

Register Early  
and Save!

**5th Annual International Conference**

# Anti-angiogenesis

November 12-13, 2007 • Courtyard Boston Tremont Hotel • Boston, MA

*Anti-vascular agents remain an exciting opportunity for clinical development and this conference highlights both novel industry clinical candidates and new directions from keynote academic speakers*

## Keynote Presentations

### Anti-angiogenic Therapy in Combination with Radiotherapy: From Bench to Bedside and Back

Kevin Camphausen, M.D., National Cancer Institute

### The Role of Circulating Endothelial and Progenitor Cells in Cancer

Dan G. Duda, DMD, Ph.D.,  
Massachusetts General Hospital

### The Anti-angiogenesis Activity of Axon Guidance Mediators

Michael Klagsbrun, Ph.D.,  
Harvard Medical School/Children's Hospital, Boston

### Drug Delivery Systems for Angiogenesis Inhibitors and Other Biomolecules

Robert S. Langer, Sc.D.,  
Massachusetts Institute of Technology

### Mining the Urinary Proteome: Cancer Biomarker Discovery

Marsha A. Moses, Ph.D.,  
Harvard Medical School/Children's Hospital, Boston

### Selective Inhibition of Pathologic Angiogenesis in Artherosclerosis

Karen S. Moulton, M.D.,  
Harvard Medical School/Children's Hospital, Boston

### Integration of Anti-angiogenic Therapy with Radiation Therapy and other Modalities

Michael S. O'Reilly, M.D.,  
The University of Texas M. D. Anderson Cancer Center

### Integrin $\alpha 4\beta 1$ Activation During Bone Marrow Derived Cell Trafficking Promotes Tumor Neovascularization and Growth

Michael C. Schmid, Ph.D.,  
University of California San Diego

## Media Partners



Cancer Drug News



The American Journal of  
**PATHOLOGY**

TheScientist  
MAGAZINE OF THE LIFE SCIENCES



# 5th Annual International Conference Anti-angiogenesis

The 5th Anti-angiogenesis conference is aimed to update attendees on new drugs and to better describe what makes an efficacious anti-angiogenic drug. The conference will also cover how anti-angiogenic drugs fit into the realm of anti-vascular modalities, understand why and what it takes to improve anti-angiogenic drug activity through combination strategies and appropriate drug delivery, and to learn how to follow activity of anti-angiogenic drugs through measurement and validity of biomarkers.

The field of angiogenesis is nearly forty years old now yet only a handful of drugs have been approved targeting angiogenesis. Such approved drugs are the multi-kinase inhibitors Sutent (sunitinib) approved in January of 2006 and Nexavar (sorafenib) approved in December of 2005, and the only "blockbuster" Avastin approved in

February of 2004 being an anti-VEGF antibody. Clearly, targeting the vasculature with anti-angiogenesis drugs does not appear to be an easy task. Anti-angiogenic drugs, via their novel mechanisms, offer an opportunity to be used in combination with conventional drugs without significant toxicity. Hence understanding how to work with this new class drugs will enable clinicians and drug developers to better serve the public to help to increase not just the life but the quality of life of the cancer patient.

Drug companies, academic groups and contract research industries will benefit from this conference by meeting key industries and opinion leaders in an opportune environment for networking, establishing meaningful relationships and learning from the presenters in a workable attendee group number.

## Main Conference

### Monday, November 12, 2007

#### 8:30 Chairpersons' Remarks

David Sherris, Ph.D., *President and CEO, Paloma Pharmaceuticals, Inc.*  
Marsha A. Moses, Ph.D., *Professor, Surgery, Harvard Medical School/ Children's Hospital, Boston*  
Dian Olson, B.Sc., *Scientist, Discovery Oncology, Biogen Idec*

#### KEYNOTE PRESENTATION

#### 8:45 Drug Delivery Systems for Angiogenesis Inhibitors and Other Biomolecules

Advances in drug delivery are creating new opportunities for cancer therapy. Our early research enabled the development of principles for controlled release of peptides and proteins, paving the way for the isolation of angiogenesis stimulators and inhibitors and new treatments for advanced prostate cancer. We have also created new degradable materials that have led to new treatments for brain cancer. Drug delivery technologies including novel polymers and intelligent microchips promise to create new treatments for cancer and other illnesses. New aerosol designs as well new nanoparticle delivery systems for nucleic acids will also be discussed.

