SL201 Are auxin transport and auxin action on cell elongation interlinked through the auxin efflux system ?

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Cell-to-cell transport of auxin in plant tissues involves two distinct cellular processes occuring at the plasma membrane, namely influx (uptake) of auxin and its efflux (exit). The latter is mediated through auxin efflux carrier and considered to be rate-limiting in the auxin transport system. Phytotropins such as 1-N-naphthylphthalamic acid (NPA) inhibit auxin transport by blocking auxin efflux upon their specific binding to the carrier complex (phytotropin receptor). Either changes in the number of NPA binding sites or changes in the binding affinity are known to result in altered auxin transport in vivo. Inhibition of polar auxin transport by ethylene is attributable to ethylene-induced reduction in the number of NPA binding sites. Results from studies using isolated microsomal vesicles indicate that increased affinity of NPA binding to the receptor is caused by inhibition of phosphoprotein dephosphrylation. In vivo auxin transport as well as efflux of intravesicular auxin in microsomal membranes are promoted, and the NPA inhibition of these processes is abolished, by phosphatase inhibitors. Findings of auxin transport studies with cycloheximide implicate cellular induction of proteineous inhibitor of auxin transport upon wounding A strong similarity in molecular specificity for auxin transport on one hand and auxin action on cell elongation on the other hand has been noted. Also, several striking similarities exist between auxin action and its transport. These include adaptation (desensitization) of the system, optimal curves, initial kinetics, and the role of ethylene therein. In *Ranunculus* petioles where cell elongation is known to be rather promoted by ethylene, auxin transport is likewise promoted by the gas. In tissue segments, intracellular auxin level can be brought about by phytotropins specifically blocking auxin exit without affecting auxin uptake. Auxin-induced ethylene production in tissue segments treated with phytotropins is known to be actually increased compared with the phytotropin-free control. However, both NPA and its analog 2-(1-pyrenoyl) benzoic acid (PBA) inhibit auxin-induced cell elongation in *Ranunculus* petioles. Recently NPA inhibition of cell elongation in light-grown *Arabidopsis* has been reported by others. A possible link between auxin transport and auxin action on cell elongation through a common system will be discussed.