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Fibers. A Model for Studying Cell Differentiation, Cell Elongation, and Cell Wall Biosynthesis¹

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A prominent anatomical feature in the inflorescence stems of *Arabidopsis* is the presence of fiber cells in the interfascicular regions (Fig. 1). The feasibility of using interfascicular fibers in the inflorescence stems of *Arabidopsis* as a model for studying cell differentiation, cell elongation, and cell wall biosynthesis has increased significantly since the completion of the *Arabidopsis* genome sequencing project. Because fibers are not essential for plant survival under greenhouse conditions, it is conceivable that mutants disrupting fiber cell differentiation would not be lethal and thus can be isolated. Considering the possibility that fiber and xylem cells evolved via activation of the same mechanisms for secondary wall formation (Mauseth, 1988), the study of fiber cell differentiation may also help us understand the molecular mechanisms regulating xylem cell differentiation. Recent studies on several *Arabidopsis* mutants have already demonstrated the feasibility of studying fiber differentiation in this model organism (Turner and Somerville, 1997; Zhong et al., 1997; Turner and Hall, 2000; Burk et al., 2001). The findings in these studies indicate that the molecular mechanisms underlying fiber differentiation have broad implications in our understanding of cell differentiation, cell elongation, and cell wall biosynthesis. In this essay, we show that the sclerenchyma cells present in the interfascicular regions of *Arabidopsis* inflorescence stems are fiber cells. We also present examples of mutants with defects in the development of interfascicular fiber cells.

Interfascicular fiber cells with thick secondary cell wall (Fig. 1, A and B) are formed when internodes of *Arabidopsis* inflorescence stems cease elongation. These fibers provide mechanical support to the heavy plant body as evidenced by the *ifl1* mutant in which lack of interfascicular fibers causes a pendent shoot phenotype (Zhong et al., 1997). Anatomical examination shows that in wild-type *Arabidopsis* inflorescence stems, three or four layers of interfascicular cells located next to the endodermis differentiate into

fiber cells (Fig. 1A; Zhong and Ye, 1999). These developing fiber cells are easily recognized in elongating internodes by their tapered ends (Fig. 2A). They undergo remarkable elongation and appear to reach their maximum length before massive secondary wall thickening occurs (G. Freshour, M.G. Hahn, and Z.-H. Ye, unpublished data). Based on their morphology and elongation pattern, these interfascicular sclerenchyma cells are apparently fiber cells (Fig. 2, B and C).

Because of their thick cell wall at maturity, which can be easily recognized by histological staining (Fig. 2B), fiber cells have traditionally been used for studying cell differentiation (Aloni, 1987). Early studies by Aloni (1976, 1978) and Sachs (1972) have convincingly shown that auxin polar transport regulates fiber differentiation, and auxin together with gibberellin and cytokinin is required for normal development of fiber cells (Aloni, 1987). Inspired by these early pioneering works, we screened the inflorescence stems of *Arabidopsis* for mutants with defects in the differentiation of interfascicular fibers. The *ifl1* mutant thus isolated completely disrupts normal differentiation of interfascicular fiber cells (Zhong et al., 1997). The interfascicular cells next to the endodermis remain parenchymatous with rectangular shapes (Fig. 2D), indicating that the mutation blocks the initiation of fiber cell differentiation. It is interesting that some interfascicular cells that are not destined to become fibers are ectopically induced to form short fiber-like cells in the *ifl1* mutant (Fig. 2D). The *IFL1/REV* gene has been shown to encode a homeodomain Leu-zipper protein (Zhong and Ye, 1999; Ratcliffe et al., 2000). We have found recently that the *ifl1* mutations dramatically reduce the auxin polar transport activity in both inflorescence stems and hypocotyls, and auxin polar transport inhibitors alter the normal differentiation of interfascicular fibers in the inflorescence stems of wild-type *Arabidopsis* (Zhong and Ye, 2001). These findings directly link the *IFL1/REV* functions to the early physiological studies regarding the role of auxin flow in fiber differentiation.

After initiation of cell differentiation, fiber precursor cells undergo significant elongation at both ends, a phenomenon that is referred as intrusive growth (Mauseth, 1988). A recorded extreme example is *Boehmeria nivea* in which fiber precursor cells are about

