

Calcium- and BTB domain protein-modulated PINOID protein kinase directs polar auxin transport

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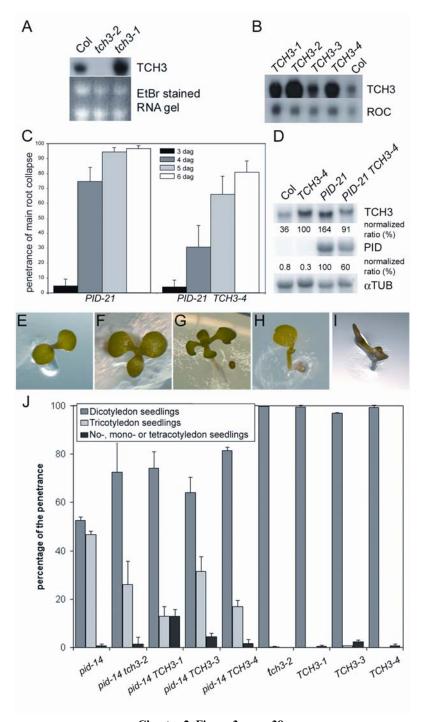
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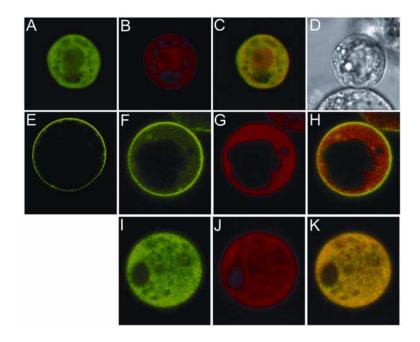
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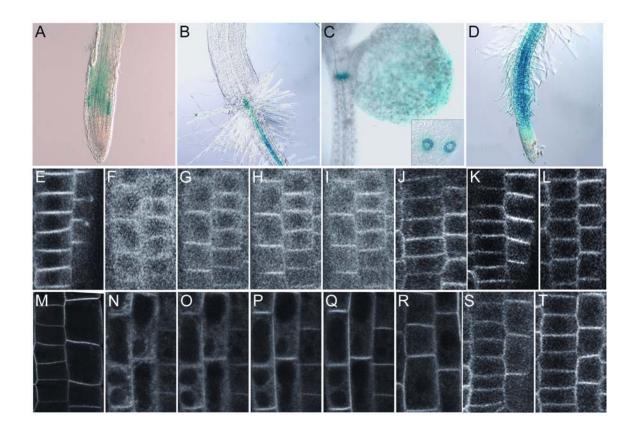
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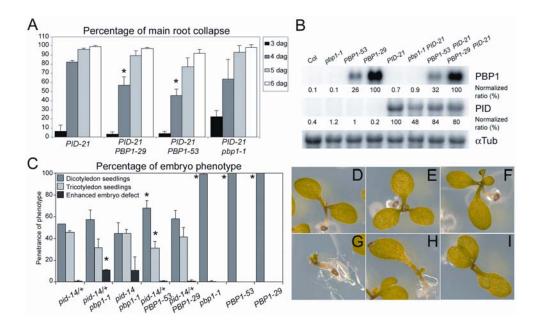
Chapter 2, Figure 3, page 38.
TOUCH3 is a negative regulator of PINOID *in vivo*.



Chapter 2, Figure 4, page 40.
TOUCH3 and PINOID co-localization is auxin-dependent.



Chapter 2, Figure 6, page 42.
TOUCH3 and auxin cause PINOID to dissociate from the plasma membrane.

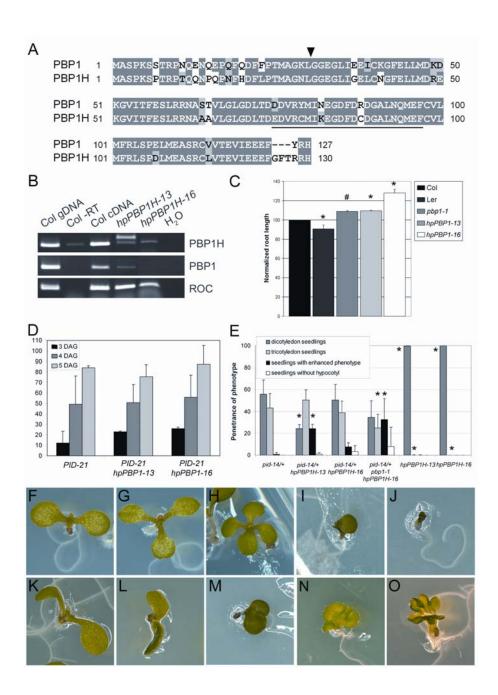


Chapter 3, Figure 2, page 60.

pbp1-1 loss-of-function enhances pid-14 embryo phenotypes.

