B·DEBATE International Center for Scientific Debate BARCELONA

CRAG CENTRE DE RECERCA EN AGRIGENÒMICA CSIC IRTA UAB

MORTEN PETERSEN CV

SPEAKER AT:

THE DEATH OF PLANT CELLS.

FROM PROTEASES TO FIELD APPLICATIONS

October, 2nd and 3rd , 2013, Barcelona

Morten Petersen, Associate Professor at the Department of Biology, Copenhagen University, Copenhagen

Morten Petersen obtained an MSc in Biology from U. Copenhagen on research he performed with Prof. Peter Ulvskov, then of the Danish Agronomic Institute of the Ministry of Agriculture on pod shatter in oil seed rape. He then obtained a PhD in 2001 from U. Copenhagen. His thesis work was carried out with Prof. John Mundy at the Institute of Molecular Biology, U. Copenhagen. His thesis research led to the discovery of the first MAP kinase knock-out in plants. His Post-Doctoral research focused on comparative studies of programmed cell death using post-genomic models including the plant Arabidopsis, the worm C. elegans, and humans. From 2003 Morten Petersen initiated work with C. elegans during visits to the group of Prof. Michael Hengartner (U. Zurich) and in 2005 he also initiated work with human cell cultures with the group of Professor Niels Ødum at Copenhagen University. During the following years he focused his research on autophagic cell death in plants. Since 2009 his research, as an Associate Professor at the Department of Biology, Copenhagen University, has focused on basal cell biological processes, including MAP kinase signalling in plant innate immunity and autophagic cell death in plants and animals. Apart from basal discoveries he has also focused on applied research. Currently, this includes ways to increase stress tolerance in crops and rapid production of cereals with increased resistance to microbial pathogens.

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Pleiotropic Effects of Autophagy and NPR1 Deficiencies in Cell Death During Immune Responses and Development

Autophagy is a homeostatic degradation and recycling process that is also involved in defense against microbial pathogens and in certain forms of cellular suicide. Autophagy has been proposed to negatively regulate immunity associated cell death related to the hypersensitive response (HR), as older autophagy-deficient (atg) mutants are unable to contain this type of cell death 5-10 days after infection. Such propagating cell death was found to require Non-expressor of PR genes (NPR1), but did not occur in younger atg mutants. In contrast, we find that npr1 mutants are not impaired in rapid programmed cell death activation upon pathogen recognition. More importantly and for the first time, we provide molecular evidence that the NPR1 dependent spreading cell death in older atg mutants originates from an inability to cope with the excessive accumulation of ubiquitinated proteins and subsequent ER stress which derive from salicylic acid (SA)-dependent systemic acquired resistance (SAR). Thus, autophagy does not regulate cell death via a negative feedback loop through NPR1. Instead, npr1 loss-of-function indirectly rescues older atg mutants because atg npr1 double mutants are insensitive to SA and fail to induce SAR that would otherwise produce terminal stress in autophagy deficient cells.

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