis, a specialized machinery (which includes notably the ESCRT (endosomal sorting complex required for transport) complex) cuts the tube of plasma membrane connecting the daughter cells. This severing process is called 'abscission'[2,3].

While the beginning of cell division, from prophase to early cytokinesis, collectively usually lasts around an hour[4], abscission is often much slower. For example, in mammalian cells in culture, abscission is completed a few hours after the end of primary constriction, resulting in sister cells connected for a long time through cytoplasmic bridges[5]. Beyond cell culture, cytoplasmic bridges are surprisingly widespread in multicellular organisms[6]. Here, we review the diversity of eukaryotic species and developmental stages where abscission is delayed or incomplete, leading to long-lasting bridges. We show that incomplete abscission is present in almost all lineages of eukaryotes with clonal multicellularity (i.e. that develop by serial division without sister-cell separation). Notably, cytoplasmic bridges resulting from incomplete abscission are present in all five known instances of 'complex multicellularity', defined as multicellularity involving cell differentiation, communication between cells, and a complex three-dimensional organization in which some cells are not in direct contact with the environment: animals, fungi, land plants, red algae, and brown algae[7] (Figure 1).

There are three groups of eukaryotes: single-celled forms (in which cytokinesis is complete, ensuring rapid re-establishment of the unicellular lifestyle after division), multicellular forms that develop by aggregation of initially separate cells (such as *Dictyostelium discoideum*), and clonally multicellular forms where multicellularity is established by serial cell division of an initial cell without sister-cell separation. Even though bridges are