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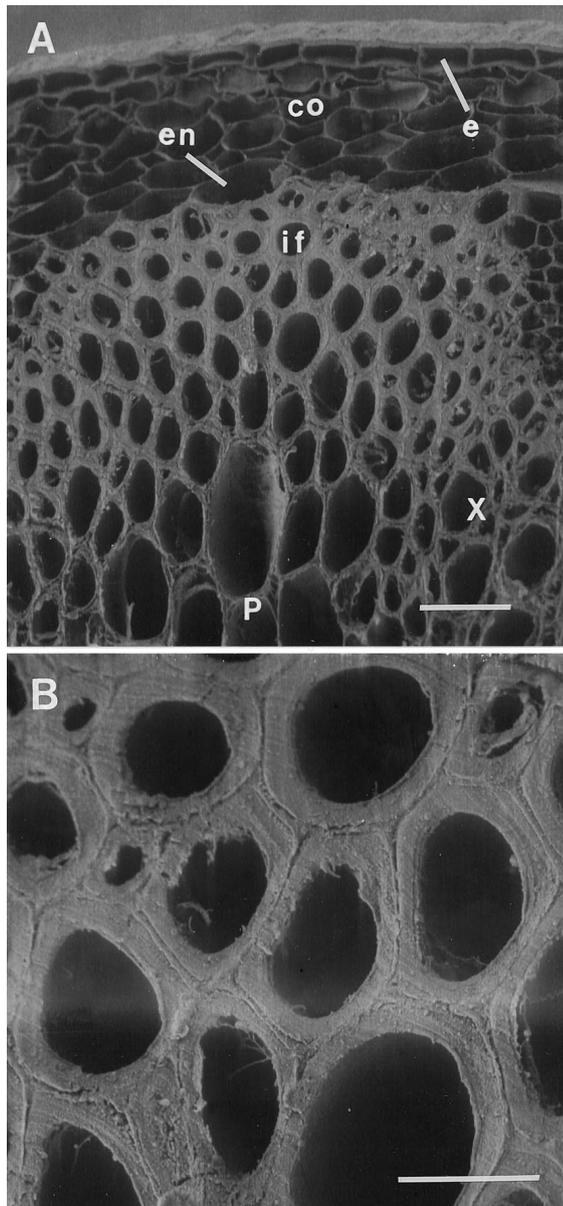


Figure 1. Scanning electron micrograph of cross sections of interfascicular regions in the inflorescence stems of wild-type Arabidopsis. A, Section showing layers of interfascicular fiber cells. B, Close-up of the interfascicular fiber cells with thick secondary wall. co, Cortex; e, epidermis; en, endodermis; if, interfascicular fiber; p, parenchyma; x, xylem. Bar in A = 25 μm ; bar in B = 10 μm .

20 μm long and they can elongate up to 550 μm . This suggests that fiber cells are an excellent system for studying the molecular mechanisms controlling cell elongation. We have recently isolated an Arabidopsis *fra2* mutant with a dramatic reduction in fiber length (Burk et al., 2001; Fig. 2E). The *fra2* mutation, which appears to be allelic to the *bot1* locus (Bichet et al., 2001), is also shown to reduce cell elongation in all plant organs. The *FRA2* gene encodes a protein with high similarity to katanin (hence, *FRA2* is renamed as *AtKTN1*; Burk et al., 2001). Katanin from animals has

been proposed to regulate microtubule disassembly by severing microtubules (Hartman et al., 1998). The putative function of *AtKTN1* as a microtubule-severing protein is supported by the evidence that the *fra2* mutation causes delays in the disappearance of the perinuclear microtubule arrays and in the establishment of transverse cortical microtubule arrays in elongating cells (Burk et al., 2001). This suggests that the microtubule-severing activity might play an important role in regulating the dynamic changes of microtubules during the initiation and continuation of cell elongation.

After elongation, fiber cells are thickened with a massive amount of secondary wall that enables fibers to function as an excellent mechanical tissue (Mauseth, 1988; Fig. 1B). Thus, it is conceivable that fiber cells are an ideal system for isolation of genes involved in secondary wall thickening. This has been demonstrated by the study of *irx* mutants and the *gpx* mutant. The *irx* mutations dramatically reduce the secondary wall thickening of both interfascicular fiber cells and xylem cells, and this reduction in secondary wall thickening is directly caused by a decrease of cellulose deposition (Turner and Somerville, 1997). The *IRX1* and *IRX3* genes have recently been cloned and they are shown to encode distinct classes of catalytic subunits of cellulose synthase (Taylor et al., 1999, 2000). It has been proposed that both *IRX1* and *IRX3* are part of the cellulose synthase complex, which is essential for secondary wall thickening. The *gpx* mutant exhibits a lack of secondary wall thickening in some of the interfascicular fiber cells and vessel elements (Turner and Hall, 2000), and it has been suggested that the *GPX* gene product regulates the deposition of secondary cell wall.

The Arabidopsis mutant examples presented above have clearly demonstrated the usefulness of fibers for studying various aspects of cell differentiation. It is apparent that we are far from a complete understanding of the molecular mechanisms underlying cell differentiation, cell elongation, and secondary wall thickening during fiber formation. We anticipate that further studies on the fiber differentiation in the inflorescence stems of Arabidopsis will yield many exciting insights into how cell differentiation is regulated at the molecular level.