Robert S. Langer, Sc.D., *Institute Professor, Chemical Engineering, Massachusetts Institute of Technology*

#### ANTIBODY THERAPEUTICS

#### 9:30 Targeting Tie-1 Sensitizes Tumors to Angiogenesis Inhibitors

Tie-1 RTK plays critical role in vascular development. DX-2240 is a human IgG1 binding Tie-1 with high affinity, inhibiting endothelial cell tube formation. DX-2240 retards tumor progression in a variety of cancer xenograft models. Increased anti-tumor activity was shown with bevacizumab and DX-2240 in a pancreatic tumor xenograft model and enhanced in a sequential dosing regimen. DX-2240 pretreatment followed by bevacizumab resulted in decreased in tumor growth and delay in reaching tumor target size, similarly with sequential dosing of sorafenib with DX-2240. Combining angiogenesis inhibitors sequentially has potential of increasing inhibition of tumor growth and decreasing frequency of tumor resistance..

Daniel T. Dransfield, Ph.D., *Senior Director, Cell Biology, Dyax*

10:00 *Networking Break*

#### KEYNOTE PRESENTATION

#### 10:30 The Role of Circulating Endothelial and Progenitor Cells in Cancer

It remains unclear what extent bone marrow-derived cells incorporate perfused tumor vessels. The utility as biomarkers of circulating endothelial cells and progenitor cells during cancer therapy with anti-angiogenic agents are unclear. We developed models characterizing local vessel-derived and bone marrow-derived endothelial cells (BMD-ECs) based on a set of endothelial markers and morphology. Their contribution to perfused blood vessels in tumors using transplanted and spontaneous primary and metastatic tumor models was qualified, and, the origin, viability and phenotype of cells expressing endothelial markers circulating in the blood were evaluated. We evaluated the circulation kinetics of these cells as a biomarker of efficacy anti-VEGF agents in clinical trials. We demonstrated BMD-ECs incorporate in perfused tumor vessels which contribution varies with organ site and mouse strain. VEGF blockade in cancer patients decreases number of circulating endothelial cells and progenitor cells. Kinetics of circulating endothelial cells and progenitor cells may have predictive value in patients receiving anti-VEGF therapy.

Dan G. Duda, D.M.D., Ph.D., *Assistant Professor, Massachusetts General Hospital/Harvard Medical School*

#### 11:15 Role of Stromal & Vascular Cells & Related Cytokines in Therapeutic Resistance

Non-malignant cells involved in malignant disease are targets for anticancer therapy. As normal cells including vascular components and stromal cells are co-opted by malignant disease, they de-differentiate to proliferative capacity. Stem and progenitor cells are mobilized by malignant disease becoming involved in vascular growth and stromal development. Mesenchymal stem or stromal cells can contribute to progression of malignant disease. Scientists are determining whether targets from tumor subpopulation analysis can be used therapeutically. Endosialin was identified as a tumor endothelial marker by SAGE and is expressed by endothelial precursor cells, pericytes, mesenchymal stem cells, a subset of T-cells and mesenchymal malignant cells. Endosialin is a Group XIV thrombomodulin C-lectin domain protein. Its absence of expression in mature endothelium differentiates it from thrombomodulin. Although its function is unknown, its expression pattern may make it a favorable target for cancer therapy.

Beverly A. Teicher, Ph.D., *Vice President, Oncology Research, Genzyme Corporation*

## 11:45 Development of 2C3 as a Clinical Candidate for Inhibition of VEGF Activation of VEGF Receptor 2

Selective inhibition of VEGF receptor (VEGFR) 2 by mouse monoclonal anti-VEGF antibody 2C3, reduces tumor growth, augments the anti-tumor activity of chemotherapy, and alters immune cell infiltration of solid tumors. A fully human version of 2C3 has been developed, termed R3. Its effects on VEGF activation of VEGFR1 and VEGFR2, and its ability to inhibit tumor growth in vivo will be discussed.

