Cell Death and Differentiation (2012) 19, 184

www.nature.com/cdd

Meeting Report

ICDS 2011 meeting 'Signaling in cell death survival, proliferation and degeneration'

RA Lockshin*[,1], SS Smaili[2], S Lavandero[3,4] and Z Zakeri*[,1]

Cell Death and Differentiation (2012) **19,** 184; doi:10.1038/cdd.2011.169; published online 18 November 2011

Symposium of the International Cell Death Society Cheiro de Mato Resort of Maripora, São Paulo, Brazil on 10–13 June 2011

The Symposium of the International Cell Death Society 'Signaling in cell death survival, proliferation and degeneration' was held at the Cheiro de Mato Resort of Maripora, close to São Paulo, Brazil on 10–13 June 2011. This meeting addressed the triggers of cell death, non-apoptotic aspects of cell death, how pathogens affect cell death, and new therapeutic approaches. It attracted attendees from 15 countries. The meeting opened with a tribute to Jürg Tschopp and the ICDS Award lecture by Guido Kroemer, 'Cancer cell death: killing the immortal', which emphasized immunogenic chemotherapy, an argument furthered by a presentation by Laurence Zitvogel. Immunogenic chemotherapy addresses the immunogenic effects of tumor cell death induced by a variety of cytotoxic drugs. Tumor-derived antigen presentation can be mediated by the alarmin HMGB1 (released by dying tumor cells in response to chemo/radiotherapy) and by TLR4 on dendritic cells. TLR4 recognizes tumor-derived antigens, leading to T-cell activation and to the induction of an antitumor immune response. Kroemer noted that most anticancer drugs elicit non-immunogenic apoptosis. Laurence Zitvogel emphasized off-target roles of adenine nucleotides in anticancer treatments. The microenvironment of the cancer cell is affected by galectine 3 (Roger Chammas) and multiple regulators of TRAIL (Gustavo Amarante-Mendes), as well as microRNA-10B (Flavia Maziero Andreghetto). Novel means of regulating cell death in cancer therapy included a natural cytotoxic peptide from snake venom (Mirian Hayashi), immunization together with interferon, homoharingtonine, and histone acetylases (Ruan Medrano, Jarmila Stremenova, and Jo-Ann de la Mare). The role of caveolin-1 is ambiguous; its function depends on the cell context and its relationship with survivin and cyclooxygenase 2 (Andrew Quest).

Many speakers emphasized that cell metabolism is critical for establishing the threshold of cell death. Nika Danial described the integration between glucose metabolism and apoptosis by Bcl-2 family proteins, while Shazib Pervaiz described redox regulation controlling cell death in cancer cells, and Ute Moll noted the role of p53 in oxidative stress in ischemic stroke. Rafael Linden argued that extracellular environment and scaffolding were determinant. Roberto Bravo and Leticia Rodrigues emphasized the role of calcium as a messenger between ER, lysosomes, and other organelles. Francesco Cecconi argued for the role of apoptosome in the onset of Alzheimer's disease, while Vilma Martins emphasized the role of prion protein.

Interaction between autophagy and apoptosis emerged as a major theme, as was emphasized by Christian Seitz, Anna-Mart Engelbrecht, Alicia Melendez, Gian Maria Fimia, and Raymond Birge. Pathogens manipulate cell death primarily by regulating autophagy. Bacteria use autophagy to prevent cell death and viruses can either induce or inhibit autophagy to infect cells. Viruses can effectively use autophagy to protect cells from death. In such a situation, the immune system can overcome tumor and virus strategies to keep cells alive.

Many molecules whose function has been described for apoptosis participate in activities unrelated to apoptosis. Bcl-2 family members can regulate UPR, caspase 7 can control bone formation, and several caspases are activated to influence synaptic growth and function (Claudio Hetz, Eva Matalova, and Francesco Cecconi).

The ICDS 2011 illustrated the strong state of the field in many countries, especially South America, and the value of cultural exchange and collaboration. On a scientific basis, it is noticeable that the fate of cells depends not only on the machinery of cell death, but also, and most importantly, on their current state and history. The immunogenicity of the dying cell influences the outcome in chemotherapy, while the interaction between apoptosis and autophagy and the mechanisms activated for autophagosome formation are important steps. Furthermore, the metabolic state, the source of nutrition, and the communication between cells adjust the threshold of the cell's commitment to die, providing a take-home message for the future.

[1]Department of Biology, Queens College, City University of New York, New York, NY, USA; [2]Department of Pharmacology, School of Medicine, Universidade Federal de São Paulo (EPM-UNIFESP), São Paulo, Brazil; [3]Centro Estudios Moleculares de la Célula, Facultad Ciencias Químicas y Farmaceuticas and Facultad Medicina, Universidad de Chile, Santiago, Chile and [4]University of Texas Southwestern Medical Center, Dallas, TX, USA
*Corresponding author: RA Lockshin or Z Zakeri, Department of Biology, Queens College, City University of New York, New York, NY, USA. Tel: 71 89 97 3417; Fax: 71 89 97 3429; E-mail: rlockshin@gmail.com (RAL) or E-mail: zahra_zakeri@hotmail.com (ZZ)