CHAPTER 12

A New Member of the CtBP/BARS Family from Plants: Angustifolia

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Abstract

The ANGUSTIFOLIA (AN) gene in *Arabidopsis thaliana* (L.) Heynh. is the first homolog of the CtBP/BARS gene family identified in plants and is responsible for the polarity-dependent control of leaf cell expansion. This review compares the sequence homology and functional similarity of the AN protein with authentic animal CtBP/BARS family proteins. AN homologs have been found in both angiosperms and mosses, suggesting AN is conserved in terrestrial plant genomes. The AN subfamily is unique in having not only the D-isomer-specific 2-hydroxy acid dehydrogenase (D2-HDH) motif that is conserved among the CtBP/BARS family but also putative LxCxE/D and nuclear localization signal (NLS) motifs and a long C-terminal region. The absence of the catalytic triad, which is conserved in all D2-HDH sequences and is believed to be essential for the corepression activity of CtBP, suggests that AN might differ, at least in part, from CtBPs in molecular function. In addition, the distribution and density of the Golgi apparatus is normal in a null allele of the *an* mutant, suggesting that AN might not have a BARS function. An analysis of cytoskeletons in *an* mutant leaf cells suggests that *AN* might play an important role in controlling the arrangement of cortical microtubules that is plant-specific cytoskeletons. With all these attributes, AN appears to be the third member of an enigmatic family, CBA = CtBP/BARS/AN, which regulates aspects of developmental and organelle control in animals and plants.

Angustifolia—A Polarity-Dependent Regulator of Leaf Cell Expansion

Focusing on mechanisms that govern the polarized growth of leaves in the model plant *Arabidopsis thaliana* (L.) Heynh. (arabidopsis), we used mutational studies to identify two genes that act independently of each other to regulate polar cell elongation in leaves: *AN* regulates the width of leaves, and *ROT3* regulates the length.[1-4] The *angustifolia* (*an*) mutant of arabidopsis (Fig. 1) was originally isolated by Rédei.[5] The mutation in the leaf-specific *an* phenotype is caused by a specific defect in the elongation of leaf cells in the transverse (width) direction[1,2](Fig. 1A,C). This polar defect was observed in all the leaf cells examined, including epidermal cells, trichomes, and parenchymatous cells (Fig. 1B-D). The altered direction of cell elongation was particularly evident in palisade cells, where expansion in the leaf-width direction

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CtBP Family Proteins, edited by G. Chinnadurai.

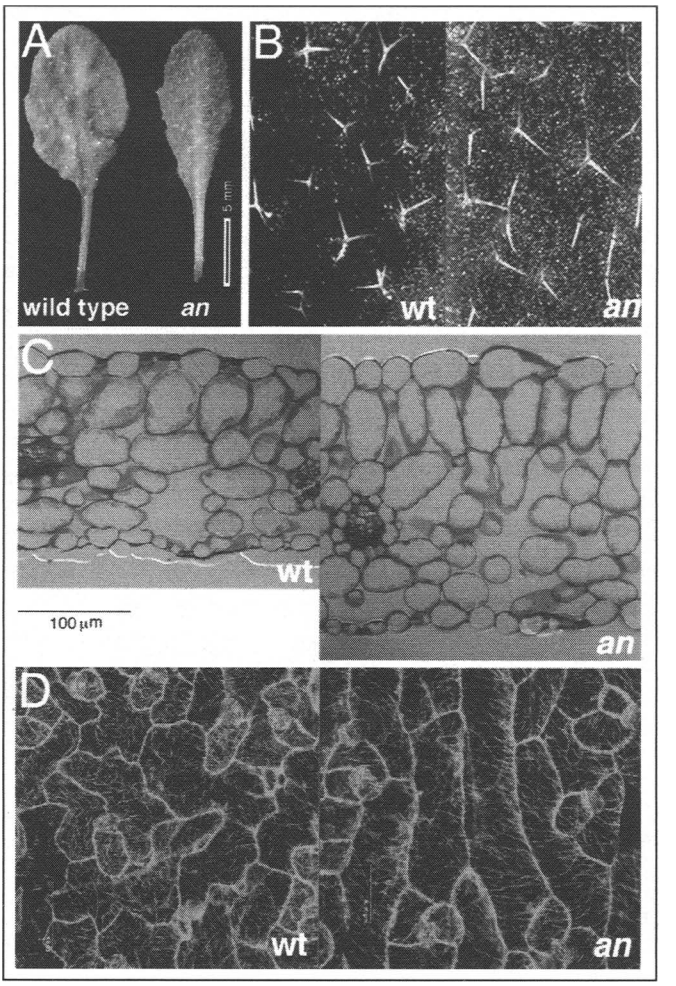

Figure 1. Morphological phenotypes of the *angustifolia* (*an*) mutant of arabidopsis. A) Gross morphology of leaves of the wild type and *an* mutant. Note the narrow shape of the *an* leaf. Bar, 5 mm. B) Trichomes on the leaf. Wild-type trichomes are three-branched in most cases (left), whereas *an* trichomes are two-branched. The longitudinal direction of the panel corresponds to the leaf-length direction. C) Cross-section of leaves of the wild type (left) and *an* mutant (right). Note the narrow, longer shape (in the leaf-thickness direction) of the *an* leaf cells. Bar, 100 μm. D) Arrangement of cortical microtubules (MTs) in leaf epidermal cells. The longitudinal direction of the panel corresponds to the leaf-length direction. Compared with wild-type MTs, the *an* MTs are arranged more simply and parallel to the leaf-width direction. Modified from Kim et al.[6]

was decreased, while elongation in the leaf-thickness direction was increased (Fig. 1C). Coincident with the defect in the palisade cells, the number of protrusions in epidermal cells was decreased, particularly in the leaf-width direction (Fig. 1D). A decreased number of branchings in trichome cells is also attributable to the same defect (Fig. 1B). Thus, the *AN* gene is thought to be the key gene to regulating the polar elongation of leaf cells in the leaf-width direction.[2] Cytological analysis showed that the *an* mutant has abnormally arranged cortical microtubules in leaf cells (Fig. 1D),[6,7] suggesting that *AN* might regulate polarity-dependent elongation of leaf cells via control of the arrangement of cortical microtubules.[6]