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THE ROLE OF BRASSINOSTEROIDS IN REGULATION OF PHOSPHOLIPID SIGNALING IN PLANT CELLS

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Introduction. Brassinosteroids are plant steroid hormones that play key role in the regulation of plant growth and stress tolerance. The aim of this work was to investigate the brassinosteroid-induced dynamics of phospholipid signalling in normal growth conditions and under salt stress, and to assess the roles of different lipid signalling pathways in the production of lipid second messengers.

Methods. Levels of fluorescently-labelled lipid products of BODIPY-phosphatidylcholine hydrolysis were analyzed in seedlings of *Brassica napus*, wild-type and AtCAX1-overexpressing *Nicotiana tabacum*. Plants were grown in solution containing salt (NaCl) or brassinosteroids (24-epibrassinolide) during 7 days and then separated roots were additionally treated for 2 hours by brassinosteroids or salt, respectively. Another set of plants was pretreated by N-ethylmaleimide, an inhibitor of phosphatidic acid phosphatase, or R59022, an inhibitor of diacylglycerol kinase, and then subjected to brassinosteroids for 2 h.

Results. Salt stress and brassinosteroids induced dramatic elevation of second messenger phosphatidic acid (PA) and diacylglycerol (DAG) levels in *B. napus* plants. Moreover, DAG and PA were shown to accumulate on a higher level when plants were initially grown in brassinosteroid-containing solution and then treated by salt. Accumulation of DAG and PA in response to brassinosteroids was reduced in plants subjected to R59022, but not NEM.

Discussion. DAG accumulation in response to brassinosteroids is mediated by non-specific phospholipase C hydrolyzing phosphatidylcholine, in spite of phosphatidic acid phosphatase that dephosphorylates PA. PA accumulation induced by brassinosteroids is mediated by further activation of diacylglycerol kinase phosphorylating DAG, the product of non-specific phospholipase C.

Conclusions. The results suggest that brassinosteroid signalling and brassinosteroid-induced plant adaptation to salt stress are mediated by DAG and PA as lipid second messengers.

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