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PINOID IS POSITIVE REGULATOR OF AUXIN EFFLUX FROM THE CELL

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Cell-to-cell auxin transport is thought to be achieved via auxin influx and efflux proteins in the plasma membrane. The mutants' phenotypic relationship has implied that the efflux carriers (PINs) would be regulated by PINOID (PID), a serine/threonine protein kinase. Here, we address the positive linearity between PINs and PID using the auxin-sensitive root hair cell system of *Arabidopsis thaliana*. Overexpression of *PID* (PIDox), specifically in the root hair cell using the *AtEXPA7*promoter, suppressed root hair growth. Root hair cell-specific overexpression of *PIN3* (PIN3ox) also strongly inhibited root hair growth. However, PIDox and PIN3ox transformants restored root hairs almost to the wild-type level by exogenousauxin or by the chemicals inhibiting PID and PIN activities. The PID-GFP and PIN3-GFP fusion proteins were localized at the hair cell boundary, and this localization was disrupted by brefeldin-A and staurosporine. The mutant PID defective in its kinase activity, however, completely impaired the capability of localizing at the cell boundary and inhibiting root hair growth. Our results suggest that PID facilitates the PIN activity in vicinity to them most likely by modulating the membrane trafficking of PIN-harboring vesicles. In the root hair single cell system, PIDox hyper-activates auxin efflux from the hair cell, resulting in the shortage of intracellular auxin and consequently the inhibition of root hair growth.