B·DEBATE International Center for Scientific Debate BARCELONA

CRAG CENTRE DE RECERCA EN AGRIGENÒMICA CSIC IRTA UAB

CRISTINA MUÑOZ-PINEDO CV

SPEAKER AT:

THE DEATH OF PLANT CELLS.
FROM PROTEASES TO FIELD APPLICATIONS

October, 2nd and 3rd , 2013, Barcelona

Cristina Muñoz-Pinedo, Leader of the Cell Death Regulation Group at IDIBELL, Barcelona, Spain

Cristina Muñoz-Pinedo leads the Cell Death Regulation group at IDIBELL (Bellvitge Biomedical Research Institute) in Barcelona, Spain. She studied Biology in the University of Sevilla, and she started her scientific career in the field of cell death and cancer metabolism under the supervision of Dr. Abelardo López-Rivas at the CSIC, in Granada, Spain. After several short stays in international laboratories she received her PhD from the University of Granada in 2001. She then moved to San Diego to work under the supervision of Doug Green at the La Jolla Institute for Allergy and Immunology, where she studied the role of the mitochondria during cell death as an initiator of the apoptotic process and as a "victim" of caspase activation. After a short stay in St. Jude Children's Research Hospital, in 2006 she moved back to Spain to start a lab whose main interest is to understand why and how cells die when deprived of nutrients. Her lab is trying to apply this knowledge to improve treatment of cancer (to kill cancer cells with metabolic inhibitors) and stroke (to prevent ischemic cell death).

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Necrosis, Typical and Atypical Apoptosis Induced by Glucose Deprivation in Cancer Cells. Role of Autophagy

Glucose depletion has been shown to kill tumor cells either by necrosis (non-apoptotic cell death) or by the mitochondrial pathway of apoptosis. We observed that several tumor cell lines of different origins die in a non-apoptotic manner when deprived of glucose. However, HeLa cells and mouse embryonic fibroblasts (MEF) died by apoptosis. Surprisingly, apoptosis was independent of the mitochondrial pathway, since it occurred in Bax, Bak-deficient MEF and in Bcl-xL-overexpressing HeLa cells. Apoptosis was mediated by caspase-8. However, neither the classical death receptor pathway nor the caspase-activating complex ripoptosome were involved in apoptosis. In response to nutrient shortage or organelle damage, cells undergo macroautophagy. Starvation of glucose, an essential nutrient, is thought to promote autophagy in mammalian cells. We thus aimed to determine the role of autophagy in cell death induced by glucose deprivation. Inhibition of autophagy by chemical or genetic means by using 3-methyladenine, chloroquine, a dominant negative form of ATG4B or silencing Beclin-1, Atg7 or p62 indicated that macroautophagy does not protect cells undergoing necrosis or apoptosis upon glucose deprivation. Moreover, glucose deprivation did not induce autophagic flux in any of the four cell lines analyzed, even though mTOR was inhibited. Indeed, glucose deprivation inhibited basal autophagic flux. In contrast, the glycolytic inhibitor 2-deoxyglucose induced pro-survival autophagy. Further analyses indicated that in the absence of glucose, autophagic flux induced by other stimuli is inhibited. These data suggest that the role of autophagy in response to nutrient starvation in animal cells should be reconsidered.

B·DEBATE IS AN INITIATIVE OF: biocat "la Caixa" Foundation

WITH THE COLLABORATION OF: VIB Agrisera Antibodies for plant sciences