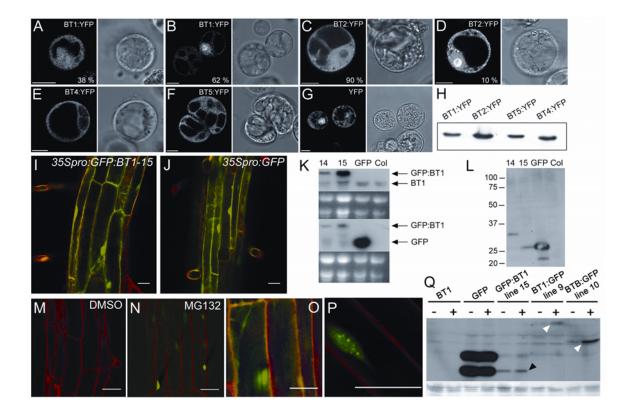


Chapter 3, Figure 3, page 63. *pbp1-1* loss-of-function partially rescues *pid-14* inflorescences.



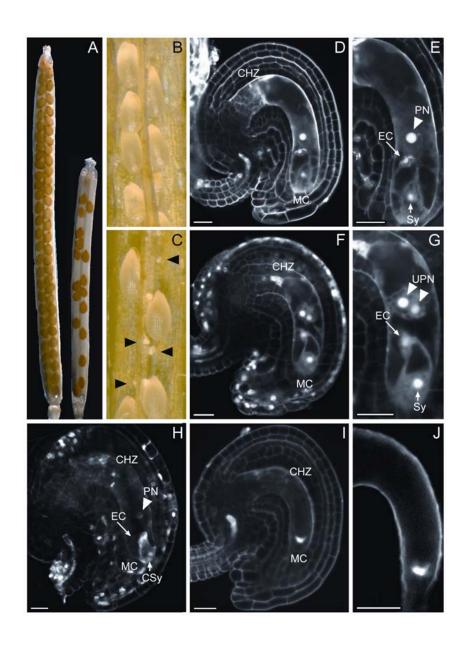
Chapter 3, Figure 5, page 65.

PBP1 and PBP1H act redundantly on root growth, embryo patterning and leaf phyllotaxis.

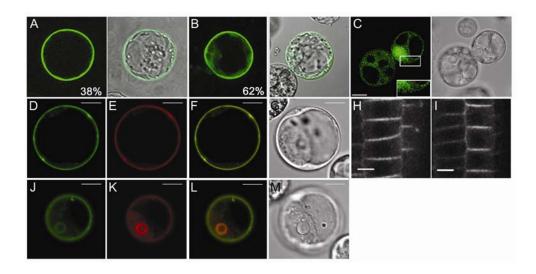


Chapter 4, Figure 2, page 81.

The expression pattern of the BT proteins suggests a genetic redundancy among the family members.

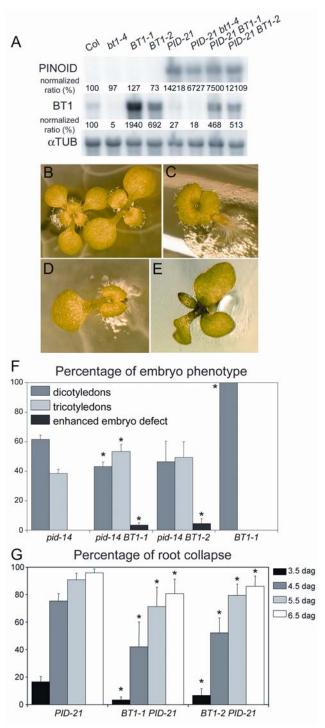


Chapter 4, Figure 4, page 86.The quintuple *bt* loss-of-function is gametophytic lethal.



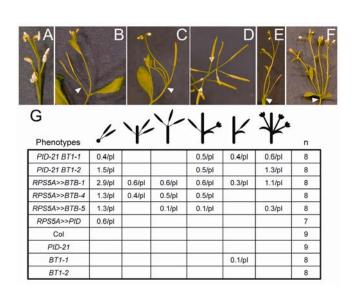
Chapter 5, Figure 3, page 105.

BT1 and PINOID co-localize at the plasma membrane in Arabidopsis protoplasts.



Chapter 5, Figure 4, page 107.

Overexpression of *BT1* enhances *pid-14* embryo phenotypes and inhibits *35Spro:PID-21* root meristem collapse.



Chapter 5, Figure 6, page 110.

RPS5Apro>>BTB expression results in axilliary branch and inflorescence meristem defects.