**Rolf A. Brekken, Ph.D.**, *Peregrine Pharmaceuticals Collaborator, Assistant Professor, Surgery and Pharmacology, University of Texas Southwestern Medical Center*

12:15 *Lunch on Your Own*

### KEYNOTE PRESENTATION

#### 1:45 Anti-angiogenic Therapy in Combination with Radiotherapy: From Bench to Bedside and Back

Radiotherapy (RT) plays a key role in treatment of cancer. Future improvements in tumor control will likely involve combination of RT with targeted therapies. Preclinical evaluations of combined anti-angiogenic therapy with RT have yielded promising results with increased tumor "cure." Whether the improvement in tumor control in the laboratory will translate into improved outcomes in the clinic remains to be seen. Multiple differences between these agents and cytotoxic chemotherapy must be taken into account when designing clinical trials evaluating their effectiveness in combination with RT. I will discuss important considerations for designing clinical trials of angiogenesis inhibitors with RT.

**Kevin Camphausen, M.D.**, *Head, Imaging and Molecular Therapeutics, and Chief, Imaging and Molecular Therapeutics Section, National Cancer Institute*

#### 2:30 Selective Antibody-based Targeting of MMP-14 Inhibits Tumor Growth and Angiogenesis

Membrane bound metalloproteinase MMP-14 was proposed to play a role in tumor growth/metastasis by degrading ECM components and activating proteinases. Combining human antibody phage display with automated selection and screening strategies, DX-2400 was isolated, a potent and selective antibody inhibitor of MMP-14. DX-2400 inhibits HUVEC tube formation and retards tumor progression/metastasis in murine tumor models. Immunohistochemical analysis reveals a decrease in Ki67-positive tumor cells and CD31-positive tumor microvessels in DX-2400-treated animals, demonstrating anti-tumoral and anti-angiogenic activity. Administration of DX-2400 decreases total tumor MMP activity confirming MMP-14 in activating MMP activity and validated in vivo MMP-14 in tumor growth and angiogenesis.

**Laetitia Devy, Ph.D.**, *Principal Scientist, Dyax*

#### 3:00 Targeting the Urokinase Plasminogen Activator Receptor (uPAR) as a Therapeutic Strategy for the Inhibition of Angiogenesis and Tumor Growth

uPAR is involved in activities required for tumor angiogenesis/metastasis, including ECM remodeling, cell signaling, migration, and growth. uPAR does not contain a transmembrane domain, associating with integrins, matrix components, GPCRs, and transmembrane receptors. Immunization and screening was used to raise anti-uPAR monoclonal antibodies with distinct functions. One, ATN-658, inhibits endothelial progenitor cell proliferation in vitro and tumor proliferation, migration, and invasion in vivo. ATN-658 has been humanized with IND filing anticipated mid-2008. ATN-658 is expected to be the first uPAR targeted therapy to enter the clinic allowing testing of uPAR as an important target in tumor angiogenesis, growth, and progression.

**Andrew P. Mazar, Ph. D.**, *Chief Scientific Officer and Senior Vice President, Research and Development, Attenuon, L.L.C.*

3:30 *Networking Break and Poster/Exhibit Viewing*

### KEYNOTE PRESENTATION

#### 4:00 Mining the Urinary Proteome: Cancer Biomarker Discovery

Within the context of our laboratory's Urinary Proteomics Initiative, we have identified and validated a number of novel urinary cancer biomarkers some of which are regulators of angiogenesis. These non-invasive biomarkers not only sensitively and specifically predict disease status and stage in patients with a variety of human cancers, but can also be used to monitor therapeutic efficacy and cancer progression, with some serving as risk assessment tools as well. The discovery and validation of some of these biomarkers, as well as their potential as cancer diagnostics and prognostics, will be discussed.

**Marsha A. Moses, Ph.D.**, *Professor, Surgery, Harvard Medical School/Children's Hospital, Boston*

### SIGNAL TRANSDUCTION

#### 4:45 Palomid 529, a Broad Spectrum Signal Transduction Inhibitor Affecting the Akt/mTor Pathway

Paloma Pharmaceuticals, through its proprietary library of small molecule drugs, is developing a dual acting anti-angiogenic and anti-tumor agent, Palomid 529. Palomid 529 is the result of three generations of de novo design, synthetic chemistry and efficacy models. Work will be described to highlight the activity of Palomid 529 as an Akt/mTor inhibitor and as a potent anti-cancer agent in both in vitro and in vivo models.