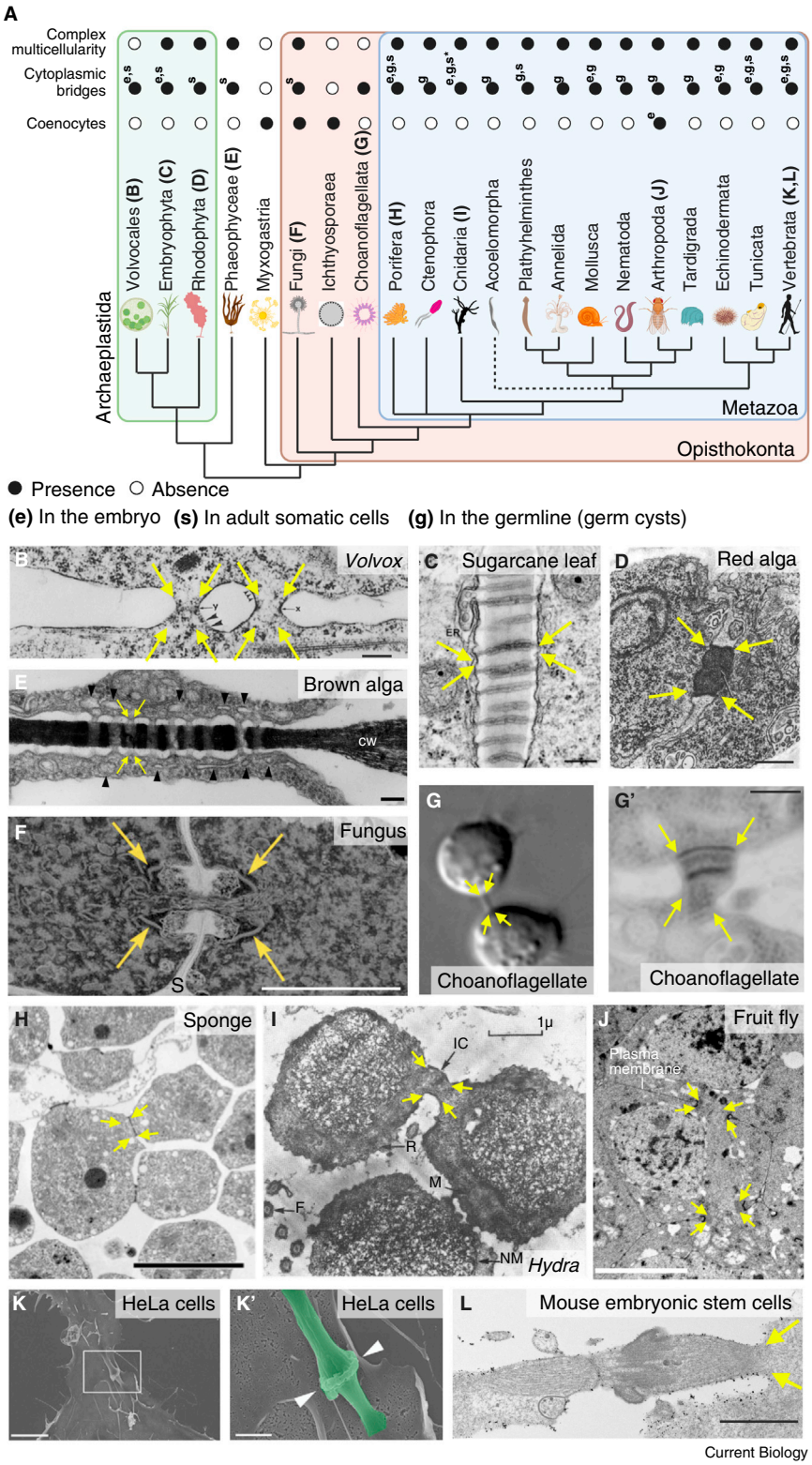

Figure 1. Cytoplasmic bridges are common in clonally multicellular eukaryotes.
(A) Phylogenetic distribution of cytoplasmic bridges in clonally multicellular eukaryotes. All organism silhouettes are from the BioRender library except *Hydra vulgaris* (Cnidaria, from http://phylopic.org/), *Volvox carteri* (Volvocales, from https://www.vecteezy.com/), Ichthyosporaea (from[187]) and Rhodophyta and Choanoflagellata (custom-drawn). (B–K) Morphology of cytoplasmic bridges. In all panels, yellow arrows indicate connections of the bridge to the plasma membranes of both neighboring cells. (B) Bridges in *Volvox carteri*. Scale bar: 0.25 μm. (Reproduced with permission from[130].) (C) Plasmodesmata in a sugarcane leaf. (Reproduced with permission from Springer[188].) ER, endoplasmic reticulum. Scale bar: 200 nm. (D) Plugged junction in the marine red alga *Pseudogloiophloea confuse*. (Reproduced from Wiley (CC BY 3.0)[103].) Scale bar: 0.5 μm. (E) Plasmodesmata in the brown alga *Saccharina japonica*. (Reproduced with permission from Springer Nature (CC BY 4.0)[107].) Arrowheads: membranous structures near the plasmodesmata. cw, cell wall. Scale bar: 100 nm. (F) Septal pore in the filamentous fungus *Rhizoctonia solani*. Arrows: septal pore caps that flank the septum (S). (Reproduced from Wiley (CC BY 3.0)[189].) Scale bar: 500 nm. (G) Two choanoflagellates (*Salpingoeca rosetta*) linked by a bridge. (G′) *Salpingoeca rosetta* bridge observed by TEM. Scale bar: 200 nm. (Reproduced from Dayel *et al.*[116].) (H) Plugged junctions in the hexactinellid sponge *Rhabdocalyptus dawsoni*. Scale bar: 1 μm. (Image courtesy of Sally Leys, University of Alberta.) (I) Bridge (interconnection, IC) between two interstitial cells of *Hydra* differentiating into spermatozoa. NM, nuclear membrane; M, mitochondria; R, ribosomes; F, flagellum. Scale bar: 1 μm. (Reproduced from University of Windsor[190].) (J) Ring canals in the *Drosophila melanogaster* ovary. Ultra-thin sections of wild-type germarium observed by TEM. Scale bar: 8 μm. (Image courtesy of Jean-René Huynh, CIRB, Collège de France.) (K) Bridge between two HeLa cells observed by SEM. Scale bar: 4 μm. (Reproduced with permission from[191].) (K′) Bridge between two HeLa cells observed by SEM, higher magnification (green). Arrowheads point to the midbody. Scale bar: 1 μm. (Reproduced with permission from the *Journal of Cell Science*[191].) (L) Bridge between two pluripotent mouse embryonic stem cells. Scale bar: 1 μm.

near-universally present in clonally multicellular eukaryotes, they are essentially absent from the two other types of eukaryotes. Importantly, these three morphotypes (unicellular, aggregative multicellular, and clonal multicellular) do not correspond to monophyletic clades, but are scattered across the whole eukaryotic tree of life[8,9]. As such, it is traditionally assumed that the last eukaryotic common ancestor was unicellular, with both aggregative and clonal multicellularity having evolved independently multiple times. Taken together, these observations raise an intriguing possibility: that cytoplasmic bridges might have repeatedly served as a basis for the recurrent evolution of clonal multicellularity across eukaryotes by allowing cells to remain attached after division.

In this review, we document this pattern and discuss possible explanations. We first map the phylogenetic

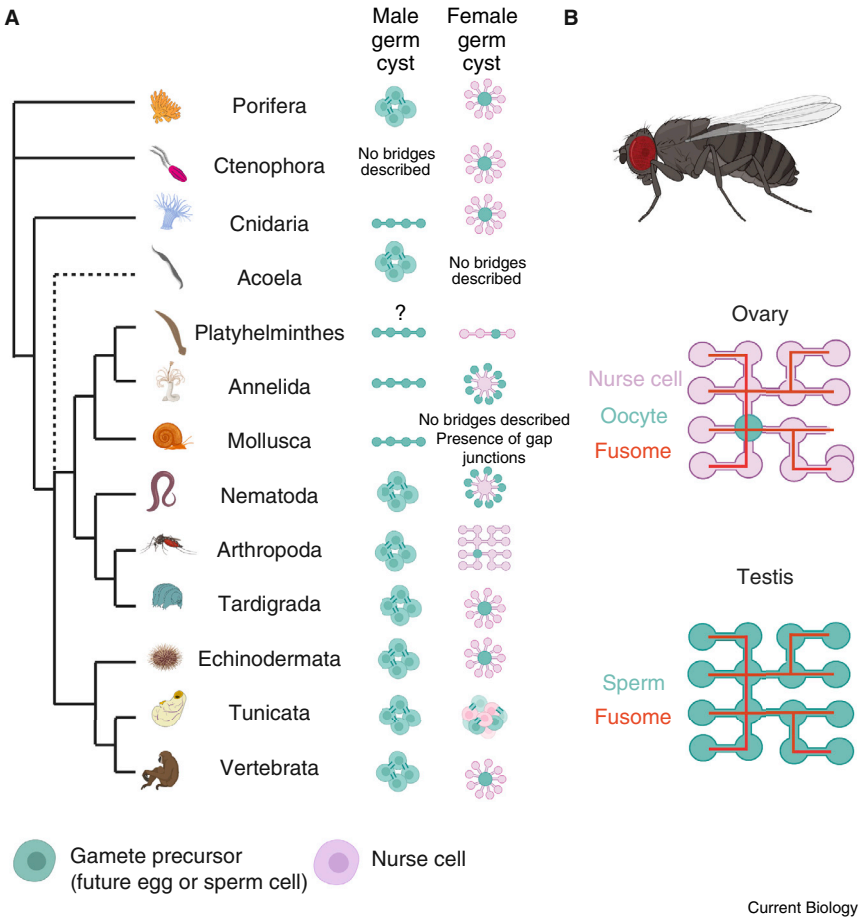

Figure 2. Cytoplasmic bridges in the germline.
(A) Morphology of germline cysts in metazoans (Table S1 and references therein). (B) Examples of the *Drosophila melanogaster* germline; in the male and the female germline, cells divide successively leading to the formation of a cluster 'cyst' of 16 cells. In the female, only the central cell with four bridges becomes an oocyte and all surrounding cells are nursing cells. In the male, all cells become sperm.

distribution of cytoplasmic bridges across clonal multicellular forms. We then propose that cytoplasmic bridges were ancestrally present in all complex multicellular clades (including animals), in which they might have served as the first structural basis for multicellularity before the evolution of specialized mechanisms for cell–cell cohesion. We discuss potential regulatory functions for the bridges, including controlled cell–cell communication, control of developmental progression and buffering of intercellular competition. Finally, we discuss the molecular basis for incomplete abscission and propose that the 'genetic toolkit' of abscission regulators in the last common eukaryotic ancestor gave it the potential to easily — and repeatedly — evolve cytoplasmic bridges.

