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Coordination of cell polarity and the patterning of leaf vein networks

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During development, the behavior of cells in tissues is coordinated along specific orientations or directions by coordinating the polar localization of components in those cells. The coordination of such cell polarity is perhaps nowhere more spectacular than in developing leaves, where the polarity of hundreds of cells is coordinated in the leaf epidermis and inner tissue to pattern vein networks. Available evidence suggests that the spectacular coordination of cell polarity that patterns vein networks is controlled by auxin transport and levels, and by genes that have been implicated in the polar localization of auxin transporters.

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Cell polarity and its coordination

During development, cell behaviors such as expansion and division are coordinated between cells along preferential or exclusive orientations or directions [1]. In plants, for example, cells in epidermal files of the root form hairs by locally expanding at their basal outer side [2,3], and cells in epidermal sheets of shoot organ primordia expand and divide along the proximodistal orientation [4,5]. How are these orientations and directions specified within cells and coordinated between cells?

In animals, where this question has been addressed extensively, the anisotropic localization of cellular components such as proteins provides cells with an internal compass that points in a specific direction [6]. These cell anisotropies, or cell polarities, are then coordinated between cells, often by mechanisms that rely on direct interaction between proteins bridging the plasma membranes of neighboring cells. These types of mechanisms are precluded in plants by a wall that separates the cells' plasma membranes. How then is cell polarity coordinated in plants?

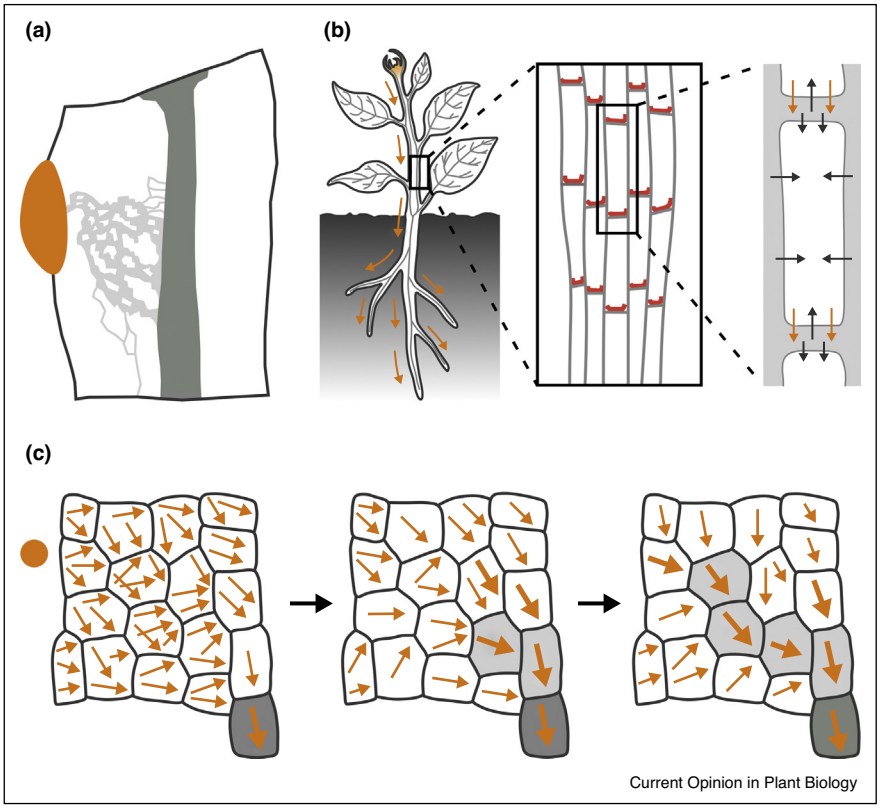
Here we will review evidence that the patterning of leaf vascular networks is an expression of coordination of cell polarity and that regulators of leaf vascular patterning encode regulators of such coordination. Therefore, available evidence suggests that understanding how leaf vascular networks are patterned will help understand how cell polarity is coordinated in plants. Other aspects of vascular development in leaves and other organs (e.g., [7–10,11•,12,13,14•,15•,16•,17•,18•,19•]), and coordination of cell polarity during other developmental processes (e.g., [20–23,24•,25•]), have been reviewed recently and comprehensively elsewhere.

