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Meeting Report

ICDS meeting in Shanghai

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The 7th International Cell Death Society Symposium, 'Targeting Cell Death Pathways for Human Diseases', was held in June 2008 in Shanghai, China. This is a summary of the meeting.

The International Cell Death Society held its biannual meeting, 'Targeting Cell Death Pathways for Human Diseases,' in Shanghai Mega City, China, from 6 to 9 June 2008. The meeting provided an opportunity for scientists from East and West to meet and learn how each approached theoretical and practical problems in adapting our knowledge of cell death mechanisms toward therapeutic goals. It was organized by Zahra Zakeri, Junying Yuan, and Jiarui Wu.

The program began with awards to H Robert Horvitz for his major contributions to the field and to Zahra Zakeri, President of the International Cell Death Society, as Ambassador for Science, honoring her efforts to bring together scientists from around the world. Dr Horvitz then traced, in a keynote talk, how the clarity of genetics in *Caenorhabditis* has led to an understanding of the several cell death pathways in mammals, and suggestions as to what may be found in the future. This talk was followed by presentations from Xiaolin Zhang (AstraZeneca), En Li (Novartis), and Li Chen (Roche), who described the advantages and disadvantages of pharmaceutical research in China, and how their companies are exploring new targets in signaling, cell targeting, and specific uptake of drugs, as they explore means of controlling specific cancers, virus-generated diseases, diabetes, and circulatory diseases.

Further sessions addressed the different modes of cell death, including autophagy (with considerable discussion of micro- and macroautophagy and the molecules that control them), controlled necrosis, the interaction and feedback among the pathways, and the control of autophagy in cell death; mechanisms of signaling that target cells for death, especially in death receptor-initiated apoptosis; means by which natural killer cells identify and attack their targets; and regulation of cell death activated through metabolic controls. In these latter sessions, many relevant parameters were identified, including chaperones, lipids such as erucylphosphohomocholine, components of oxidative and glycolytic pathways, calcium and its regulators, mechanisms of balancing Bax and Bak, the importance of cell substrata and communication from extracellular matrix to nucleus, prions, and biological and artificial regulators of caspases. Mechanisms of phagocytosis and the implications of perturbation of phagocytosis on autoimmune disease, malignancy, propagation of viruses, and other diseases were the subjects of other sessions.

Prospects for therapy included manipulating p53 and its family members, introduction of antisense oligonucleotides, consideration of the substantial role of redox state, oxidized and reduced dinucleotides, and availability of substrates for oxidative and glycolytic pathways. Overall, there are promising prospects for individual targeting of cells through surface receptors, oligonucleotide delivery, delivery through pinocytosis or microphagocytosis, specific monoclonal antibodies, partitioning molecules according to respiratory state or exploiting differences in metabolism between targeted cells and healthy cells, and rendering cells more or less susceptible to immune attack. Guido Kroemer, in a wrap-up talk, emphasized the importance of tailoring therapies to work cooperatively with natural immune mechanisms, which recognize and respond to 'danger' and 'eat-me' signals toward therapeutic goals to protect or destroy specific cells.

This brief but packed meeting drew a balanced mixture of invited speakers from 13 countries, including junior- and senior-level speakers, and an equivalently broad selection of posters. It provided a culturally and scientifically enriching experience for all.

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