Cytoplasmic bridges across eukaryotic multicellularity

A look at the eukaryotic tree of life shows that cytoplasmic bridges are surprisingly widespread across multicellular eukaryotes (Figure 1A): they are present in each of the five groups with complex multicellularity (animals, land plants, fungi, red algae, and brown algae[7]) as well as in multiple models of 'simple multicellularity' (i.e. multicellularity without communication between cells and in which all cells are directly exposed to their environment) such as choanoflagellates, ulvophytes, and volvocale algae. In all of these clades, cells are connected by bridges whose defining feature is the continuity of the plasma membrane between cells (Figure 1B–K) but which otherwise vary both in number and in internal structure: some bridges contain microtubules and a midbody (typically in cultured animal cells and animal embryos; Figure 1K,L) while others are devoid of midbodies and have lost their microtubule network (typically animal germ cells; Figure 1J), allowing passage of molecules and even organelles between cells. In this section, we will discuss the presence of cytoplasmic bridges across clonally multicellular eukaryotes, starting with animals, followed by other complex multicellular eukaryotes. Of note, we will discuss different time scales of cytoplasmic bridge stability, from relatively short term (as in embryos or stem cells that maintain bridges for hours but eventually cut them), to long term (as in germline bridges that can be maintained for days).

Cytoplasmic bridges in animals

Virtually every animal species investigated so far displays cytoplasmic bridges in at least one tissue: the germline, where germ cells are linked by bridges into structures called 'germ cysts' (Figures 1J and 2)[10]. Germ cysts have been reported in the male germline of all species studied (except ctenophores; Figure 2A) and are also common in the female germline (except in a few species of arthropods, annelids and mollusks; Table S1 in Supplemental information, published with this article online)[11–49]. All cells within male germ cysts eventually develop into spermatozoa, while cells in female germ cysts develop either into oocytes or into nurse cells. The structure of female germ cysts is variable and observed configurations include strings of cells[11], branched clusters[12] or monolayers of oocytes connected to a central cytoplasmic core by one bridge each[28] (Figure 2A and Table S1). In the few species where bridges are absent from the female germline, all germ cells can become oocytes[28,50].

The best-studied germ cysts are those of insects[26], in which cytoplasmic bridges are classically called 'ring canals' and connect cells dividing synchronously in a cyst. Ring canals form by incomplete cytokinesis, as long deduced from electron microscopic observation of fixed material covering different stages in their development[51,52] and since confirmed by live imaging[53].

In spite of their formation by incomplete cytokinesis, ring canals are devoid of microtubules or a midbody[51]. Instead, they contain an electron-dense rim of filamentous actin stabilized by cross-linkers such as filamin[54–56] as well as an atypical membrane-bound organelle, the fusome[57], which mediates intercellular continuity of the endoplasmic reticulum (ER) across the cyst[58] (Figure 1B). Ring canal formation is best understood in the fruit fly *Drosophila melanogaster* (Figure 2), where it involves premature arrest of the actomyosin-mediated primary constriction[24]. In *Drosophila* females, cyst formation is initiated by an asymmetric division of the germ stem cells (GSC), which undergo complete but slow abscission (10–12 h). One of the resulting daughter cells then divides four times to form a 16-cell cyst with a stereotypical organization, in which two cells have four ring canals, two have three, four have two and eight have one (Figure 2). Intriguingly, the oocyte always derives from one of the two cells with four ring canals[23]. The other cyst cells enter meiosis, but arrest early and become nurse cells[22]. Although germline bridges across animals usually lack microtubules and a midbody, a few exceptions are known: microtubules are present in the male germline bridges in mice[59], and a midbody persists in female bridges in tardigrades[20]. This shows that, although germline bridges are conserved, their ultrastructure can change during evolution.

Beyond germ cells, cytoplasmic bridges are often present in cells with high developmental potential — including blastomeres (the first cells formed by embryonic cleavage), embryonic stem cells, and some adult pluripotent stem cells. In mammals, cytoplasmic bridges are present at least up to the preimplantation blastocyst in humans[60] and mice[61,62]. Live imaging in the mouse embryo has shown that the bridges contain microtubules whose minus ends are anchored within the bridge while their plus ends extend into the cytoplasm of both sister cells[62]. Bridges connect all pairs of sister cells, and disassemble when the next division starts. Cytoplasmic bridges are also found in naïve mouse embryonic stem cells[63] (Figure 1L), which resemble cells of the inner cell mass of the peri-implantation blastocyst (a later embryonic stage)[64]. Intriguingly, the duration of abscission and therefore the number of bridges must decrease to allow developmental progression[63]. Outside mammals, bridges also connect blastomeres in zebrafish[65,66], sea anemone[67], and squid embryos[68].

