

Polar cell growth

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VI. POLAR CELL GROWTH

A.1. MONOPOLAR OUTGROWTH (EMERGENCE)

In our present state of knowledge, cytoplasmic *microtubules* are dispensable for bud outgrowth (see I) but required for specific, single or double budding of yeast cells or fungal spores to direct their mono- or dipolar axiation toward the site(s) of bud formation. By contrast, polarly localized actin *microfilaments* appear to be an absolute requirement for the budding processes.

a² *Yeast budding*

The cortical actin cytoskeleton seems to specify sites of growth of the yeast cell surface (Adams and Pringle, 1984, see I; Novick and Botstein, 1985). An actin-binding protein (ABP1p) might be involved in the spatial organization of cell surface growth and the identification of C-terminal protein domains suggests that such domains might serve to bring together signal transduction proteins and their targets or regulators, or both, in the membrane cytoskeleton (Drubin *et al.*, 1990).

The cytoskeletal network in the budding yeast cell (*Saccharomyces cerevisiae*) behaves as a parallelly distributed processor, as suggested by the finding of a protein (SPA2) associated with actively growing regions of the cell surface (Snyder, 1989). Such polarization of the growth process is disturbed in mutant cells displaying an inability to stop growing under nutrient-limiting conditions which often results in multiple budding (multipolar growth, see I, p. 187).

b¹ *Fungal spores*

In the germinating spores of *Mucor rouxii* the change in growth pattern from spherical to polarized correlates with the degree of DNA methylation and this, in turn, may be controlled by polyamine levels. The establishment of the polarized phase of growth in *M. rouxii* probably occurs through the regulation of the genes involved in the synthesis of products necessary for apical growth of the hyphae (Cano *et al.*, 1988).

c) *Dimorphism*

Quite recently, Crombie *et al.* (1990) have shown that the sites of budding and germ tube formation on yeast cells of *Candida albicans* were polarized preferentially towards the cathode. Buds were found to be less polarized than germ tubes at any given applied voltage. Moreover, polarization of germ tubes was biphasic.

2. TIP GROWTH

b) *Fungal hyphae*

In the models of hyphal tip growth, electric current does not always enter the growing end (*Allomyces* hypha drives an outward protonic current, see Youatt *et al.*, 1988 in I). As recently commented by Gow (1989) “Most of the evidence suggesting that ionic currents are involved in establishing and maintaining polar growth is essentially correlative, and it is not yet clear whether the current is a cause or consequence of polarity”. However, Gow leaves open the possibility that “Cytoplasmic proton and calcium-ion gradients and fixed-charged gradients resulting from asymmetric transport of calcium into a cell may be involved in localizing growth”. The same conclusions have recently been reached about differentiation at egg germinations of brown and red algae (Quatrano and Kropf, 1989; Waaland, 1989; see VII.C.3.a).

In hyphal tips of the oomycete *Saprolegnia ferax*, Heath and Kaminskyj (1989) observed that “all the organelles and the microtubules are non uniformly distributed, each showing a characteristic longitudinal gradient starting at a different point behind the tip”. A few microtubules can reach the extreme tip but they were more abundant sub-apically. The authors concluded that “the correlated patterns of organelle and cytoskeleton organization from this and previous work show that neither the microtubules nor the detected arrays of actin are sufficient to account for most organelle arrangements”.

The role of microtubules at the onset and maintenance of polarized growth of hyphae is still unclear. Intact microtubular tracks are required to initiate dominant, monopolar outgrowth from macroconidia of *Neurospora crassa* (Caesar *et al.*, 1988, see in I). However, further elongation of hyphae deprived of microtubules can still occur contortionally, with a damped polarity (Howard and Aist, 1980, see I).

Germlings of the bean rust fungus *Uromyces appendiculatus* treated with the microtubule-binding drug griseofulvin continued polarized apical growth even though showing changes in the morphology of their apical and subapical regions (Hoch *et al.*, 1987).

i) *Animal neurites*

A major question in developmental neurobiology is how developing nerve cells accurately extend processes to establish connections with their target cells (see Lasek and Black, 1988). This unsolved problem of polarized growth involves “both the nature of cues for growth cone guidance and also the question of how growth cones survey their environment for cues and respond by altering their direction of migration” (Bentley and Toroian-Raymond, 1986, see I). According to Lamoureux *et al.* (1989) “there is also controversy over whether axonal elongation is the result of a pulling growth cone and the role of tension in axonal elongation”.

Earlier in this decade, the consensus was that axons or neurites elongated from tension generated by forward motility of the growth cone (Landis, 1983; Letourneau, 1982). It was presumed that contractile filopodia were the source of the tension moving the growth cone (Bray, 1982; Trinkaus, 1985). But this view was challenged by experiments showing that neurites elongate, albeit abnormally, in the presence of cytochalasin, which inhibits growth cone and filopodial movements (Marsh and Letourneau, 1984).

Bentley and Toroian-Raymond (1986) also reported an examination of the migration of pioneer growth cones deprived of filopodia by culture in agents which disrupt actin microfilaments. Under these conditions, axons continue to extend but a large percentage of growth cones are highly disoriented. Their results indicate that filopodia are not necessary for axonal elongation *in vivo* but that they are important for correctly oriented growth cone steering.

Additionally, high resolution, video-enhanced observations of growth cone activity argue against filopodial shortening as a source of tension, suggesting instead that an extrusion of cytoplasm rather than a pulling process, is the key event in neurite elongation (Goldberg and Burmeister, 1986; Bray, 1986; Aletta and Greene, 1988, ref. in Lamoureux *et al.*, 1989). Studies of slow axonal transport (Lasek, 1986) indicate that much slower cytoskeletal pushing underlies axonal elongation and direct measurements of neurite force as a function of growth cone advance show that they are linearly related and accompanied by apparent neurite growth (Lamoureux *et al.* (1989). No increase in force occurs in neurites whose growth cone fails to advance.

According to Mitchison and Kirschner (1988) there are three phases of axonal development: an actin based-system in which the leading edge becomes orientated, a consolidation phase in which filopodial microtubules become stabilized in their direction of future growth and a conversion phase to stable microtubules bundled within the axonal tube. The protein factor tau stimulates the conversion phase. However, tau expression is insufficient to induce polarity but tau antisense oligonucleotides can inhibit neurite polarity (Kosik and Finch, 1987).

Pulse-labelling studies performed both in mature nerve and in cell culture provided most of our knowledge of the axonal transport of cytoskeletal proteins. In 1975, Ochs has put forward his unitary hypothesis of axonal transport according to which proteins achieve different transport rates by having different affinities for a single moving vector. Tubulin and actin molecules are the essential components of the axonal cytoskeleton and considered by some (Black and Lasek, 1980) as a static complex travelling down the axon, a view challenged by others (ref. in Okabe and Hirokawa, 1990) who observed a gradual recovery of photobleached zones rather than their movement or spreading along the axon, both in neurons injected with fluorescein-labelled tubulin and actin. Therefore, these cytoskeletal components can be considered as "dynamic structures that continue to assemble along the length of the axon" (Okabe and Hirokawa, 1990).

In most recent and interesting experiments, Schnell and Schwab (1990) have shown that axonal regeneration and elongation in the rat spinal cord can be produced by the neutralization by monoclonal antibodies of myelin-associated neurite growth inhibitors.