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V. POLAR CELL MOVEMENTS

B.1. *Cilia-flagella*

In the green unicellular alga *Chlamydomonas,* a component of contractile flagella roots is the centrosome-associated phosphoprotein centrin. This type of structural organization contributes to define its cell polarity through cell axiation (Fig. 1, in Salisbury, 1989).

2. *Gliding movements*

Bacterial gliding motility appears to be dependent on the establishment of transmembrane potential and any depolarization (*not* depolymerization as wrongly written in **I** p. 132) by protonophores such as 2,4-DNP or CCCP results in a cessation of motility.

3. *Amoeboid motion (transient polarity)*

Both the single headed myosin I and the double headed myosin II are mechanochemical enzymes which generate force through the hydrolysis of ATP when complexed with F-actin.

Fukui *et al.* (1989) show by immunofluorescence microscopy that non-filamentous myosin-I occurs at the leading edges of the lamellipodial projections of migrating *Dictyostelium* amoebae, which are devoid of myosin II, whereas filamentous myosin II is concentrated in the posterior zone of the cells. The authors suggested on the basis of these locations of the two forms of myosin and their known biochemical and biophysical properties that "actomyosin I may contribute to the forces that cause extension at the leading edge of a motile cell, while the contraction of actomyosin II at the rear squeezes the cell mass forward. Myosin I isoenzymes might have similar roles in metazoan cells, for example at the leading edges of neuronal growth cones, and in the extension of lamellipodia and pseudopodia of leukocytes, macrophages and fibroblasts." These observations suggest that "actomyosin I-dependent force-generating activity occurs at the leading edge (as in pseudopodia extension) and that actomyosin II-dependent force-generating activity occurs at the trailing end of a migrating *Dictyostelium* amoeba (causing the cell mass to move forwards)". This could explain "how myosin II-minus mutants can form smaller-than-normal pseudopodia at a relatively normal rate. Membrane-bound *Acanthamoeba* myosin I can generate force against actin cables however, and both *Acanthamoeba* and *Dictyostelium* myosin I will crosslink actin filaments and generate force between crosslinked filaments".

None of Fukui *et al.* (1989) observations is compatible with the participation either of other processes in amoeboid movement, such as membrane flow or the remodelling of the actin matrix, or of myosin I and myosin II in other motile activities. The significance of Fukui's team results is that they show the presence in the leading edge of a migrating cell of myosin I, which in conjunction with F-actin is known to be capable of producing force and movement.

To explain the rearward movements of membrane proteins in locomoting polymorphonuclear leukocytes, the experimentally best supported model implies the cytoskeleton (see **I**, pp. 133-137). The retrograde lipid flow hypothesis has been proposed by Bretscher (1984) as an alternative explanation for the rearward movements of membrane proteins. However, recently used techniques of low-light-level fluorescence microscopy and digital image-processing of photobleached images disprove that lipid flow model (Lee *et al.*, 1990). By further implicating cytoskeleton in proteins movements, they also validate the conclusion of Sheetz *et al.* (1989) that such a membrane flow in the leading edge of amoeboid cells does not drive rearward movements of membrane glycoproteins.

About the motor of amoeboid motion, there is much evidence linking actin-based system to the generation of motile structures in the cell (Bray and Vasiliev, 1989). Nevertheless, a mutant of *Dictyostelium discoideum* deficient in α-actinin and in which movements are unimpaired has been obtained by Gerisch's group (Wallraff *et al.*, 1986; Schleicher *et al.*, 1988). "Motile life without myosin" also exists as shown by mutants of *D. discoideum* that lack normal myosin-II (Knecht and Loomis, 1987; De Lozanne and Spudich, 1987, see **I**, p. 134). Since, André *et al.* (1989) have described a strain of this slime mold lacking severin (actin-filament fragmenting protein) even though still able to move. A relative interpretation of these findings is that "there is an extensive overlapping redundancy in the activity of actin-binding proteins *in vitro* and more than one way to crosslink, fragment or even to move actin filaments" (Bray and Vasiliev, 1989). There is analogy between the behavior of such parallely distributed processor of the locomotive cytoskeleton of *Dictyostelium* amoebae and of the cytoskeletal network intervening at yeast budding (see VI.A.1.a[2]).