Adult pluripotent stem cells can also be linked by cytoplasmic bridges. In the cnidarian *Hydra*, interstitial cells that can regenerate all cell types of the adult body occur in pairs linked by bridges resulting from incomplete abscission (Figure 1I). Interstitial cells give rise to precursors that divide synchronously (still with incomplete abscission) to form nests of 4, 8 or 16 cells linked by bridges, all of which differentiate into the same cell type[69]. Conversely, when interstitial cells divide with complete abscission, the two sister cells can differentiate into distinct cell types. In the ctenophore *Pleurobrachia pileus*, putative adult pluripotent stem cells expressing classic pluripotency factors such as *piwi*, *bruno* and *vasa* in the tentacle roots are connected by cytoplasmic bridges — but it is unknown whether they result from incomplete abscission[70]. In other animals (including tunicates, annelids, planarians, sponges and acoels), *piwi*-positive adult pluripotent stem cells often occur in clusters, but it is unclear whether they are linked by bridges[71–81].

Finally, cytoplasmic bridges are rare between terminally differentiated cells, but a few examples are known. In the *Drosophila* egg chamber, somatic follicular cells are connected by permeable ring canals[82,83]. Bridges can connect pairs of neurosensory cells or cnidoblasts in *Hydra*[84] or pairs of red blood cell precursors in mice[85]. In mammals, Gonadotropin-releasing hormone (GnrH)-secreting neurons are connected by bridges — which might form through cell–cell fusion rather than incomplete abscission, although live imaging data are lacking[86]. Finally, an extreme example is provided by glass sponges, in which a multinucleated connective tissue, the trabecular syncytium, is connected to multiple other cell types by open or closed cytoplasmic bridges (Figure 1H) which are thought to form by cell fusion[42,74,87].

Altogether, the broad taxonomic distribution of cytoplasmic bridges in animals supports the idea that they were present in the last common animal ancestor — at least in the germline, and possibly during early embryogenesis and between stem cells.

Cytoplasmic bridges in fungi, land plants, and algae

Cytoplasmic bridges are present not only in multicellular animals but also in the four other groups of complex multicellular eukaryotes — even though they all evolved complex multicellularity independently of each other[7]. In land plants, fungi, red algae and brown algae, almost all somatic cells are connected to at least some of their neighbors by cytoplasmic bridges called 'plasmodesmata' in land plants (Figure 1C) and in brown algae (Figure 1E), 'septal pores' in fungi (Figure 1F), and 'pit connections' in red algae (Figure 1D).

The structure of cytoplasmic bridges differs between these groups in a way that reflects their respective mode of cytokinesis. Fungi[88] and red algae[89] undergo cytokinesis by progressive membrane constriction (like animals) and incomplete constriction thus results in the maintenance of one cytoplasmic bridge between a given pair of sister cells. Filamentous fungi form 'hyphae', multicellular filaments composed of rows of cells linked by cytoplasmic bridges called septal pores[90] (Figure 1F). Septal pores result from constriction of a cytokinetic ring leaving a small pore open, as originally deduced from microscopic examination of fixed material[91,92] and confirmed by live imaging since then[93]. An extracellular wall is then deposited around the pore[94]. The pore is more or less selective depending on species and mediates intercellular transfers of ions and small molecules[95]. Septal pores are generally open but can be plugged through various mechanisms, for example, in response to wounding[96]. Septal pores are associated with membrane bound organelles (the Woronin bodies, which contribute to plugging[96,97]) and contain microtubules; indeed, septa have been suggested to anchor a microtubule-organizing center in *Aspergillus nidulans*[98]. In red algae, cells are connected by broad cytoplasmic bridges called pit connections or 'pit plugs' which are about one micron wide[99–102]. Examination of fixed material indicates that pit connections usually form by incomplete cell division[103,104] but occasionally form by cell fusion in a few species[102]. Pit connections contain a 'plug' of polysaccharides surrounded by a lipid bilayer, which likely imposes a size limit on diffusion but appears permeant to small molecules such as ions and amino acids[99].

In land plants and in brown algae, cytokinesis occurs by a fundamentally different mechanism than animal cytokinesis: fusion of secretory vesicles containing cell wall material at the site of cytokinesis[105]. Unlike ring constriction, this process can be interrupted in several sites at once, resulting in multiple

bridges — named plasmodesmata — between a single pair of sister cells (Figure 1C,E). Plasmodesmata formation has long been studied by electron microscopic observation of fixed samples in land plants[106] and brown algae[107] and has been more recently directly observed by live imaging in land plants[108]. In land plants, cell wall deposition is interrupted at sites where cortical endoplasmic reticulum stays trapped, which defines the positions of future plasmodesmata (Figure 1C). As a result, plasmodesmata contain an ER tubule (the desmotubule) continuous with the ER of both neighboring cells and surrounded by a cytoplasmic sleeve. Transfer through plasmodesmata is fundamental to plant development and physiology, and molecules transported include water, ions, nutrients, RNA and proteins[109–111]. Brown algae (which are not closely related to green or red algae[112]) possess plasmodesmata that are strikingly similar in structure and formation to those of land plants (but without a desmotubule) and that mediate long-distance transport of metabolites[99,101,102,107,113]. Importantly, in both land plants and brown algae, primary plasmodesmata formed by incomplete cytokinesis coexist with secondary plasmodesmata formed by cell–cell fusion[101,102].

The broad occurrence of cytoplasmic bridges in all five clades with complex multicellularity suggests two possible explanations: either bridges evolved late after multicellularity, in line with increasing complexity in those lineages (for example, to assist in intercellular communication); or they were already present at early, simple stages of the evolution of multicellularity, before spatial cell differentiation and complex anatomies appeared. How can we distinguish between these two options? A key source of information is the anatomy of eukaryotes with simple clonal multicellularity — and notably, those which are closely related to complex multicellular forms.

Cytoplasmic bridges in simple multicellular eukaryotes

Beyond the five groups with macroscopic, complex multicellularity, many eukaryotic lineages have evolved the ability to undergo serial cell division without separation of sister cells to give rise to simple multicellular colonies (i.e. where all cells appear identical and in direct contact with the environment). Interestingly, simple multicellularity is often found in the close relatives of complex multicellular clades, thus providing a potential proxy to the early stages of the evolution of multicellularity in those groups.

