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Insights into Septins in Fungal Pathogenesis

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INTRODUCTION

Initially discovered in Saccharomyces cerevisiae, septins are a family of filament-forming GTP-binding proteins that are present in numerous eukaryotic cells and performing important roles in life. Septins were given their name because research has shown that they can create the ring of filaments at the mother-bud neck, which is where the septum, or cell wall, separating the mother and daughter cell, is located. Septins are changeable at both the N- and C-termini and are GTP-binding proteins with a P-loop. A highly conserved central core found in many species of septins is made up of a polybasic domain at the N-terminus that has been demonstrated to bind to phosphoinositide on the plasma membrane. The GTP binding domain is located downstream, and it terminates with a distinct septin element that sets septins apart.

Recent discoveries have shown that septins (Ford and Pringle, 2021) were built to function as complexes rather than single protein entities and form complex septin ultrastructures, such as filaments, rings and gauzes. How do these different structures form and what roles do these different structures play? In S. cerevisiae, the molecular structure of the septin complex was solved by using single-particle electron microscopy and proposed that Cdc10 (Fig 1) occupies the central position of the core complex and Cdc11 lies at its extremities, leading to the following arrangement for the octamer: Cdc11-Cdc12-Cdc3-Cdc10-Cdc10-Cdc3-Cdc12-Cdc11. The linear rod lacks polarity. When the salt ion concentration is low, the N-C interface of Cdc11 in the rod is connected to form filaments. In addition, the predicted C-terminal coiled-coil domains of Cdc3 and Cdc12 are required to assemble into filaments. In S. cerevisiae, septins comprise another hetero-octamer : Shs1-Cdc12-Cdc3-Cdc10-Cdc10-Cdc3-Cdc12-Shs1.

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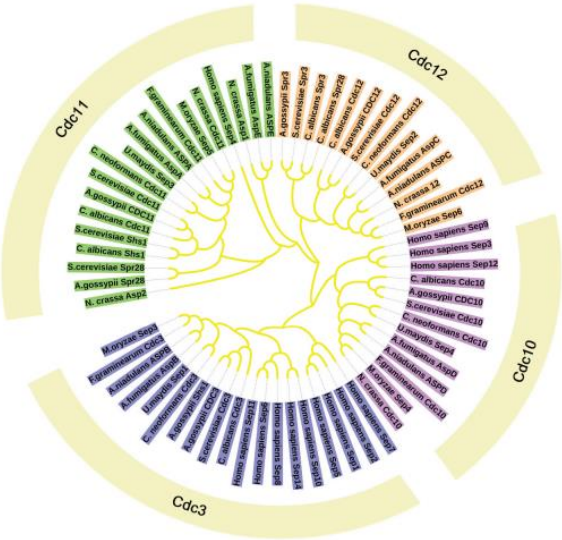

Fig 1- Phylogeny and evolutionary history of septins. Maximum likelihood phylogenetic tree of 65 septin proteins from 11 fully sequenced species that are reported in eukaryotic lineages. The categorization of different septins by multiple copies of coiled coils found in a single protein: the Cdc11 group with two coiled-coil domains, the Cdc3 and Cdc12 groups with one coiled-coil domain and the Cdc10 group with no coiled-coil domain.

Structure of Septins

When the cell wall is eliminated by enzymatic digestion, the filament structures transform into ring- and gauze-like structures (Figure 2). These findings imply that septins do not operate as individual proteins but rather as complexes. The ring formations were visible at the mother-bud neck in yeast and the appressorium-penetration peg neck linked to virulence and the curved membrane in rice blast fungus (Oses-Ruiz *et al.*, 2017).

According to recent studies, septin proteins bind phosphatidylinositol phosphates (pips) at the appressorium pore membrane, causing the pip to assemble into a ring and facilitating the creation of a penetration peg that is necessary for *M. oryzae* to infect its host. Septins can create structures called gauzes, which are made up of a meshwork of septin filaments in yeast, in addition to filaments and rings (Hall and Russel, 2004).

