cycle starts again as Bud2/5 and Rsr1 recognise the markings of Bud3/4 and Axl1/2 at the bud scar site, causing them to localise at the new bud site (figure 2).

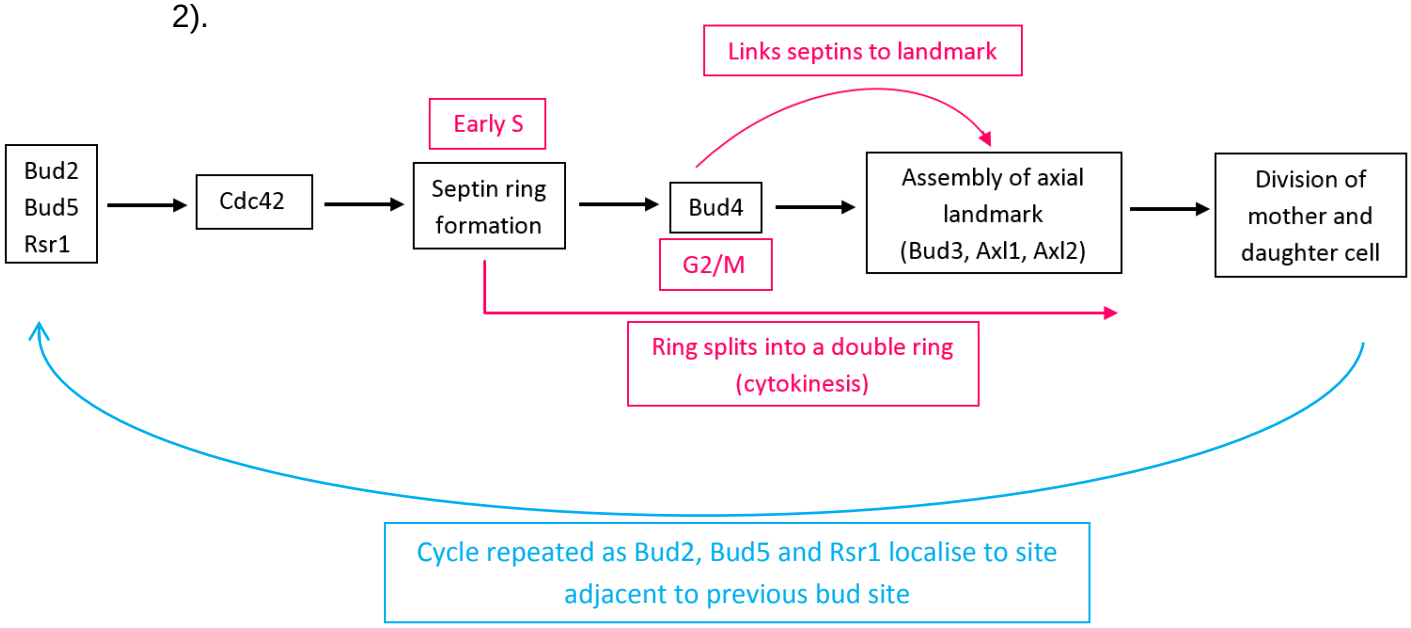


***Figure 2 –*** Schematic summarising the pathway leading to the formation of the axial landmark in haploid *S. cerevisiae* yeasts. The previous cell cycle sets up the location of the new bud-site, therefore the direction of polarisation of the cytoskeleton in late G1 is already determined in the previous cycle.



Actin is also essential during cell division (figure 1). An actin contractile ring forms from actin cables at the bud neck during cytokinesis, via the support of Cdc42 and formins e.g. Bni1 and Bnr1[10]. Formins are large actin binding polypeptides which assist in the assembly of the actin ring from long actin filaments. This ring is critical during cytokinesis in directing new wall assembly to the division site and providing the mechanical force needed to separate the mother and daughter cell[7].

**Bipolar budding pattern**

Cytoskeletal polarisation is also necessary for the growth of *S. cerevisiae* diploid cells; however directionality differs due to which bud-site selection genes are activated. The axial budding protein, Axl1, is repressed in bipolar budding allowing Rax1, which accumulates at the bud site, to set-up the bipolar landmark[11] (figure 3). In turn this landmark recruits Bud8 and Bud9 to accumulate within specific locations. The Rax1 signal remains persistent throughout the cell cycle, maintaining bipolar budding[2].