The closest living relatives of animals are the choanoflagellates, which can exist as single cells or undergo serial cell division to form colonies[114] (Figure 1G,G'). Interestingly, in most (but not all[115]) multicellular choanoflagellates, cells are linked by cytoplasmic bridges. Consistent with the idea that bridges form by incomplete abscission, only one bridge is observed between each pair of cells[116–118]. In the simplest forms, bridges are the only element maintaining stability of the colony, while in more complex colonies they are complemented by a shared extracellular matrix (ECM)[116,119–121]. Choanoflagellate cytoplasmic bridges have a stereotypic structure with two dense plates that resemble the animal midbody (but no microtubules), suggestive of a single origin of cytoplasmic bridges in choanoflagellates[8,117,122] and possibly of homology with those of animals. This also suggests that the last common ancestor of choanoflagellates and animals might have had a simple form of facultative multicellularity in which cells were linked (partly or exclusively) by bridges. Animal multicellularity would then have been elaborated and stabilized by the evolution of additional supporting elements, such as ECM[8] and cell–cell adhesions[123,124].

Similarly, the closest relatives of land plants comprise several clades of green algae with simple multicellularity, three of which have plasmodesmata resembling those of plants: Klebsormidiophyceae, Charophyceae and Coleochaetophyceae[99,101,102]. Only the immediate sister group of land plants (the Zygnematophyceae[125,126]) lacks plasmodesmata[102]. This has been interpreted as supporting a single origin of plasmodesmata in the simple algal ancestors of land plants (followed by loss in Zygnematophyceae[127]), although convergent evolution of plasmodesmata in these multiple groups cannot be fully ruled out[128].

Finally, cytoplasmic bridges are common in other groups of eukaryotes with simple clonal multicellularity (without complex relatives). These include ulvophytes[113] and volvocales[129], two independently evolved groups of green algae. The different species of volvocales span a remarkably complete range of steps in the evolution of multicellularity[129]. All known species possess cytoplasmic bridges that form through incomplete cytokinesis, as traditionally supported by electron microscopy of fixed samples[130] (although confocal live imaging has recently become possible[131]). The smallest multicellular volvocales (such as the 4-celled *Tetrabaena socialis*) display cytoplasmic bridges, formed by incomplete division, that persist during the entire life of the colony (Figure 1B)[132]. In species with larger colonies (ranging from the 16-celled *Gonium*[133] to *Volvox*[130,134,135] and *Astrephomene*[136] with hundreds of cells), bridges form by incomplete cytokinesis at early developmental stages but are lost later, and ECM eventually ensures intercellular cohesion alone. Volvocales thus might illustrate the initial acquisition of clonal multicellularity by incomplete abscission, and the eventual replacement of cytoplasmic bridges by more robust cohesion mechanisms during both evolution and development.

Multicellularity without cytoplasmic bridges and cytoplasmic bridges without multicellularity

Taken together, these data suggest that cytoplasmic bridges have been a frequent contributor to the recurrent evolution of clonal multicellularity in eukaryotes. However, exceptions exist. Cytoplasmic continuity has been taken to an extreme in a few groups that evolved multicellularity by dispensing with cytokinesis altogether and developing into large multinucleated cells, or a 'coenocyte': these include the slime mold *Physarum*[137], ichthyosporeans[138–140], and chytrid fungi[141]. Across the diversity of fungi, there is a continuum from coenocytes to full cellularization, and nuclei within hyphae are not always separated by septa[142–144]. However, cytoplasmic continuity is not always present, and at least one clonally multicellular clade, golden algae, seems to entirely lack cytoplasmic bridges[101,102]. Finally (and perhaps surprisingly), cytoplasmic bridges can exist in the absence of multicellularity: in the single-celled amoebae *Dictyostelium discoideum* and *Entamoeba invadens*, sister cells remain linked by a tubular tether that persists until it is mechanically severed by forces resulting either from cell crawling[145,146] or from the action of 'midwife cells'[147].

Altogether, the widespread presence of cytoplasmic bridges in multicellular organisms suggests that clonal multicellularity often evolves alongside cytoplasmic bridges and might sometimes start with bridges only. This broad distribution of