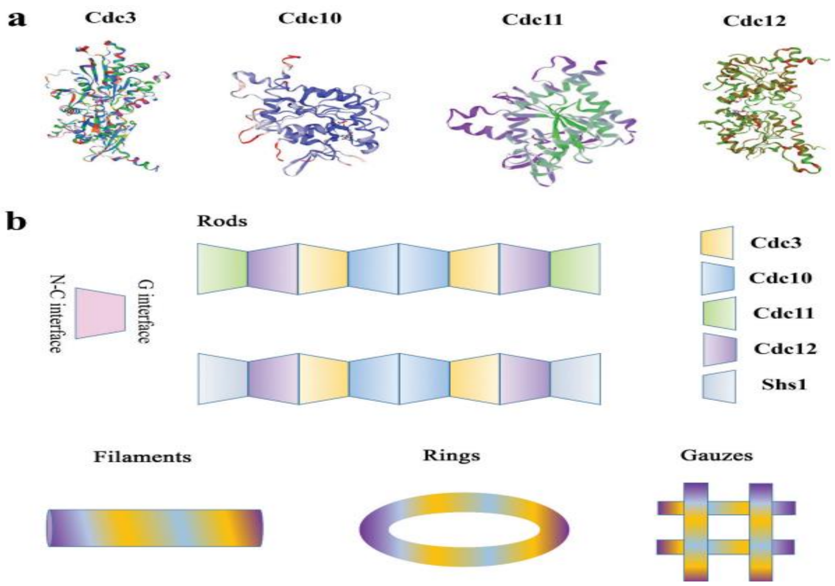

Figure 2 – Structure of the septins

Functions of septins in host infection by pathogens

In order to maintain the localization of the Wiskott-Aldrich syndrome protein (WASP) and the inverse-binamphiphysin-RVS-domain (I-BAR) protein Rvs167, the septin ring also serves as a diffusion barrier Las17. The WASP is engaged in the polymerization of F-actin, while the BAR protein is involved in membrane curvature at the penetration peg's tip. Furthermore, due to their incapacity to pierce leaves, septin mutants in *M. oryzae* exhibited a significantly decreased pathogenicity, indicating that septins were required for the development of rice blast disease. Recent research has demonstrated that the fungal mitogen mediates *M. oryzae's* intercellular infection. MoPmk1 activated protein kinase (MAPK).

Roles of septins in autophagy

Autophagy is an intracellular waste treatment and recycling mechanism that has been retained throughout evolution. It entails the production of double-membrane vesicles termed autophagosomes, which then engulf the intracellular material that is meant to be recycled in order to degrade. Numerous cues can initiate autophagy; the most well researched forms of autophagy are those resulting from inadequate nutrition or stimulation of certain breakdown targets (Kershaw *et al*., 2019).

Septins, which are cytoskeleton proteins, serve as a "scaffold" to entice autophagy-related proteins to participate in the structure of autophagy. Furthermore, septins may regulate autophagy in order to be involved in it.

The autophagosome membrane is shaped by the actin-related protein 2/3 (Arp2/3) complex, which also helps move autophagosomes once they separate. Autophagy and microtubule dynamics are connected as well.A portion of unstable microtubules appear to be necessary for the development of autophagosomes caused by starvation, whereas stable microtubules may be necessary for mature autophagosome motility prior to lysosome fusion.

On the other hand, little is known about septins' function in the autophagy process. Early studies on the connection between septins and autophagy were conducted on bacterial pathogens. Research has revealed that certain bacterial infections possess defense mechanisms against autophagy or employ it to enhance intracellular survival.

Septins involved in exocytosis and endocytosis pathways

- Cells transport materials into and out of their bodies through processes called endocytosis and exocytosis that are too big to fit through the lipid bilayer of the cell membrane.
- The process by which cells take up foreign substances through vesicles is known as endocytosis. The mechanism by which cells move materials from within to the exterior of the cell is called exocytosis (Warenda *et al*., 2023).
- Septins interact with the various parts of the exocytic machinery. Through their interactions with the elements of the exocyst complex in mammalian cells, septins can control the transit of vesicles to the fusion site of the plasma membrane.
- Membrane fusion and synaptic vesicle transit depend on a few of the proteins that interact with septins.
- Ion channels, their clustering, and the release of synaptic vesicles are all under the regulation of septins.

CONCLUSION

Septins seem to be essential for fungi to control their surface topologies, which allows them to invade a variety of substrates, including, crucially in this case, living host tissue. Because they can stiffen the cortex to stop abnormal polar expansion and assemble precisely where branching and re-polarization are needed, septins may play such a role.

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However, it's still unknown how the distribution of septins is determined.

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