**David Sherris, Ph.D.**, *President and CEO, Paloma Pharmaceuticals, Inc.*

5:15 *End of Day One*

## Tuesday November 13, 2007

#### 8:30 Chairpersons' Remarks

**David Sherris, Ph.D.**, *President and CEO, Paloma Pharmaceuticals, Inc.*  
**Marsha A. Moses, Ph.D.**, *Professor, Surgery, Harvard Medical School/Children's Hospital, Boston*  
**Dian Olson, B.Sc.**, *Scientist, Discovery Oncology, Biogen Idec*

### KEYNOTE PRESENTATION

#### 8:45 Integration of Anti-angiogenic Therapy with Radiation Therapy and other Modalities

Although anti-angiogenic agents offer therapeutic potential, studies suggest these agents will have a delayed onset of activity and may only induce disease stabilization. Use of radiation therapy for cancer is associated with therapeutic challenges that are distinct from those that might be expected with anti-angiogenic agents. Multimodality therapy that includes anti-angiogenesis offers potential for improved efficacy and diminished toxicity in treatment of cancer and other angiogenesis dependent diseases. Additional work remains to provide a rational basis for combination of angiogenesis inhibitors with other modalities so these agents can be successfully incorporated into existing therapy in a timely and rational manner.

**Michael S. O'Reilly, M.D.**, *Assistant Professor, Cancer Biology and Radiation Oncology, The University of Texas M. D. Anderson Cancer Center*

#### 9:30 EZN-2968 is a Potent and Specific Locked Nucleic Acid Oligonucleotide Against Hypoxia Inducible factor-1 $\alpha$ (HIF-1 $\alpha$ ) In Vitro and In Vivo

EZN-2968 is a locked nucleic acid antisense oligonucleotide inhibiting expression of HIF-1 $\alpha$  having higher affinity, higher specificity, and higher resistance against degradation. In human prostate and glioblastoma cells, EZN-2968 induced potent, selective, and durable antagonism of HIF-1 $\alpha$  expression, both under normoxic and hypoxic conditions. Down-regulation of HIF-1 $\alpha$  by EZN-2968 reduces transcriptional targets VEGF and matrix metalloproteinase 2 and significant inhibits HUVEC tube formation. In normal mice it induces dose-dependent down-regulation of endogenous HIF-1 $\alpha$  in liver and kidney. Anti-tumor effects in murine xenografts include reduction of tumor weight and cell viability, and reduced tumor infiltration by host cells when EZN-2968 was transfected into DU145 prostate cells before implantation of the tumor combined with systemic treatment with EZN-2968 post-tumor implantation. EZN-2968 is a potent, selective inhibitor of HIF-1 $\alpha$  warranting clinical investigation.

**Ivan D Horak, M.D., FACP**, *Executive Vice President and Chief Scientific Officer, Enzon Pharmaceuticals, Inc.*

10:00 *Networking Break and Poster/Exhibit Viewing*

### KEYNOTE PRESENTATION

#### 10:30 The Anti-angiogenesis Activity of Axon Guidance Mediators

Proteins mediating axon guidance also mediate angiogenesis as in class-3 semaphorins (SEMA3) and their receptors the neuropilins (NRP) which bind VEGF165. SEMA3A repels axons and collapse growth cones, and inhibit endothelial cell migration and capillary sprouting. SEMA3F is a potent inhibitor of tumor angiogenesis/metastasis. SEMA3A and SEMA3F competitively inhibit VEGF binding to NRPs repelling endothelial cells and collapsing endothelial cell cytoskeleton, mechanisms similar to axon guidance. Generation of mutations in soluble NRPs having high VEGF165 affinity blocking VEGF binding to endothelial cells may be a promising anti-angiogenic strategy. Axon guidance mediators may be an excellent source of anti-angiogenesis factors.

**Michael Klagsbrun, Ph.D.**, *Patricia A. Donahoe Professor of Surgery, Harvard Medical School/Children's Hospital, Boston*

## 11:15 Targeting Tumor Angiogenesis with HDAC Inhibitors: NVP-LAQ824

Histone deacetylases inhibitors (HDACs) have been shown to exert their antitumor effect by gene and protein expression modulation. Recent data suggest that inhibitors of class II HDACs (i.e. NVP-LAQ824) are strong inducers of HIF-1 $\alpha$  protein degradation both in cancer and endothelial cells, and reduce down-stream angiogenesis-related gene expression. HDACs represent a potential therapeutic tool to improve anti-angiogenesis treatments and to overcome anti-VEGF therapy resistance.

**Roberto Pili, M.D.**, *Novartis Collaborator and Assistant Professor, Oncology and Urology, Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins*

## 11:45 Technology Workshop

For more information about technology workshop opportunities, please contact Kristen Schott at 508-614-1239 or kschott@ibcusa.com.