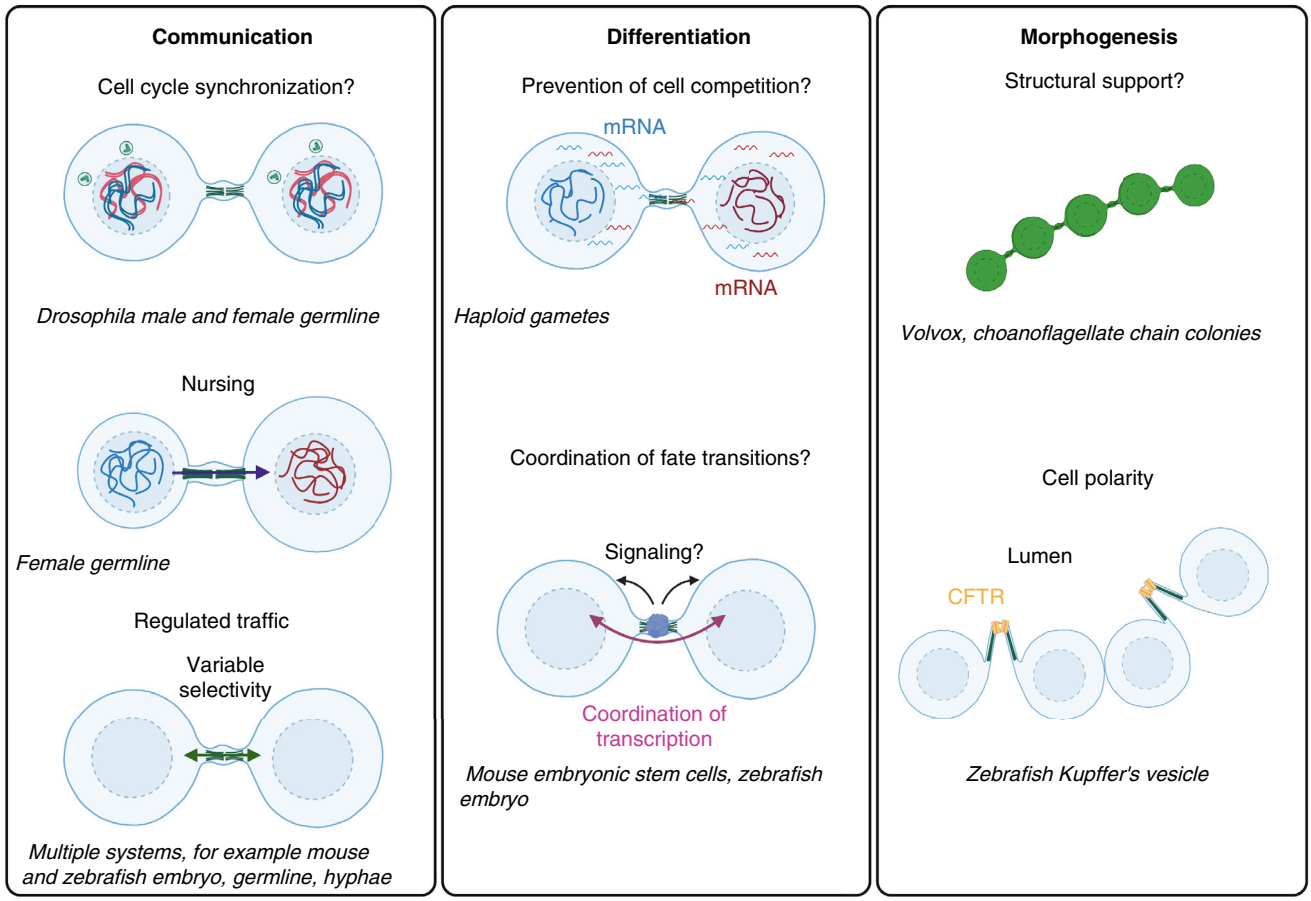

Figure 3. Hypothetical functions of cytoplasmic bridges.
Three possible functions for cytoplasmic bridges have been described: communication between cells (left, including synchronization of the cell cycle, nursing of specialized cells, and regulated traffic), regulation of differentiation (middle) including prevention of cell–cell competition and inhibition of differentiation, and morphogenesis (right) including mechanical support and participation in the establishment of cell and organ polarity.

cytoplasmic bridges suggests that they frequently play important functions in multicellular development and physiology.

Possible functions of cytoplasmic bridges

Structural support and morphogenesis

Bridges can ensure mechanical cohesion between linked cells (Figure 3, right panel). In *Volvox*, bridges likely maintain cohesion between the cells during embryo inversion[130,148]. In choanoflagellate chain colonies, bridges are the only element connecting cells, and are thus solely responsible for maintaining multicellularity[116]. Bridges allow colonial life that might confer adaptive advantages such as sharing of nutrients between cells or an increase in size, which could in turn prevent phagocytosis by predators like ciliates and amoebae that prefer single cells over large colonies[149]. Transport along bridges can contribute to aligning cell polarity between neighboring cells. For example, the proper positioning of the lumen in Kupffer's vesicle (an organ in the zebrafish embryo) depends on the repositioning of bridges after cell division which controls the targeting of a specific lumenogenesis protein to the apical membrane[150] (Figure 3, right panel).

Prevention of cell–cell competition by pooling gene expression

Any multicellular form is vulnerable to cheating genotypes. One possible role of bridges might be to pool gene expression between neighboring cells, thus preventing cheaters from taking over (Figure 3, middle panel). In animals, different genotypes coexist in the germline as a result of meiosis, making the risk of competition particularly elevated. A role in suppressing competition could explain, at least in part, the widespread presence of bridges in the germline across animals. There is evidence that bridges perform this function in the male germline: in mouse developing sperm cells, bridges have been shown to homogenize gene expression by transfer of mRNA and proteins, thus making haploid cells phenotypically diploid[151,152]. However, cheater genotypes have evolved evasion mechanisms preventing transport of their expression products through the bridges: this is true of segregation distorters such as the *t* haplotype in mice[153]. In somatic cells (which usually lack bridges), the expansion of cheater clones might instead more often be prevented by terminal differentiation, which permanently restricts proliferation potential.

Maintenance of pluripotency or coordination of differentiation

If bridges can homogenize gene expression state between cells, they might also prevent differentiation of cells connected to a pluripotent or multipotent progenitor. Thus, complete abscission might in some cases be necessary to initiate differentiation.

As discussed above, cytoplasmic bridges seem common in undifferentiated cells such as early embryonic cells, and several lines of evidence suggest that bridges are involved in the equilibration of cell state. In zebrafish embryos, exogenously expressed cytoplasmic fluorescent proteins are exchanged through cytoplasmic bridges between sister cells[65] and gene expression profiles are more similar between connected than between unconnected cells[66] (Figure 3, middle panel). In *Hydra*, cells do not differentiate until they have completed abscission from interstitial stem cells[69]. In mouse embryonic stem cells, a switch to fast abscission is necessary for loss of pluripotency[63]. Moreover, in several types of stem cells, inducing differentiation accelerates the release of midbodies by the cells into the surrounding medium[63,154]. Reciprocally, forcing retention of midbody remnants by downregulating autophagy increases the efficiency of reprogramming into inducible pluripotent stem cells[155].

In some cases, instead of preventing differentiation, cytoplasmic bridges connect differentiating cells; in such cases, all cells connected by bridges adopt the same fate (e.g. in *Hydra*). Similarly, in maize, the homeobox transcription factor Knotted1 (a determinant of meristem identity) as well as its mRNA are transferred between adjacent cells through plasmodesmata[156,157]. In such cases, bridges might coordinate differentiation rather than prevent it. More work will shed light on the contexts and mechanisms by which bridges might perform these two contrasting functions.

