
HANELE TUOMINEN

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SPEAKER AT:

THE DEATH OF PLANT CELLS. FROM PROTEASES TO FIELD APPLICATIONS



October, 2nd and 3rd, 2013, Barcelona

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Hannele Tuominen received a PhD at the Swedish University for Agricultural Sciences in Umeå, Sweden and is, after a post doc period at the Institute of Biotechnology in the University of Helsinki in Finland, active as an associate professor at Umeå Plant Science Centre (UPSC) in Umeå, Sweden. She has a long-term interest in wood formation in forest trees and in the herbaceous model system *Arabidopsis thaliana* that also makes quite a bit of "wood" especially in the hypocotyl after a couple of months' growth period. Her current research focuses on cell death in xylem elements and how this impacts on wood properties and biomass production of forest trees. Current interests include comparative studies between different developmental cell death processes in *Arabidopsis* and the analysis of xylem cell death in a new model organism, the Norway spruce.

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Post Mortem Program During Developmental Cell Death in Arabidopsis

Xylem cell death is required for proper function of the vessel elements in water transport. We have identified *Arabidopsis thaliana* METACASPASE9 (AtMC9) as a putative regulator of vessel cell death. Reverse genetic analysis of AtMC9 T-DNA knock-out lines showed normal progression of protoxylem vessel cell death in roots of young seedlings. However, detailed electron microscopic analysis of the mutant revealed a delay in post-mortem clearance of the protoxylem vessel elements, supporting a role for AtMC9 in autolytic processes during vessel cell death. Localization of the AtMC9 protein in cytoplasmic aggregates of the late maturing vessel elements and the effect of AtMC9 on both stability and activities of vacuolar localized papain-like cysteine proteases (PLCPs) supported this conclusion. Additional insight was obtained in newly established *Arabidopsis* tracheary element (TE) cell cultures, where almost complete suppression of AtMC9 expression in several transgenic RNAi lines resulted in altered cysteine protease activities and increased presence of undegraded acidic bodies in the vacuole during TE differentiation. We propose therefore that AtMC9 is part of a control mechanism which guarantees, in living TEs, degradation of acidic bodies of the vacuole and, in dead TEs, complete degradation of the vessel cell contents by activating unknown PLCPs. We also present results on an upstream regulatory mechanism of AtMC9 function, and finally propose function of AtMC9 and related hydrolytic enzymes in another developmental context.

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