

Keystone Symposia: Molecular and Cellular Biology of Immune Escape in Cancer

(Joint with meeting on: Role of Inflammation in Oncogenesis)

Sponsored by Genentech BioOncology

February 7–12, 2010 • Keystone Resort • Keystone, Colorado • USA

Scientific Organizers: George C. Prendergast and Dmitry I. Gabrilovich

JOINT PROGRAM FACULTY & TALKS

Frances R. Balkwill[♦], Queen Mary University of London, Barts and The London Medical School, UK

Inflammatory Cytokines and Autocrine Tumor-Promoting Networks

Thomas Blankenstein, Max Delbrück Center for Molecular Medicine, Germany
No Escape

Vincenzo Bronte, Istituto Oncologico Veneto, Italy
Mechanism of the Function of Myeloid-Derived Suppressor Cells

Lieping Chen, Johns Hopkins School of Medicine, USA
B7 Family of Molecules and Immune Suppression in Cancer

Mario P. Colombo, Fondazione IRCCS Istituto Nazionale Tumori, Italy
OX40 Costimulation in Treg Functions

Lisa M. Coussens[◇], University of California, San Francisco, USA
Role of Inflammation in Tumor Progression

Gerard I. Evan[◇], University of California, San Francisco, USA
Immune Function and Angiogenesis in Myc-mediated Tumor Formation

Richard A. Flavell, Yale University School of Medicine, USA
TGF-beta Signaling in Tumoral Immune Suppression

Dmitry I. Gabrilovich, University of South Florida, USA
Regulation of Myeloid-Derived Suppressor Cells in Cancer

Thomas F. Gajewski[◇], University of Chicago, USA
T Cell Defects in the Cancer Context

Elizabeth M. Jaffee, Johns Hopkins University, USA
New Clinical Approaches to Reducing Immune Escape in Cancer

Michael Karin[◇], University of California, San Diego, USA
NF-kappaB in Inflammation, Progression and Immune Response

Hyam I. Levitsky, Johns Hopkins School of Medicine, USA
Mechanism of T-Cell Tolerance in Cancer

Claire E. Lewis[◇], University of Sheffield Medical School, UK
Macrophages as Key Mediators of Tumor Angiogenesis: Responses to Hypoxia

David H. Munn, Medical College of Georgia, USA
Indoleamine 2,3-Dioxygenase in Tumor Immune Escape

Randolph J. Noelle, Dartmouth Medical School, USA
Immune Suppressive Mast Cells in Cancer

Augusto C. Ochoa, Louisiana State University Health Sciences Center, USA
Arginine Metabolism and Tumor Escape

Suzanne Ostrand-Rosenberg, University of Maryland, USA
Inflammation, Macrophages and Cancer

Drew M. Pardoll, Johns Hopkins School of Medicine, USA
Mechanism of T-Cell Tolerance in Cancer Mediated by Inhibitory Receptors

George C. Prendergast, Lankenau Institute for Medical Research, USA
Regulation and Therapeutic Inhibition of IDO/IDO2 in Cancer

Gabriel Rabinovich, Instituto de Biología y Medicina Experimental, Argentina
Turning Sweet on Immunity: Galectin-glycan Interactions in T-cell and Dendritic Cell Physiology

Licia Rivoltini, Fondazione IRCCS Istituto Nazionale Tumori, Italy
Tumor-Derived Microvesicles and Immune Defects in Human Cancer

Hans Schreiber[◇], University Of Chicago, USA
Role of Tumor Stroma in Immune Escape in Cancer

Robert D. Schreiber[♦], Washington University School of Medicine, USA
Co-Evolution of Immunoediting on Oncogenesis during Tumor Formation

Michael R. Shurin, University of Pittsburgh, USA
Dendritic Cells in Immune Escape in Cancer

Harald von Boehmer, Harvard Medical School, USA
Antigen-Specific Regulatory T Cells

Erwin F. Wagner[◇], Spanish National Cancer Research Centre, Spain
AP-1(Fos/Jun) in Inflammatory Disease and Cancer

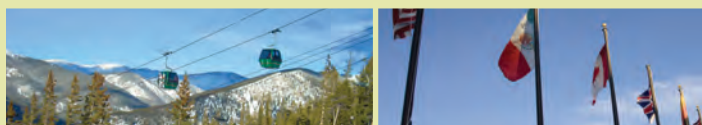
Rong-Fu Wang, Baylor College of Medicine, USA
Immunosuppressive gamma-delta T Cells

Jeffrey Weber, H. Lee Moffitt Cancer Center, USA
Regulation of Immune Responses in Cancer via Checkpoint Blockade of Negative Receptors in Melanoma

Jedd D. Wolchok, Memorial Sloan-Kettering Cancer Center, USA
Therapeutic Regulation of Immune Responses in Cancer Patients Using CTLA-4 Antibodies

Hua E. Yu[◇], Beckman Research Institute, City of Hope, USA
The Role of STAT3 in Tumor Progression and Immune Defects in Cancer

[♦]Keynote speaker. [◇]Speaker in a joint session. Programs subject to change.
Current as of September 21, 2009



It is now widely recognized that the immune microenvironment of a tumor provides critical support in determining its progression versus dormancy or destruction. To gain immune benefits the tumor must evolve mechanisms of immune escape. Study of this crucial process requires cross-fertilization between molecular cell biologists and tumor immunologists who do not tend to interact. This problem is also integrated with the problem of cancer inflammation that has captured the attention of tumor biologists focused on transgenic mouse models and clinical settings. This conference – the first of its kind – should integrate perspectives from a diverse set of researchers in cancer, immunology and molecular therapeutics, focusing specifically on immune escape and tumor-induced immune suppression as a multidisciplinary problem. Cancer is initiated by the accumulation of genetic and epigenetic changes in a normal cell, but its progression depends on the stromal and immune microenvironment of the initiated cell. While intrinsic signals within the initiated cell drive neoplastic transformation and genomic plasticity, extrinsic signals delivered by immune cells are critical in dictating whether progression versus dormancy or destruction of an initiated lesion takes place, and also whether metastasis may occur. The high degree of genomic plasticity in cancer cells permits them to develop sophisticated ways to prevent the immune system from recognizing and eliminating tumor cells. It has become increasingly clear in recent years that abnormalities in the immune system that are induced by tumors not only hamper natural anti-tumor immune surveillance but also limit the efficacy of immunotherapy and even traditional chemotherapy and radiotherapy. Thus, tumor-induced immune abnormalities may not only impact the clinical course of disease but also the prospects for its therapeutic management. Recent results indicate that we are on the verge of a real breakthrough in our understanding of how tumors thwart the immune system and how correcting immune escape could vastly improve cancer therapy.

PROGRAM PLENARY SESSIONS & WORKSHOPS:

- Tumor Microenvironment and Immune Escape (Joint)
- Workshop 1: Regulation of Immune Escape by Microenvironments
- Antigen-Specific Mechanisms of Tumor Escape
- Myeloid Cells in Immune Suppression & Tumor Progression
- Regulatory T & NKT Cells in Immune Escape
- Transcriptional Regulation of Immune Escape (Joint)
- Workshop 2: Tumor Recruitment of Immune Cells: Roles in Tumor Progression & Immune Escape
- Immune Inhibitory Receptors in Cancer
- Metabolic Pathways Involved in Immune Escape
- Workshop 3: Novel Strategies to Reverse Immune Escape in Cancer
- Therapeutic Development to Correct Immune Escape in Cancer

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