eXtra Botany

Insight

Myosin XI motors: back on the scene at the division machine

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Plant myosin XI motors are renowned for their function in cytoplasmic streaming. But the cell biology of myosin XI has taken an unexpected turn as a study by Abu-Abied *et al.* (2018) suggests a role of the motor in auxin transport and cell division plane alignment.

Only two types of myosins are present in green algae and land plants, myosin VIII and myosin XI, but in land plants each type is represented by a small gene family (Nebenfuhr and Dixit, 2018). Strictly speaking, there is one more myosin-like sequence present in the genomes of plants, the chimeric motor KCBP, which sports an N-terminal MyTH4-FERM domain with significant BLAST homology to myosin VII. However, the motor domain of KCBP is a 14-type kinesin (Reddy and Day, 2000). While a function of myosin VIII in cell division has been reported before, myosin XI is probably best known for its impact on cytoplasmic streaming (Tominaga *et al.*, 2013). Indeed, some myosin XI motors are the fastest known cytoskeletal motors, and these are responsible for the remarkably vigorous cyclosis observed in *Chara* and related algae (Higashi-Fujime *et al.*, 1995).

Recent research has revealed details of myosin XI-mediated motility in plants (Sparkes *et al.*, 2008; Avisar *et al.*, 2009), and it was found that specific myosin receptors, so-called MyoB proteins, are involved in myosin attachment to certain cargo (Peremyslov *et al.*, 2013). Previous research has also used mutants to pinpoint the molecular functions of myosin XI. Knocking out myosin XI in Arabidopsis (frequently using a '3KO' triple mutant of a group of closely related myosin XI genes) produced several phenotypes: trichomes, root hairs and pavement cells all showed defects in cellular differentiation (Peremyslov *et al.*, 2010; Ojangu *et al.*, 2012; Duan and Tominaga, 2018).

Myosin XI 3KO mutants have auxin-related and cell division phenotypes

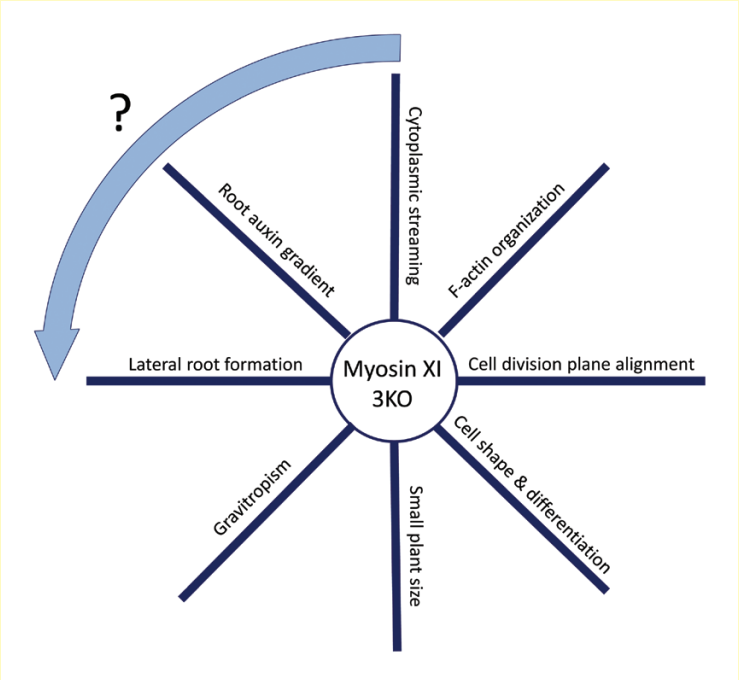
The new paper by Abu-Abied *et al.* (2018) goes much further, reporting on analyses of the root growth of myosin 3KO plants in detail. Surprisingly, they found that the plants show several defects that point to the phytohormone auxin: myosin 3KO plants have surplus lateral roots, they mislocalize the PIN1 auxin exporters of stele cells and they show an aberrant auxin gradient in the root. But the authors found additional phenotypes: myosin 3KO plants show striking division-plane orientation defects in the stele, milder defects of the same type in endodermal/epidermal cells of the root meristem, and an increase in the time cells spend in mitosis and cytokinesis. There had been little evidence that myosin XI is involved in auxin signalling and cell division, and so the paper contributes a significant step forward in our understanding of myosin XI function in vascular plants.

