Title Actomyosin mediates gravisensing and early transduction events in reoriented cut snapdragon spikes

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Abstract

We investigated the involvement of the actomyosin network in the early events of the gravitropic response of cut snapdragon (*Antirrhinum majus* L.) spikes. The effects of the actin-modulating drug, cytochalasin D (CD) and/or the myosin inhibitor, 2,3-butanedione-2-monoxime (BDM) on amyloplast displacement, lateral auxin transport and consequently on stem bending were examined. The inhibitory effect on cytoskeleton integrity was studied by using indirect immunofluorescence double-labeling of actin and myosin. Our results demonstrate that no organizational changes in actin filaments occurred in cortical and endodermal cells of the stem bending zone during reorientation. These results suggest that actin depolymerization is not required for amyloplast sedimentation. Unlike the chloroplasts in the cortex, the amyloplasts in the endodermis were surrounded by actin and myosin, indicating that amyloplasts may be attached to the actin filaments via the motor protein, myosin. This suggests the involvement of myosin as part of the actomyosin complex in amyloplast movement in vertical as well as in reoriented stems. This suggestion was supported by the findings showing that: (a) BDM or CD disrupted the normal organization of actin either by altering characteristic distribution patterns of myosin-like protein in the cortex (BDM), or by causing actin fragmentation (CD); (b) both compounds inhibited the gravity-induced amyloplast displacement in the endodermis. Additionally, these compounds also inhibited lateral auxin transport across the stem and stem gravitropic bending. Our study suggests that during stem reorientation amyloplasts possibly remain attached to the actin filaments, using myosin as a motor protein. Thus, gravisensing and early transduction events in the gravitropic response of snapdragon spikes, manifested by amyloplast displacement and lateral auxin transport, are mediated by the actomyosin complex.