B·DEBATE International Center for Scientific Debate BARCELONA

CRAG CENTRE DE RECERCA EN AGRIGENÒMICA CSIC IRTA UAB

RICHARD VIERSTRA CV

SPEAKER AT:

THE DEATH OF PLANT CELLS.

FROM PROTEASES TO FIELD APPLICATIONS

October, 2nd and 3rd , 2013, Barcelona

Richard Vierstra, Professor of Genetics in University of Wisconsin, Madison, USA

Dr. Richard Vierstra earned his BS in biology and chemistry at the University of Connecticut in 1972, his Ph.D in plant biology from the DOE-Plant Research Laboratory at Michigan State University in 1980, and was a postdoctoral fellow with Dr. Peter Quail at the University of Wisconsin (1980-1983). He joined that faculty of the University of Wisconsin in 1984 and is currently the Stanley J. Peloquin Professor of Genetics. Rick was a Fulbright senior scholar at the University of Melbourne Australia (1993-1994). He served on the editorial boards of Plant Physiology and the Journal of Biological Chemistry and has been on numerous NIH, NSF, DOE and USDA study sections, including section chair for the USDA-NRICGP Plant Genetics Mechanisms program in 2001. Rick currently is a member of the Board and Treasurer for the International Society of Plant Molecular Biologists and on the Executive Committee for the American Society of Plant Biologists. Rick's research interests include: (1) mechanisms, regulation, and evolution of the ubiquitin/26S proteasome proteolytic system using Arabidopsis as the model; (2) roles of autophagy in plant growth development and survival under nutrient limitations; (3) functions of SUMOylation during the plant stress response; and (4) understanding the phytochrome signaling system in plants and microorganisms at the atomic level, using x-ray crystallography, NMR spectroscopy, and single particle EM. This work has resulted in almost 220 research publications, the training of 41 postdoctoral fellows, 25 Ph.D students and a host of undergraduates, and numerous awards including Fellow of AAAS (2002).

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Functions and Regulation of ATG8-Mediated Autophagy in Plants

Autophagy-mediated turnover plays an essential role in cellular housekeeping by removing damaged organelles and unwanted cytoplasmic constituents, and is critical for plant defense and robust nutrient recycling, especially during nitrogen and fixed-carbon starvation. This 'self eating' is mediated by a conjugation system that modifies a pair of ubiquitin-like proteins ATG12 and ATG8 to eventually form an autophagic vesicle coated with the ATG8-phosphatidylethanolamine (PE) adduct. The ATG8-PE serves two purposes, one is to help direct the transport of autophagic vesicles to the vacuole for breakdown, and the other is to serve as a docking platform for a suite of ATG8-interacting proteins that selectively tether appropriate cargo within the vesicle lumen before enclosure. Using an extensive collection of atg mutants in Arabidopsis, we have characterized the pathway responsible for ATG8/12 conjugation and the kinase cascade that connects metabolic and environmental cues to the formation and transport of autophagic vesicles. A key regulatory step is controlled by the ATG1/13 kinase complex that works downstream of the TOR signaling system and ATG8/12 modification. It is not only necessary for vacuolar deposition of autophagic bodies but also becomes a substrate through the interaction of several subunits with ATG8, thus providing feedback control on autophagic turnover. In addition to bulk turnover, we found that the ATG system is responsible for the turnover of organelles like mitochondria and peroxisomes and large cytoplasmic complexes such as the 26S proteasome when dysfunctional or no longer needed. Proteasome turnover appears to be mediated by prior ubiquitylation of numerous core subunits. Using maize as a model, we have also begun to describe the roles of ATG8-mediated autophagy in crops. Our initial genetic studies revealed a key role in productivity by mobilizing nitrogen when limiting.

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WITH THE COLLABORATION OF: VIB Agrisera Antibodies for plant sciences