As exciting as these novel phenotypes may be, it is not that easy to put them into a simple coherent model. Are these phenotypes interrelated, and if yes, what is cause and what is effect? As humans we tend to infer causal relationships where sometimes there are just correlations. When speaking to my students I ask them initially to view the different phenotypes presented by a given mutant as entirely independent. I suggest comparing a set of phenotypes with the spokes of a wheel (see Box 1): *a priori* we know only about one causal relationship, which is the lack of a protein as the basis for all the separate phenotypes observed (here: the myosin XI motor). The establishment of further causal relationships needs careful experimental testing. Coming back to the myosin 3KO phenotypes presented by Abu-Abied and colleagues: could it be that the auxin effects are the basis for the cell division defects? Or is it rather the other way around?

While this is a tricky question, Abu-Abied *et al.* provide some evidence that myosin XI has a genuine function in cell division, suggesting that the cell division phenotypes observed may be independent from the defect in auxin signalling. Plant cell division involves the succession of three microtubule arrays, the preprophase band, the mitotic spindle and the cytokinetic phragmoplast. The phragmoplast expands centrifugally and assembles in its midline the cell plate, the nascent division wall. In wild-type cells, the phragmoplast and cell plate grow back to the position on the parental membrane that was occupied and marked by the preprophase band before mitosis (Lloyd and Buschmann, 2007). By following a complemented 3KO mutant expressing YFP coupled to a myosin XI isotype through cell division, Abu-Abied *et al.* found that the motor associates with early cell plates and with the rim of the expanding cell plate during late cytokinesis. Interestingly, myosin XI of Arabidopsis

Box 1. Phenotypic wheel for the Arabidopsis *myosin 3KO* mutant

The *myosin 3KO* mutant has many described phenotypes, as indicated on the spokes of the wheel (though not all phenotypes are depicted). It is tempting to speculate about causal relationships between these phenotypes, though this depiction emphasizes that we should initially view the different phenotypes presented by a given mutant as independent. However, one speculative interaction is indicated by the arrow.



is also found at the parental membrane for some time during pro-metaphase and then again during cytokinesis (Box 2). This membrane region is also known as the cortical division zone and is occupied by the preprophase band before nuclear envelope breakdown (Smertenko *et al.*, 2017). Given that the myosin 3KO mutant has defects in division plane orientation, the localization pattern described may be taken as support for the idea that myosin XI has a function in cell plate guidance. In another recent paper, Sun *et al.* (2018) observed a similar localization of a myosin XI paralogue to cell plates of the moss *Physcomitrella*.

Have you seen the bridge?

Research on the mechanism of cell division plane orientation and, specifically, cell plate guidance has produced a set of novel molecular players in recent years. Most of the proteins discovered point directly towards involvement of the microtubule cytoskeleton, perhaps because the respective plant mutants have strong phenotypes. However, imaging and inhibitor studies have previously shown that the actin cytoskeleton must have an important role in the process of cell plate guidance (Traas *et al.*, 1987; Sano *et al.*, 2005). It is therefore safe to assume that the 'bridge' connecting the phragmoplast and cell plate with the plasma membrane area of the cortical division zone should be made of both F-actin and microtubules.