Nursing and transport of nutrients

Bridges can allow transport of nutrients from a nursing cell towards another cell such as a developing gamete (Figure 3, left panel). For example, bridges mediate transfer of nutrients from the nurse cells to the oocytes in flies[10], annelids[37], cnidarians[40], tardigrades[19] and sponges[44]. Bridges also likely allow somatic cells to nurse germ cells in *Volvox*[158]. In plants, flow of sap through the plasmodesmata connecting specialized vascular cells (xylem and phloem) is fundamental to the transport of water, minerals and nutrients across the organism[159]. Similarly, septal pores play a crucial role in transport of nutrients and signals through fungal hyphae[160].

Cell cycle synchronization

It has been proposed in many biological contexts, in particular in male germ cysts, that connections between cells synchronize their cell cycles[10] (Figure 3, left panel). In the *Drosophila* ovary, cytoplasmic bridges allow the oriented transfer of cell cycle regulators that control the synchronous endoreplication within groups of nurse cells surrounding the oocyte and allow them to coordinately grow in size and thus serve as a reservoir for the oocyte[161]. Mutations that disrupt fusome function result in asynchronous cell divisions, suggesting a function of the fusome in the intercellular transfer of cell cycle regulators[162,163].

Controlled and selective transport through bridges

Compared to multinucleation with full cytoplasmic continuity, bridges afford less complete intercellular communication, but allow regulated transport (Figure 3, left panel). Indeed, bridge permeability is often selective, though variable between species and stages. The least selective bridges are probably the ring canals of the germline which allow transfer of mRNA, proteins, and organelles[151,152]. In the mammalian testis, transport of granules and macromolecules has been observed[152,164]. At the opposite extreme, hyphal pores and land plant plasmodesmata can be entirely plugged (for example after wound healing[96] or in some cell cycle phases[143] in fungi, or in response to chemical stress in land plants[165]). In early animal embryos and stem cells, bridges allow intercellular transport of overexpressed cytoplasmic fluorescent proteins[63,65]. This transport might be microtubule-based, as in the zebrafish embryo where it appears faster than simple diffusion[65] and in the mouse embryo where bridge microtubules allow transport of E-Cadherin vesicles[62].

Altogether, cytoplasmic bridges function by allowing complete or limited continuity between two or more sister cells. More research will be needed to understand the structural and molecular bases for the regulation of permeability in each model system.

The molecular basis for the evolution of cytoplasmic bridges

Molecular mechanisms of incomplete abscission

Most molecular knowledge on the control of abscission comes from studies of mammalian cells. In those cells, cytokinesis proceeds in three steps[3] (Figure 4A): firstly, the formation of a core of signaling proteins at the center of the spindle during primary constriction (pink in Figure 4A); secondly, secondary constriction and formation of the midbody (blue in Figure 4A); and thirdly, the recruitment of the ESCRT complex and abscission (green in Figure 4A).

In animals, bridges can be stabilized over short periods of time (hours), for example when DNA is lagging at the future site of abscission in cultured cells (NoCut checkpoint), or for longer periods (days), as in germline cysts. The molecular mechanisms for long-term maintenance of bridges within germ cysts are incompletely understood, but seems to involve dephosphorylation of Myosin II in *Drosophila*, which halts the constriction of the acto-myosin ring[24]. In mice testes, incomplete abscission requires instead the testis-specific protein TEX14, which sequesters the ESCRT regulator CEP55. This prevents CEP55 from interacting with its partners ALIX and TSG101, and ultimately from recruiting ESCRT at the abscission site[13].

Shorter-term (hours) stabilization of bridges often relies on the activity of the kinase Aurora B, which notably acts as part of the NoCut checkpoint[166,167]. The NoCut pathway involves the phosphorylation and stabilization of the midbody protein MKLP1 by Aurora B[167]. Aurora B also delays abscission by phosphorylation of the ESCRT-III component CHMP4C[168,169], which retains Vps4 and ALIX away from the final site of abscission[170] in 'abscission checkpoint bodies'[171].

Besides its role in the NoCut checkpoint, Aurora B is also involved in limiting the speed of abscission in some constitutively slow-dividing cells. For example, in *Drosophila* female germline stem cells, Aurora B localizes at the fusome and inhibits Cyclin B by phosphorylating it, thus delaying mitotic exit and abscission[172]. In male germline stem cells where bridges have a shorter lifetime, bridge maintenance seems to rely on a secondary