## 12:15 Networking Lunch and Poster/Exhibit Viewing

### KEYNOTE PRESENTATION

#### 1:30 Integrin $\alpha 4 \beta 1$ Activation During Bone Marrow Derived Cell Trafficking Promotes Tumor Neovascularization and Growth

Bone marrow derived cells utilize integrin  $\alpha 4 \beta 1$  to adhere tumor endothelium and traverse tumor blood vessels. Cytokines activate integrin  $\alpha 4 \beta 1$  on bone marrow derived cells and promote adhesion of myeloid cells to endothelium. Inflammatory cytokines including IL-1 $\beta$ , SDF-1, and IL-6 stimulated myeloid cell adhesion to purified rsVCAM or endothelial cells that could be only blocked by antagonists of integrin  $\alpha 4 \beta 1$ . These cytokines induced  $\alpha 4 \beta 1$  conformational changes and clustering with paxillin. Mice transplanted with bone marrow from animals with inactivated integrin  $\alpha 4$  exhibited reduced tumor growth and decreased blood vessel density associated with reduced trafficking of myeloid cells to tumors. IL-1 $\beta$  and IL-6 expressed by inflammatory cytokines stimulate myeloid cell recruitment to tumor microenvironment by promoting integrin  $\alpha 4 \beta 1$  activation. Loss of integrin  $\alpha 4 \beta 1$  activation on bone marrow derived cells resulted in reduced tumor burden. Suppression of myeloid cell integrin  $\alpha 4 \beta 1$  function can be an effective means to suppress tumor growth.

**Michael C. Schmid, Ph.D.**, *Post-Doctoral Fellow, Moores UCSD Cancer Center, University of California San Diego*

### INTEGRINS

#### 2:15 Preclinical Validation of $\alpha 5 \beta 1$ as a Therapeutic Target

Integrin  $\alpha 5 \beta 1$  plays a critical role during angiogenesis. Its antagonist, volociximab (M200), inhibits endothelial cell growth and movement, independent of the growth factor milieu, and inhibits tumor growth in vivo. Volociximab, does not cross-react with mouse  $\alpha 5 \beta 1$ , precluding its use in mouse xenograft models. We have therefore generated a rat-anti-mouse antibody, 339.1, encompassing many of the properties of volociximab. 339.1 inhibits growth of established tumors in multiple xenograft models, effects associated with a decrease in blood vessel density. Results suggest 339.1, like volociximab, exhibits potent anti- $\alpha 5 \beta 1$  activity, confirming inhibition of integrin  $\alpha 5 \beta 1$  impedes angiogenesis slowing tumor growth.

**Vinay Bhaskar, Ph.D.**, *Senior Scientist, Translational Medicine, PDL Biopharma, Inc.*

#### 2:45 E7820 is a Unique Angiogenesis Inhibitor that Suppresses Expression of $\alpha 2$ Integrin

Please visit [www.ibclifesciences.com/antiangio](http://www.ibclifesciences.com/antiangio) for abstract.

**Daniel Rossignol, Ph.D.**, *Associate Director, Clinical Research, Eisai Medical Research Inc.*

## 3:15 Networking Break and Poster/Exhibit Viewing

### VASCULAR DISRUPTING AGENTS

#### 3:45 Discovery of a Novel Dual Activity Vascular Disrupting Agent

STA-9584 is a promising new vascular disrupting agent that blocks blood flow by specifically disrupting tumor microvasculature at both the center and periphery of tumors. Most vascular disrupting agents generate tumor-cell necrosis at the center of the tumor, leaving a small viable rim of tumor cells to quickly grow back, severely limiting single-agent efficacy. This new compound circumvents the requirement for combination therapy while maintaining an acceptable therapeutic window.

**Christopher P. Borella, Ph.D.**, *Program Manager, Synta Pharmaceuticals*

## 4:15 Anti-tumor and Anti-angiogenic Activity of LP-261, a Novel Oral Tubulin Binding Agent

LP-261 is a small molecule novel tubulin anticancer agent created using Locus' proprietary computational drug design which has advanced into clinical development. LP-261 is predicted to bind tubulin spanning colchicine and nucleotide binding sites, inducing G2/M cellular arrest in target tissues. It has demonstrated broad, potent anti-tumor activity including resistant cell lines, inhibition of endothelial cell sprouting, proliferation and migration. LP-261 shows oral availability in multiple xenografts and in an ocular angiogenesis model. Low oral doses demonstrated significant activity when combined with Bevacizumab in a colon xenograft model. Clinical development scenarios include cytotoxic single agent or low dose anti-angiogenic combination.