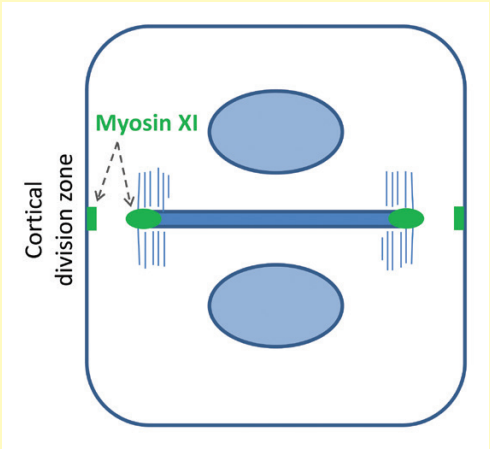
But the puzzle is incomplete as we know of only a handful of proteins involved and the mechanisms suggested to explain cell plate guidance are diverse (Lipka *et al.*, 2014; Wu and Bezanilla, 2014; Buschmann *et al.*, 2015). Importantly, recent reports suggest that myosins and a myosin-like sequence are involved. Myosin VIII, which was found to be present in the cortical division zone and on the phragmoplast of *Physcomitrella* was shown to interact with both F-actin and microtubules. Knockout mutants of myosin VIII showed division-plane orientation defects in the protonema of *Physcomitrella* (Wu and Bezanilla, 2014). Interestingly, the chimeric motor KCBP, which contains the myosin-like MyTH4-FERM domain in its N-terminus, is also found in the cortical division zone of dividing tobacco and Arabidopsis cells (Buschmann *et al.*, 2015). The paper by Abu-Abied *et al.* now shows that myosin XI of Arabidopsis associates with the cortical division zone and with the expanding cell plate. Because the 3KO mutants of myosin XI in Arabidopsis show skewed division planes in the stele the analyses provide important molecular genetic evidence that myosin is involved in the division plane alignment of higher plants.

On new tracks

Together, the new results show that the cortical division zone of plants contains a group of myosins and myosin-like sequences. This alone is curious, as the cortical division zone itself is thought be F-actin deficient (Cleary, 1995; Sano *et al.*,

Box 2. Dividing plant cell and myosin XI localization

The diagram shows a cell at cytokinesis with the phragmoplast microtubules and cell plate highlighted. Myosin XI (green) localizes to the parental plasma membrane (the cortical division zone) and to the edge of the cell plate.



2005). Non-plant eukaryotes divide by constriction using a contractile ring containing actin and myosin, while plants divide centrifugally using a phragmoplast with associated cell plate (Rasmussen *et al.*, 2011; Cheffings *et al.*, 2016). Perhaps the enigmatic cortical division zone of plants is simply a modified actomyosin ring without actin. The recent discovery of ROP (RHO of plants) signalling components localizing to the cortical division zone seems to support this notion (Zuo *et al.*, 2014; Stöckle *et al.*, 2016). However, the question arises as to what myosin is doing in the absence of actin in the cortical division zone. In the case of myosin VIII an interaction with microtubules of the phragmoplast periphery seems to be important. Given that myosin XI does not seem to interact with MyoB1 or MyoB2 receptors during cell division it is conceivable that this motor too has a microtubule-related function. One great challenge for plant cytokinesis research is to disentangle the contributions that actin and microtubule networks provide for cell plate guidance.

The paper by Abu-Abied (2018) shows convincing evidence that the 3KO myosin mutants have both auxin-related and cell division plane orientation defects. This in itself is remarkable as auxin gradients and division plane alignment are two major patterning devices employed by multicellular land plants (Buschmann and Zachgo, 2016; Du and Scheres, 2018). But it is perfectly possible that these are fully independent capacities of myosin XI: the auxin-related defects seen in myosin 3KO plants may result from dampened cytoplasmic streaming and concurrent effects on PIN1 transporter trafficking while the cytokinetic phenotype of myosin 3KO plants may point towards a direct role of myosin XI in cell plate guidance (as discussed above). Future research can clarify whether there is cross-talk between auxin transport and cell division plane orientation in the stele cells of Arabidopsis.

Keywords: Arabidopsis, cell division, microtubules, MyoB, myosin XI, polar auxin transport, root organogenesis.

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References

Abu-Abied M, Belausov E, Hagay S, Peremyslov V, Dolja V, Sadot E. 2018. Myosin XI-K is involved in root organogenesis, polar auxin transport and cell division. Journal of Experimental Botany **69,** 2869–2881.

Avisar D, Abu-Abied M, Belausov E, Sadot E, Hawes C, Sparkes IA. 2009. A comparative study of the involvement of 17 Arabidopsis myosin family members on the motility of Golgi and other organelles. Plant Physiology **150,** 700–709.

Buschmann H, Dols J, Kopischke S, Peña EJ, Andrade-Navarro MA, Heinlein M, Szymanski DB, Zachgo S, Doonan JH, Lloyd CW. 2015. Arabidopsis KCBP interacts with AIR9 but stays in the cortical division zone throughout mitosis via its MyTH4-FERM domain. Journal of Cell Science **128,** 2033–2046.

