B·DEBATE International Center for Scientific Debate BARCELONA

CRAG CENTRE DE RECERCA EN AGRIGENÒMICA CSIC IRTA UAB

PIERRE GOLSTEIN CV

SPEAKER AT:

THE DEATH OF PLANT CELLS.

FROM PROTEASES TO FIELD APPLICATIONS

October, 2nd and 3rd , 2013, Barcelona

Pierre Golstein, Centre d'Immunologie de Marseille-Luminy, INSERM-CNRS-Université de la Méditerranée, Marseille France

Pierre Goldstein, MD, PhD, trained at the Institut Pasteur, Paris, the Dept. of Tumor Biology (Pr. G. Klein) of the Karolinska Institutet, Stockholm, and the Tumor Immunology Unit (Pr. Mitchison) at University College London. He has been working at the Centre d'Immunologie INSERM-CNRS de Marseille-Luminy as a head of group for many years. He first studied mechanisms of T cell mediated cytotoxicity, demonstrated the existence of CTLA-1 and CTLA-3 (Granzymes B et A, respectively), in the perforin-granzyme pathway of cytotoxicity, and identified the Fas pathway of cytotoxicity, leading to the cloning of the Fas Ligand. This research also led to CTLA-4 and CTLA-8 (Interleukin-17), now studied by others in a therapeutic perspective. He then shifted to mechanisms of programmed cell death per se, studying first interdigital cell death in the mouse embryo, then focussing on developmental cell death in the protist Dictyostelium discoideum. Dictyostelium is an excellent genetically tractable model organism for the study of this death, which is being investigated as to its phenomenology and through genetic approaches as to its molecular bases.

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Cell Death in *Dictyostelium*

Dictyostelium discoideum, a eukaryote, a protist, a slime mold, is unicellular in favorable conditions. It shows upon starvation multicellular development leading to fruiting bodies. Each of these fruiting bodies is made of a mass of spores on top of a stalk. This stalk is made of dying or dead cells. This cell death, developmental by definition, can be mimicked in vitro in cell monolayers allowing morphological study and genetic manipulations. Dictyostelium offers two main advantages for the study of this cell death. First, its genome, now sequenced, is small, compact, and haploid, favoring insertional mutagenesis. Second, from a cell death point of view this genome does not encode caspases or bcl-2 family members, thus in this model organism there is no apoptosis machinery that could interfere with the non-apoptotic cell death mechanisms at play. The results to be reported include a description of successive steps of this developmental cell death as mimicked in vitro (including paddle cell formation, rounding, vacuolization and cellulose shell formation), the induction and role(s) of autophagy in this cell death system, the requirement for this cell death of autophagy plus a qualitatively different second signal, a preliminary account of the general design of this cell death, corresponding signaling pathways with markers thereof including required molecules identified through insertional mutagenesis, and description of mitochondrial lesions.

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