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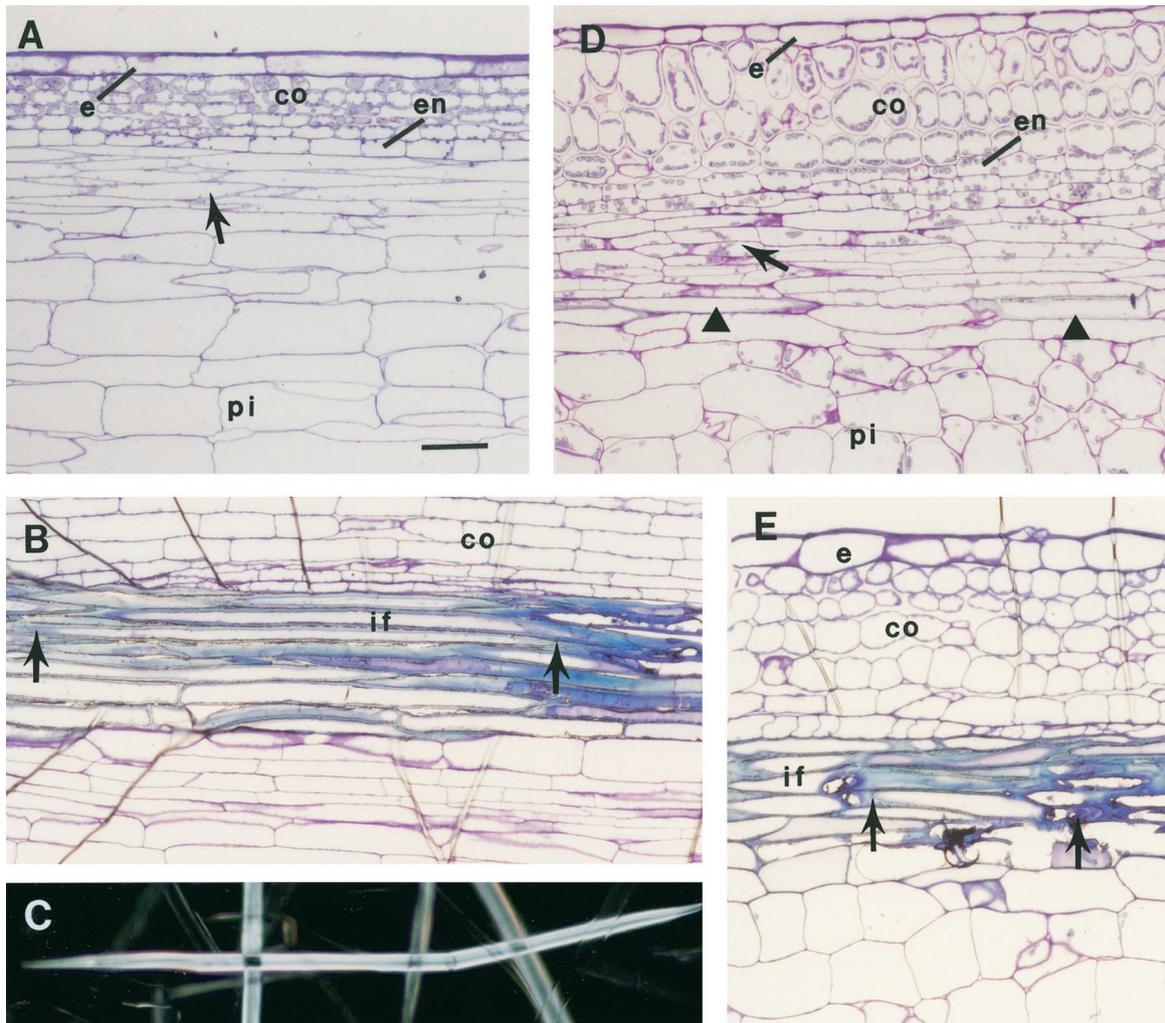


Figure 2. Anatomy of the interfascicular fibers in the inflorescence stems of wild-type *Arabidopsis*, *ifl1* and *fra2* mutants. A, Longitudinal section of the wild type showing interfascicular fiber initial cells (arrow) with two tapered ends. B, Longitudinal section of the wild type showing macerated interfascicular fiber cells with thick secondary wall. Arrows point to the ends of a fiber cell. C, Visualization of macerated wild-type fiber cells under polarized light. D, Longitudinal section of the *ifl1* mutant showing that interfascicular cells (arrow) located next to the endodermis remain parenchymatous. Note the ectopic induction of fiber-like cells (arrowheads) in the interfascicular region, which is normally not destined to form fiber cells. E, Longitudinal section of the *fra2* mutant showing interfascicular fiber cells with a dramatic reduction in length. Arrows point to the ends of a fiber cell. co, Cortex; e, epidermis; en, endodermis; if, interfascicular fiber; pi, pith. Bar in A = 84 μm for A through E.

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