Buschmann H, Zachgo S. 2016. The evolution of cell division: from streptophyte algae to land plants. Trends in Plant Science **21,** 872–883.

Cheffings TH, Burroughs NJ, Balasubramanian MK. 2016. Actomyosin ring formation and tension generation in eukaryotic cytokinesis. Current Biology **26,** R719–R737.

Cleary AL. 1995. F-actin redistributions at the division site in living Tradescantia stomatal complexes as revealed by microinjection of rhodamine-phalloidin. Protoplasma **185,** 152–165.

Du Y, Scheres B. 2018. Lateral root formation and the multiple roles of auxin. Journal of Experimental Botany **69,** 155–167.

Duan Z, Tominaga M. 2018. Actin-myosin XI: an intracellular control network in plants. Biochemical and Biophysical Research Communications doi: 10.1016/j.bbrc.2017.12.169.

Higashi-Fujime S, Ishikawa R, Iwasawa H, Kagami O, Kurimoto E, Kohama K, Hozumi T. 1995. The fastest actin-based motor protein from the green algae, Chara, and its distinct mode of interaction with actin. FEBS Letters **375,** 151–154.

Lipka E, Gadeyne A, Stöckle D, Zimmermann S, De Jaeger G, Ehrhardt DW, Kirik V, Van Damme D, Müller S. 2014. The phragmoplast-orienting kinesin-12 class proteins translate the positional information of the preprophase band to establish the cortical division zone in *Arabidopsis thaliana*. The Plant Cell **26,** 2617–2632.

Lloyd C, Buschmann H. 2007. Plant division: remembering where to build the wall. Current Biology **17,** R1053–R1055.

Nebenfuhr A, Dixit R. 2018. Kinesins and myosins: molecular motors that coordinate cellular functions in plants. Annual Review of Plant Biology doi: 10.1146/annurev-arplant-042817-040024.

Ojangu EL, Tanner K, Pata P, Jarve K, Holweg CL, Truve E, Paves H. 2012. Myosins XI-K, XI-1, and XI-2 are required for development of pavement cells, trichomes, and stigmatic papillae in Arabidopsis. BMC Plant Biology doi: 10.1186/1471-2229-12-81

Peremyslov VV, Morgun EA, Kurth EG, Makarova KS, Koonin EV, Dolja VV. 2013. Identification of myosin XI receptors in Arabidopsis defines a distinct class of transport vesicles. The Plant Cell **25,** 3022–3038.

Peremyslov VV, Prokhnevsky AI, Dolja VV. 2010. Class XI myosins are required for development, cell expansion, and F-Actin organization in Arabidopsis. The Plant Cell **22,** 1883–1897.

Rasmussen CG, Humphries JA, Smith LG. 2011. Determination of symmetric and asymmetric division planes in plant cells. Annual Review of Plant Biology **62,** 387–409.

Reddy AS, Day IS. 2000. The role of the cytoskeleton and a molecular motor in trichome morphogenesis. Trends in Plant Science **5,** 503–505.

Sano T, Higaki T, Oda Y, Hayashi T, Hasezawa S. 2005. Appearance of actin microfilament 'twin peaks' in mitosis and their function in cell plate formation, as visualized in tobacco BY-2 cells expressing GFP-fimbrin. The Plant Journal **44,** 595–605.

Smertenko A, Assaad F, Baluška F, *et al*. 2017. Plant cytokinesis: terminology for structures and processes. Trends in Cell Biology **27,** 885–894.

Sparkes IA, Teanby NA, Hawes C. 2008. Truncated myosin XI tail fusions inhibit peroxisome, Golgi, and mitochondrial movement in tobacco leaf epidermal cells: a genetic tool for the next generation. Journal of Experimental Botany **59,** 2499–2512.

Stöckle D, Herrmann A, Lipka E, Lauster T, Gavidia R, Zimmermann S, Müller S. 2016. Putative RopGAPs impact division plane selection and interact with kinesin-12 POK1. Nature Plants **2,** 16120.

Sun H, Furt F, Vidali L. 2018. Myosin XI localizes at the mitotic spindle and along the cell plate during plant cell division in *Physcomitrella patens*. Biochemical and Biophysical Research Communications doi: 10.1016/j.bbrc.2018.01.082.