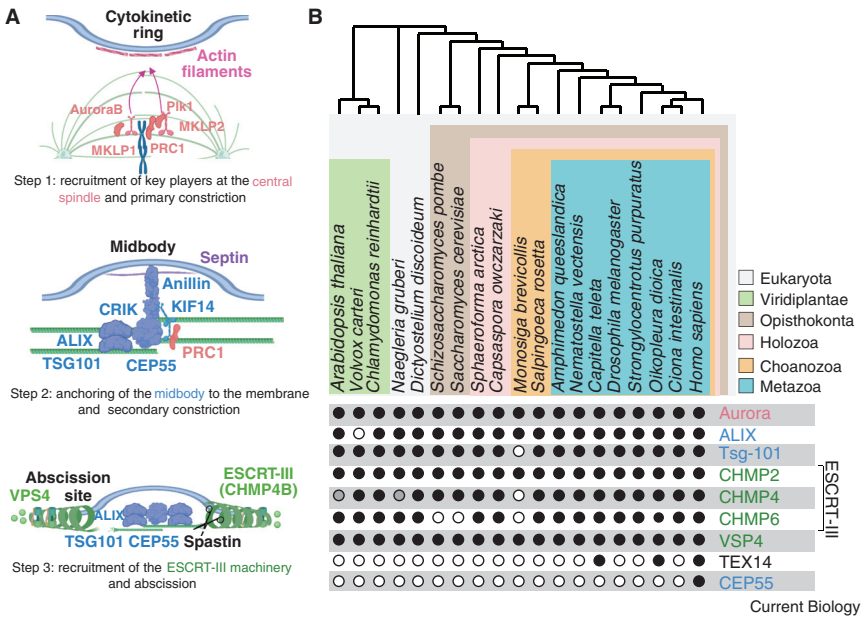

Figure 4. Evolution of the molecular mechanisms of abscission.
(A) Simplified sequence of events leading to abscission in HeLa cells. (B) Phylogenetic distribution of proteins involved in abscission discussed in the text (see Methods in Supplemental information). Dark circles: homolog present; white circles: homolog not detected; grey circles: putative homolog. We validated the TEX14 homolog of the annelid *C. teleta* by documenting broader conservation across protostomes and by detecting a degenerated homolog in the tunicate *O. dioica* (Figure S1).

acto-myosin ring blocking secondary constriction and on a delay in recruitment of a component of ESCRT, Shrub/ESCRT-III, at the abscission site[173]. This delay might be due to inhibition of ESCRT recruitment by Aurora B and/or to a lower level of ESCRT proteins (or of their recruiters) within the cell (such as ALIX)[174,175]. A similar mechanism might be at play in mouse embryonic stem cells. In zebrafish embryos, the key ESCRT component CHMP4B is not expressed until the mid-blastula transition when cytoplasmic bridges disappear[66], suggesting that transcriptional regulation of the ESCRT complex might also control abscission.

Finally, the stable cytoplasmic bridges of the animal germline contain a diversity of structures that have been hypothesized to contribute to their maintenance and stability[6,59,176], including (depending on sex and species) F-actin[177], septins[178], anilin[179], filamin[180], and myosin-II[179].

Little is known about the mechanism for bridge maintenance outside of animal cells. In plants and algae, primary plasmodesmata are formed by the trapping of the endoplasmic reticulum coming from Golgi-derived vesicles in the reforming cell wall at the end of cell division[106,181], but molecular details explaining how this is controlled are unknown.

Phylogenetic distribution of the molecular regulators of incomplete abscission

Many regulators of abscission are conserved across eukaryotes: ALIX, Aurora, ESCRT-III and Vsp4 all date back to the last eukaryotic common ancestor[182] (Figure 4B) and ESCRT-III even has homologs in Archaea and Bacteria (though their function remains unclear[183]). Around this 'core' of ancient proteins, other regulators of mammalian cell abscission evolved later, such as the midbody component MKLP1, which is specific to animals and their close relatives[182].

Intriguingly, the most recently evolved molecules are those involved in bridge formation in the germline: TEX14 only exists in bilaterians with a mosaic distribution suggesting frequent loss, and CEP55 is vertebrate-specific (Figure 4B). This raises an apparent paradox: since germline bridges are universal (and likely ancestral) in metazoans, why do some of their molecular regulators undergo a fast evolutionary turnover? Rapid molecular evolution within a conserved biological context is common in evolutionary 'arms races', such as between hosts and parasites, or between meiosis distortion segregators and the rest of the genome (for example, 'selfish' centrosomal sequences increasing their own transmission to the oocyte[184,185]). As discussed above, germline bridges might play a role in preventing the emergence of cheater gamete genotypes by pooling gene expression. The rapid evolutionary turnover of genes involved in bridge maintenance might thus be a signature of the need for constant adaptation to newly evolving cheater alleles. One might then expect these genes to not only be taxonomically restricted, but also to evolve quickly at the sequence level. It will be interesting to determine whether TEX14, CEP55, and other germline bridge molecules show fast rates of molecular evolution. Beyond that, direct testing of the arms race hypothesis will require crossings or heterologous knock-in studies.

Unique or multiple origins of cytoplasmic bridges?

The broad distribution of cytoplasmic bridges in multicellular eukaryotes — notably in forms with simple multicellularity — suggests that the suppression of abscission could be a frequent basis for the initial evolution of clonal multicellularity. Since multicellularity is classically considered to have evolved many times independently[8,186], cytoplasmic bridges might have multiple origins. Because the core molecular machinery of abscission was already present in the Last Eukaryotic Common Ancestor (LECA), including frequent regulators of incomplete abscission such as Aurora B (Figure 4B), ancient eukaryotes could have had the ability to easily evolve incomplete abscission by interrupting its progression, possibly by co-option of pre-existing regulatory checkpoints such as the NoCut pathway.

As a more speculative alternative, LECA might already have possessed cytoplasmic bridges, and thus simple multicellularity. Although this seems contradicted by the prevalence of single-celled forms across eukaryotes, multicellularity might exist undetected in some lineages, or be easy to lose. The study of the molecular mechanism for bridge maintenance in additional species will shed more light on the homology of cytoplasmic bridges.

Conclusion

While the mechanisms of cell division have been extensively studied in cultured cells, the fast abscission observed in that context is not universal. Indeed, many multicellular species present developmental stages where cells remain connected by cytoplasmic bridges after cell division. The recurrence of bridges in multicellular species suggests that they could be a frequent basis for the evolution of multicellularity, allowing mechanical support, sharing of cellular content and suppression of cheater genotypes. In animals, while bridges are ubiquitous in the germline (suggesting presence in the last common metazoan ancestor), the molecular mechanisms allowing bridge maintenance seem to vary, suggesting fast evolution that could reflect adaptation to constantly evolving cheater alleles. Comparative studies of the molecular basis for fast or slow abscission as well as functional perturbations of the bridges will shed light on the role of abscission regulation during development and homeostasis.

SUPPLEMENTAL INFORMATION

Supplemental information can be found online at https://doi.org/10.1016/j.cub.2022.03.021.

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DECLARATION OF INTERESTS

The authors declare no competing interests.

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