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Trypanosoma Brucei's KLIF-Associated Cytoskeletal Proteins Control Cytokinesis by Encouraging the Placement of Cleavage Furrows and Ingression

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Abstract

In the early divergent protozoan Trypanosoma brucei, cytokinesis happens from the anterior cell tip of the new-flagellum daughter of a dividing biflagellated cell toward the developing posterior end of the old-flagellum daughter. Along the preformed cell division fold, the cleavage furrow ingresses unidirectionally. It is controlled by an orphan kinesin known as kinesin localized to the ingressing furrow (KLIF), which localizes to the leading edge of the furrow. It is unclear how KLIF regulates furrow ingression and whether it interacts or collaborates with other cytokinesis regulatory proteins to encourage furrow ingression. In this study, we examined KLIF's functions in cleavage furrow ingression and discovered a group of cytoskeletal proteins linked to KLIF that are crucial regulators of cytokinesis.

Keywords: Cytoskeletal proteins; Cytokinesis; KLIF

Introduction

Through genetic complementation, we were able to show that furrow ingression is facilitated by the kinesin motor activity of KLIF rather than the proposed tropomyosin domain. Additionally, we demonstrated that KLIF depletion hindered the old-flagellar daughter cell's incipient posterior's resolution, which in turn delayed the ingression of the cleavage furrow at the end of cytokinesis. A subset of cytoskeleton-associated proteins (CAPs) was identified as KLIF-proximal proteins through proximity biotinylation. Functional characterization of these cytoskeletal proteins demonstrated the critical roles of CAP42 and CAP50 in promoting cleavage furrow ingression, as well as CAP46 and CAP52 in positioning the cleavage furrow. Collectively, these findings revealed the critical and unique roles that several cytoskeletal proteins play in regulating cytokinesis [1,2].

Methodology

In sub-Saharan Africa, Trypanosoma brucei is a flagellated, unicellular parasitic protozoan that causes nagana in cattle and sleeping sickness in humans. The parasite multiplies through binary fission in the midgut of the insect vector and the bloodstream of mammals, alternating between the insect vector, the tsetse fly, and the mammalian hosts. During the G1 phase of the cell cycle, a single flagellum is present in a trypanosome cell. It originates from the basal body, passes through the flagellar pocket, and subsequently attaches itself to the cell body via the flagellum attachment zone. The trypanosome cell builds a new basal body in the early stages of the cell cycle, and from this newly formed basal body, it constructs a new flagellum [3-6].

The newly formed flagellum leaves the flagellar pocket and extends toward the cell anterior after the cell cycle progresses. In the meantime, a new FAZ that is associated with the newly assembled flagellum forms. It extends from the anterior tip of the new-flagellum daughter (NFD) cell toward the flagellar pocket area in a direction opposite to that of the new flagellum. A biflagellated mother cell is divided into two uniflagellated daughter cells by cytokinesis, which starts at the anterior tip of the NFD cell and proceeds along a preformed cell division fold between the old and new flagella toward the developing posterior of the old-flagellum daughter (OFD) cell [7-9].

T. brucei has an unusual mode of cytokinesis that is actomyosin-independent. The regulatory pathway that governs cytokinesis consists of trypanosome-specific and evolutionarily conserved regulatory proteins that function at the cleavage furrow and the anterior tip of the NFD cell (or the new FAZ tip) to promote the initiation, progression, and completion of cytokinesis. Two protein kinases, the Polo-like kinase TbPLK and the Aurora B kinase TbAUK1, as well as the microtubule-severing enzyme katanin60-katanin80 complex are among the evolutionarily conserved regulators. There are several novel protein phosphatases that are specific to trypanosomes. These include KPP1 (kinetoplastid-specific protein phosphatase 1), KLIF (kinesin localized to the ingressing furrow), a putative protein that contains an EF-hand motif.

CIF2, also known as cytokinesis initiation factor 2, and a group of proteins called CIF1, CIF3, CIF4, FPRC (FAZ tip-localized protein required for cytokinesis), and FRW1 (Furrow 1) that contain α-helical motif. With the exception of CIF4 and FPRC, CIF1 interacts with all known cytokinesis regulators and recruits most of its partner proteins to the anterior tip of the NFD cell, suggesting that it orchestrates cytokinesis. TbPLK, KPP1, CIF2, and CIF3, among other CIF1-interacting proteins, maintain CIF1 localization at the anterior tip of the NFD cell or CIF1 protein stability, which allows them to regulate CIF1 through feedback. Seem to recruit CIF1 to the anterior tip of the NFD cell, acting upstream of CIF1. More research is necessary to fully understand the mechanistic roles of these cytokinesis regulators and their entire sequence of actions [10].

Results

A polarized array of subpellicular microtubules, which is replicated semiconservatively during the cell division cycle, defines the

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cytoskeleton of T. brucei cells. The posterior end of the cell is where the plus ends of the subpellicular microtubules are located. As the cell cycle enters its S-phase, these microtubules begin to elongate from their plus ends toward the posterior of the cell, while the newly assembled flagellum extends in the opposite direction, toward the anterior of the cell. Membrane invagination occurs from the anterior end of the NFD cell toward the nascent posterior of the OFD cell, forming a nascent posterior for the OFD cell at late cell cycle stages, at the ventral side of the cell near the posterior portion of the NFD cell.

Discussion

Creating a cleavage furrow those ingresses unidirectional from the anterior of the NFD cell toward the developing posterior of the OFD cell, a process known as the "cell division fold." During the last stage of cytokinesis, the posterior tip of the OFD cell and the posterior part of the NFD cell are joined by a thin cytoplasmic thread known as the cytoplasmic bridge. The cytoplasmic bridge is then broken, separating the two daughter cells. Since the knockdown of several CAPs in T. brucei resulted in cytokinesis defects it is likely that cytoskeleton-associated proteins (CAPs) are involved in cytokinesis. However, it is unclear whether CAPs are directly involved in cytokinesis and, if they are, how they promote cytokinesis.

Conclusion

This report details our investigation into the function of the orphan kinesin KLIF in cleavage furrow ingression, the identification of KLIF-associated cytoskeletal proteins through proximity biotinylation (BioID), and the description of these proteins' function in regulating cytokinesis in T. brucei's procyclic (insect) form. The findings showed that KLIF is necessary for the development of the OFD cell's nascent posterior in order to facilitate cleavage furrow ingression and cytokinesis completion. They also identified specific functions for a number of cytoskeletal proteins associated with KLIF in controlling cleavage furrow ingression and cleavage furrow positioning. These results demonstrate the critical role that CAPs play in regulating the orientation and cleavage furrow ingression necessary for faithful cytokinesis in T. brucei.

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