Tominaga M, Kimura A, Yokota E, Haraguchi T, Shimmen T, Yamamoto K, Nakano A, Ito K. 2013. Cytoplasmic streaming velocity as a plant size determinant. Developmental Cell **27,** 345–352.

Traas JA, Doonan JH, Rawlins DJ, Shaw PJ, Watts J, Lloyd CW. 1987. An actin network is present in the cytoplasm throughout the cell cycle of carrot cells and associates with the dividing nucleus. The Journal of Cell Biology **105,** 387–395.

Wu SZ, Bezanilla M. 2014. Myosin VIII associates with microtubule ends and together with actin plays a role in guiding plant cell division. eLife **3,** e03498.

Zuo Y, Oh W, Frost JA. 2014. Controlling the switches: Rho GTPase regulation during animal cell mitosis. Cellular Signalling **26,** 2998–3006.

Insight

Nematode-secreted peptides and host factor mimicry

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Plant pathogens have evolved diverse strategies to manipulate and co-opt host cellular function to enable infection. One strategy commonly employed by plant-parasitic cyst and root-knot nematodes is molecular mimicry of host proteins and small-molecule ligands. In an important new example of this phenomenon, Kim *et al.* (2018) have now identified a putatively secreted peptide from the root-knot nematode *Meloidogyne incognita* that mimics the Arabidopsis INFLORESCENCE DEFICIENT IN ABSCISSION (IDA) signaling peptide, which controls floral organ abscission and lateral root emergence.

Phytopathogens have evolved sophisticated mechanisms to enhance their ability to infect host plants. These include the molecular mimicry of cellular activities and functions, the ability to encode factors that mirror host proteins in function or structure to manipulate cellular functions for the pathogen's benefit. Such mimicry may enable the pathogen to establish infection while escaping the host's immune detection system. Although molecular mimicry is a common strategy employed by bacterial and viral pathogens in various animal systems (Stebbins and Galán, 2001; Elde and Malik, 2009), information about this phenomenon in phytopathogens is relatively limited.

The plant-parasitic cyst and root-knot nematodes, unlike other plant pathogens, appear to rely heavily on molecular mimicry to parasitize host plants (Hewezi, 2015). An example of cyst nematode mimicry is the secretion of effector proteins containing a conserved 12-amino acid C-terminal motif sharing strong sequence similarity with plant CLAVATA3/ESR (CLE) ligand peptides (Mitchum *et al.*, 2012). Similarly, multiple tandemly arrayed CLE-like motifs have been identified in various members of the secreted *Meloidogyne Avirulence Protein* (MAP) family (Rutter *et al.*, 2014). Thus, plant CLE ligands represent weak common targets for mimicry by two evolutionarily diverse species of plant-parasitic nematodes.

Other widely distributed mimics produced by plant-parasitic nematodes are the secreted chorismate mutases (Doyle and Lambert, 2003; Huang *et al.*, 2005; Vanholme *et al.*, 2009). Because of the absence of its substrate in animals, it is anticipated that parasitic nematodes mimic host chorismate mutase to alter secondary metabolic pathways, presumably to interfere with defense-related functions. Another effector that may mimic a host protein to subvert the defense response is the annexin-like effector from cyst nematodes (Patel *et al.*, 2010). It can complement the Arabidopsis *annexin1* mutant despite low amino acid sequence identity between the two proteins (Patel *et al.*, 2010). The identification of a number of effectors with putative mimicry functions from plant-parasitic nematodes suggests that these parasites use molecular mimicry to interfere with and/or exploit essential cellular functions required for parasitism.

The origin of nematode-encoded mimics

Horizontal gene transfer (HGT) seems to be the most plausible source for nematode mimics, with several potential such events having been reported in plant-parasitic nematodes (Scholl *et al.*, 2003, Danchin *et al.*, 2010; Haegeman *et al.*, 2011). This type of mimicry can be detected through sequence similarity and phylogeny. However, it is reasonable to expect that in some cases, because of the rapid evolution of parasitic nematodes, the nematode-encoded mimics may have little or no similarity to host factors that they imitate. Also, in nematode-encoded mimics generated through convergent evolution, parasitic