**William R. Moore, Jr., Ph.D.**, *Chief Scientific Officer, Locus Pharmaceuticals, Inc.*

### KEYNOTE PRESENTATION

#### 4:45 Selective Inhibition of Pathological Angiogenesis in Atherosclerosis

Cardiovascular diagnostic tests detect atherosclerotic lesions based on size and degree of stenosis limiting blood flow; however, most heart attacks and strokes arise from acute rupture of vulnerable plaques, which originally caused minimal arterial narrowing. Advanced atherosclerotic plaques develop neovascularization that invades the innermost arterial layer increasing delivery of inflammatory cells and plasma constituents via these thin-walled small vascular channels. Bleeding and hyperpermeability of intimal neovascularization may promote progression of vulnerable plaques, therefore, biomarkers for angiogenesis-associated activities in atherosclerotic plaques may be useful clinical tools monitoring persistent risk for acute complications despite control of hypercholesterolemia and other cardiovascular risk factors.

**Karen S. Moulton, M.D.**, *Assistant Professor, Surgery, Harvard Medical School/Children's Hospital, Boston*

## 5:30 End of Conference

### Call for Poster Submissions

The organizers of the Anti-angiogenesis conference recognize the significant education value in the poster presentations. Any registered conference attendee may sign up to present a poster. The deadline to submit your abstract to be included in the Conference Handbook is October 22, 2007. Please submit your abstract on line at [www.IBCLifeSciences.com/antiangio](http://www.IBCLifeSciences.com/antiangio) (see the registration page for details on the poster fee). Poster abstracts and registrations received after October 22, 2007 will be subject to availability for an onsite poster board and will not be included in the Conference Handbook. Full payment for the conference registration and poster board must be received by October 22, 2007 to be included in the Conference Handbook and poster board assignments to be made. The size of the poster board is 4'h X 8'w. Please note posters may not be used as exhibit displays or for marketing purposes and all poster submission are subject to approval by the conference organizers. Only one poster abstract will be accepted per registered attendee/author. No more than two posters will be accepted per company/organization.

### Extend your Market Reach by Sponsoring and Exhibiting

You won't want to miss this opportunity to sponsor and/or exhibit at the Anti-angiogenesis Conference. Reach out to your potential customers and partners by taking an active role in this event!

Sponsorship and Exhibiting Opportunities Include:  
**Technology Workshops, Receptions, Luncheons, Breaks, Hospitality Suites and Booths.**

Sponsorship opportunities are limited so act now.

For more information on sponsorship and exhibitions, please contact Kristen Schott, at [kschott@ibcusa.com](mailto:kschott@ibcusa.com) or 508-614-1239

5th Annual International Conference  
**Anti-angiogenesis**

November 12-13, 2007 • Courtyard Boston Tremont Hotel • Boston, MA

*The only annual conference in the field of Anti-angiogenesis for industry scientists, executives and academic scholars to learn the latest on:*

- Drug delivery of angiogenesis agents
- Advances in antibody therapies
- Integrin mediated antagonists
- Combination modalities for the clinic
- Angiogenesis biomarkers
- Small molecule drug candidates featuring anti-angiogenic and vascular disrupting agents and dual acting anti-tumor and anti-angiogenic agents

**REGISTRATION FORM**

**Anti-angiogenesis**

Product Code: D4204PDF

Name \_\_\_\_\_ Last Name \_\_\_\_\_

Title \_\_\_\_\_ E-mail \_\_\_\_\_

Company \_\_\_\_\_ Department \_\_\_\_\_

Approving Manager \_\_\_\_\_

Street Address \_\_\_\_\_ City \_\_\_\_\_ State \_\_\_\_\_ Postal Code \_\_\_\_\_ Country \_\_\_\_\_

Telephone \_\_\_\_\_ Fax \_\_\_\_\_

**DATA PROTECTION:** The personal information shown on this brochure, and/or provided by you, will be held on a database and may be shared with companies in the Informa Group. Sometimes your details may be obtained from, or made available to, external companies for marketing purposes. If you do not wish for your details to be used for this purpose, please email data-admin@ibcusa.com