Coordination of cell polarity during auxin-induced vascular-strand formation

That the formation of vascular strands is an expression of coordination of cell polarity was first suggested by experiments in which auxin had been applied to mature plant tissues [26–28]. Indeed, application of auxin to various tissues leads to the formation of continuous files of vascular cells that connect the applied auxin to the pre-existing vascular strands *basally* to the site of auxin application (Figure 1a). This polar response requires the application of polarly transported auxins [29] and is blocked by inhibitors of polar auxin transport [30], suggesting that it depends on the ability of the responding tissue to transport auxin polarly.

Normally, auxin is produced in large amounts in immature shoot-organs [31,32] and is transported to the roots through vascular strands [33,34] (Figure 1b). This apical–basal polarity of auxin transport is thought to derive from the localization of auxin efflux proteins at the basal end of auxin-transporting cells [35,36] (Figure 1b). Indeed, as a weak acid, auxin is mostly negatively charged at the neutral intracellular pH and can efficiently leave the cell only through specialized plasma-membrane-localized efflux proteins (Figure 1b). This model is certainly a simplification, but calculations based on known parameters suggest that it can account for the observed polar transport [37]; it can also account for the auxin-induced vascular-differentiation response, provided that auxin movement through a cell positively feed back on the localization of auxin efflux proteins to the site where auxin leaves the cell, as put forward by the 'auxin canalization hypothesis' [38,39].

Figure 1



Coordination of cell polarity during auxin-induced vascular strand formation. **(a)** Application of polarly transported auxins (orange) to plant tissues in which auxin is transported from top to bottom induces differentiation of vascular cells in continuous lines to form vascular strands (light gray) that connect the applied auxin to the pre-existing vascular strands (dark gray) basal to the application site. After [28,118]. **(b)** Left: auxin (orange) is produced in large amounts in immature shoot-organs and transported (orange arrows) to the roots by vascular strands. Middle: the shoot-to-root, apical–basal polarity of auxin transport derives from the polar localization of efflux transporters of the PIN-FORMED (PIN) family (red) at the basal plasma-membrane of vascular cells. Right: specialized efflux transporters are required for auxin to leave efficiently the cell (orange arrows) as auxin is mostly negatively charged at intracellular pH; by contrast, auxin is mostly uncharged at extracellular pH and can thus diffuse efficiently into the cell (dark gray arrows). Auxin can also enter the cell by the activity of transporters of the AUXIN RESISTANT1 (AUX1)/LIKE AUX1 family [119] and leave the cell by the activity of transporters of the ATP-BINDING CASSETTE B/P-GLYCOPROTEIN/MULTIDRUG RESISTANCE family [120], the nonpolar localization of which is, for simplicity, not shown. **(c)** Successive stages (connected by arrows) of vascular strand formation in response to application of auxin (orange circle) according to the 'auxin canalization hypothesis'. Positive feedback between cellular auxin efflux (orange arrows) and localization of auxin efflux proteins to the cellular site of auxin exit gradually polarize auxin transport (increasingly thicker orange-arrows). This occurs first in cells in contact with the pre-existing vascular strands (dark gray), which transport auxin along the original, apical–basal polarity of the tissue and thus orient auxin transport toward themselves. Large polar-auxin-transport capacity in selected cells leads to vascular differentiation (light gray) and drains auxin away from neighboring cells, thus inhibiting their differentiation. Reiteration of the process forms a continuous vascular strand that connects the applied auxin to the pre-existing vascular strands basal to the site of auxin application. *Source*: Figure inspired by [38].

This hypothesis proposes that in the cells between a site of auxin application and the pre-existing vascular strands, the applied auxin would initially move by diffusion in varied directions, and that in these cells, auxin efflux proteins would be localized isotropically, or nearly so, to the plasma membrane (Figure 1c). By efficiently transporting auxin along their original, apical–basal auxin-transport polarity, the pre-existing vascular strands would act as auxin sinks, thereby directing auxin movement in the neighboring cells and polarizing the localization of auxin efflux proteins in these cells. The induction of polar auxin transport in these cells would be gradually enhanced by positive feedback between auxin transport and efflux protein localization. By draining auxin increasingly more efficiently and polarly, these cells would in turn induce polar auxin transport and polarization of efflux protein localization in the cells above them, and inhibit the same processes in their lateral neighbors. Reiteration of these steps would result in preferential transport of auxin through limited cell files, which would eventually differentiate into vascular strands. During this process, chance localization of efflux proteins would be stabilized by positive feedback between auxin transport and efflux protein localization, resulting in random