A unique feature of hyphal formation is the presence of polarised lipid rafts enriched in sterols[16] (table 2). Not only do these rafts accumulate in the hyphal tip, but they co-localise with septins aiding in septum formation. In hyphae, septation occurs away from the mother-bud neck and disassembly of septins does not occur, as phosphorylated Efg1 binds to and inhibits Ace2, preventing the transcription of the septin degrading enzyme[6]. This results in the mother and daughter cells remaining firmly attached with hyphal walls that are parallel with no constrictions.

**Growth kinetics**

Growth kinetics between the three phenotypes differ for a number of reasons, including the regulation of growth mechanisms within the cell cycle. It has been found that ~70% of a yeast cells growth is due to apical expansion and ~30% general expansion, compared to >90% of filamentous growth being apical and <10% being general[17]. As shown in figure 6 there is a clear distinction between rates of surface expansion at the apical portion compared to the proximal portion near the mother-bud neck, in both budding yeast and hyphae.



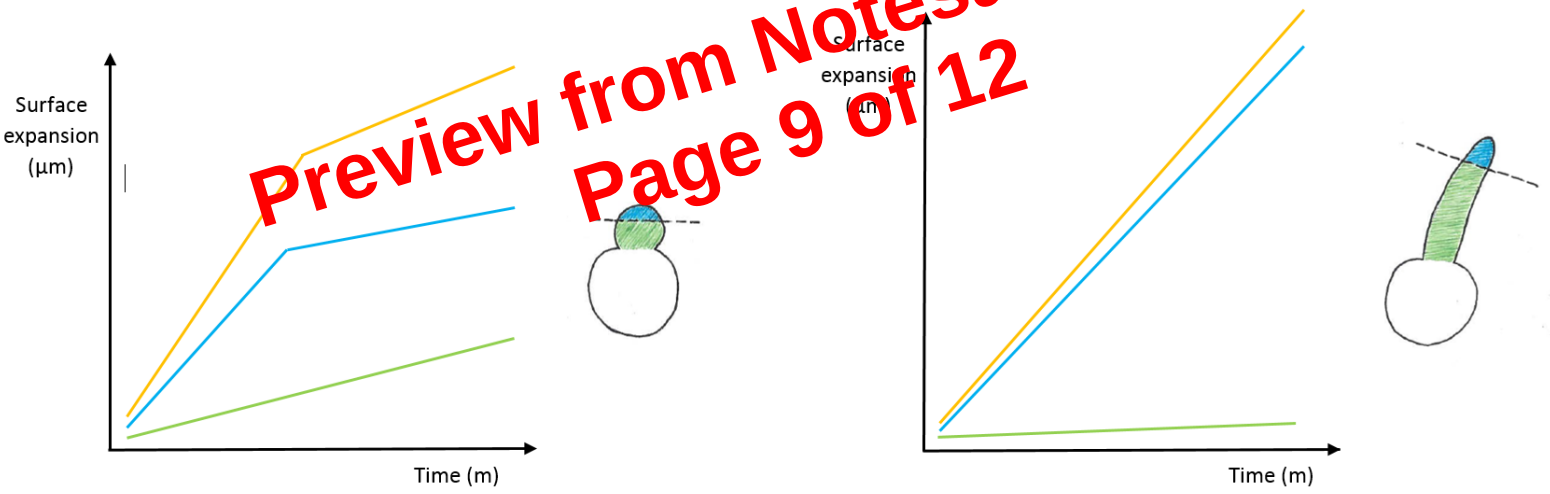


***Figure 6 –*** Line graphs showing the surface expansion of a budding yeast and hyphae. The proximal portion of the yeast bud (green) grew at a low but constant rate, whereas the equivalent portion in the hyphae grew at a negligible rate. The apical expansion zone at the cell tip (blue) grew at a fast rate in yeasts followed by a sudden reduced rate of growth. At the hyphal tip the rate of growth is comparable to that of the budding yeast; however hyphae do not undergo the transition to a lower growth rate. Graphs show the overall surface expansion of a budding yeast and hyphae (yellow) (adapted from[17,19]).