**How to Register**

**Online:** [www.IBCLifeSciences.com/antiangio](http://www.IBCLifeSciences.com/antiangio)  
**Phone:** (800) 390-4078 • **Fax:** (941) 365-0104  
**E-mail:** [reg@ibcusa.com](mailto:reg@ibcusa.com)  
**Mail:** IBC USA Conferences,  
P.O. Box 414525, Boston, MA 02241-4525

**Venue & Travel Information**

**Courtyard Boston Tremont Hotel,**  
**275 Tremont Street, Boston, MA 02116**  
**Phone (617) 426-1400 or (800) 321-2211**  
**Fax (617) 482-6730**

**TRAVEL RESERVATIONS:** For all air travel arrangements, including international, please call or write IBC's official air travel agency, Commonwealth Travel Advisors, to book your travel via IBC's airline of choice, American Airlines. E-mail: [jdwyer@traveladvisors.com](mailto:jdwyer@traveladvisors.com) or call: USA: 888.703.4286 or 508.366.3660; International: 1.508.366.3660. Please be certain to mention IBC along with the conference title, dates, and conference code D4202 when e-mailing or calling.

**DISCOUNTED HOTEL RESERVATIONS:** Please call hotel directly before **October 21, 2007** to be included in IBC's dedicated room block for this conference. Please be certain to mention IBC along with the conference title and date.

Industry Fees	On or Before August 17, 2007	On or Before September 14, 2007	On or Before October 12, 2007	After October 12, 2007**
<b>Main Conference</b>	<input type="checkbox"/> \$1399	<input type="checkbox"/> \$1499	<input type="checkbox"/> \$1599	<input type="checkbox"/> \$1699

Academic/Government Fees*	On or Before August 17, 2007	On or Before September 14, 2007	On or Before October 12, 2007	After October 12, 2007**
<b>Main Conference</b>	<input type="checkbox"/> \$599	<input type="checkbox"/> \$699	<input type="checkbox"/> \$799	<input type="checkbox"/> \$899
Reserve a Posterboard	Commercial <input type="checkbox"/> \$50		Academic <input type="checkbox"/> FREE	

\*Academic rate is extended to full-time employees of government, universities & university-affiliated hospitals only. **Total:**  
**For security precautions, a photo identification will be required of ALL attendees at check-in.**  
**\*\*For on-site registration, please add \$100 to the total fee.** Prices include lunch, refreshments and speaker documentation. See inside for policies regarding Substitutions, Cancellations and Special Needs.

**Unable to Attend? Purchase the Conference Handbook**  
This comprehensive selection of bound "hot off the press" information will be available four weeks after the conference.  
 I cannot attend. Please send \_\_\_\_\_ Main Conference Handbook(s). Enclosed is my payment for \$399 each, plus shipping and handling (\$25 in the U.S., \$45 outside the U.S.).

**Payment**  
(Required 30 days in advance of the conference. If registering within 30 days, payment is due immediately)  
 Mastercard  Visa  American Express  Check  Wire Transfer  
Please make check(s) (in U.S. funds drawn on a U.S. bank) payable to IBC USA Conferences and attach to the registration form. Confirmation of your booking will be sent. Wire Transfer: Please tell your bank to include the conference code B4178, invoice number, person attending, name and date of the conference in the transfer instructions. Wire transfers and EFT payments: please contact accounts receivable at [AR@ibcusa.com](mailto:AR@ibcusa.com) for banking details.

Card # \_\_\_\_\_ Exp. Date \_\_\_\_\_

Name (as appears on card) \_\_\_\_\_

Signature \_\_\_\_\_

**Registration Information**

**REGISTRATION SUBSTITUTIONS/CANCELLATIONS:** In order to receive a prompt refund, your notice of cancellation must be received in writing (by letter or fax) 10 working days before the conference. We regret cancellations will not be accepted after that date. However, we will be pleased to transfer your registration to another member of your company at any time. If you plan to send someone in your place, please notify us as soon as possible so that materials can be prepared. All cancellations will be subject to a \$395 processing fee. If IBC cancels an event, IBC is not responsible for any airfare, hotel or other costs incurred by registrants. Speakers subject to change without notice.

**SPECIAL NEEDS:** If you have a handicap or have special needs, please let us know in order that we may address your special needs for your attendance at this conference. For security precautions, a photo identification will be required of ALL attendees at check-in.

