



Genes Dogs and Cancer: 5th International Canine Cancer Conference
February 13-15, 2009, Orlando, Florida, USA

Call for Abstracts

The AKC Canine Health Foundation and the Program Committee for the Genes Dogs and Cancer: 5th International Canine Cancer Conference invites abstract submissions from individuals working in the field of canine cancer. The meeting will be held February 13th – 15th, 2009, in the Royal Plaza Hotel in Orlando, Florida, USA. The meeting is open to all scientists, veterinarians, and dog fanciers with a scientific interest in cancer.

Program Committee and Session Chairs

Conference Co-Chairs: Matthew Breen, Cheryl London, Elaine Ostrander

Session Chairs:

Session I: Molecular Determinants of Susceptibility and Pathogenesis: Elaine Ostrander

Session II: Molecular Markers for Diagnosis and Prognosis: Matthew Breen

Session III: Clinical Treatment and Trials: Cheryl London

Abstract Submission: The Scientific Program Committee welcomes abstracts reporting new basic or clinical research findings. Deadline for submission is November 7, 2008. Please adhere to all rules for the submission and format of abstracts. Failure to comply may be cause for rejection. Notices regarding acceptance of abstracts and program assignments will be sent by no later than November 30th, 2008.

Each abstract will be reviewed for scientific merit and content. Accepted abstracts will be assigned to one of three sessions (I: Molecular Determinants of Susceptibility and Pathogenesis; II: Molecular Markers for Diagnosis and Prognosis; III: Clinical Treatment and Trials) by the program committee. Session Chairs will select a limited number of abstracts for platform presentation. Instructions will be provided to researchers whose abstracts are to be presented as posters; approximately five posters will be provided the opportunity to make a 10-15 minute platform presentation. **Please indicate if you would like to submit your abstract for poster consideration only.**

Abstracts submitted by post-docs, residents, interns, or students will be eligible for the Young Investigator Award, which will include a certificate and a monetary award. Please be aware that submission of an abstract does not guarantee acceptance or inclusion in the competition.

All abstracts must be submitted in electronic form and sent by e-mail to: caninecancer2009@ivis.org. Submission deadline is November 7, 2008. Please refer to the submission information below for specific instructions.

Instructions for Abstract Submission

- Abstracts must consist of original material. Data and conclusions presented at the meeting must be based on the same research as the abstract.
- An author's name may appear on more than one abstract (please underline the name of the presenting author). Each author must agree in principle with the information contained within the abstract.
- Abstracts must be written in English and should contain a statement of specific objectives (unless given by the title); a brief statement of methods, if pertinent; a summary of results and a statement of conclusions. Do not use phrases such as "will be discussed" or "will be presented". Please refer to the sample abstract for a layout example.
- The abstract - excluding title, authors and affiliations - may not exceed 450 words, as determined by the character count feature of your word processor.
- The presenting author must certify that the conditions for submission have been satisfied and that all ethical and animal and human welfare considerations have been met. For online submissions, the author must verify that he/she receives confirmation from the IVIS office that the submitted abstract has been received. Confirmation of abstract receipt will be sent by e-mail within three working days.
- Submission of an abstract constitutes an agreement that the presenting author will attend the meeting if the abstract is accepted.
- If an abstract must be withdrawn, contact Erika Werne at the AKC Canine Health Foundation (phone: 919-334-4010, fax: 919-334-4011 or email eaw@akcchf.org). The notice must stipulate the reason(s) for withdrawal and attest that all authors are in agreement that the abstract must be withdrawn.

Preparation of Abstracts

- All abstracts **MUST** be submitted in electronic form by e-mail to caninecancer2009@ivis.org. Please retain a copy of the submitted abstract. In the subject heading of the e-mail message type "caninecancer2009". In the body of the message type the title of your abstract, your name, full mailing address (include zip code), phone, fax (if available) and e-mail address. If your abstract is successfully received, you will receive confirmation by email with an attachment (PDF file) of your abstract for desktop printing and review.
- To submit an abstract, write the abstract using a word-processor and save the document using your last name and first initial to name the file (for example: LASTNAME_I.doc).
- Windows, DOS, or Macintosh files are accepted. Do not send page make-up files (Quark, Pagemaker, Frame, etc.). If your word processor uses double-byte characters (as do many Japanese word processors), save the file as a single-byte or American format file. Prepare your abstract in the simplest possible form. Do **NOT** use the automatic formatting features of your software, such as hyphenation, endnotes, footnotes, headers, footers, etc. Please include

a backup copy of the abstract under a different file name, preferably saved as RTF (rich text format).

- Once received, your abstract will be read and transferred electronically to an Acrobat PDF file format, which will be e-mailed to you within three working days for proofreading.

Format for Abstracts

- Title: Use upper- and lowercase letters, do not center; end title with a period. See sample abstract.
- Authors: Start a new line; use upper- and lowercase letters; use full first names and surnames for all authors; end with a period.
- Affiliations: start a new line; use upper- and lowercase letters; include department name, institution, city, and state/country; end with a period. Use superscript numbers to identify each author with his/her institution. Do not include street addresses or zip or postal codes.
- Abstract: start a new line. The abstract must not exceed 450 words (excluding title, authors, and affiliations) before insertion of special codes, as determined by the character count feature of your word processor. Excess characters will be deleted.
- Abbreviations: Spell out all abbreviations in full at first mention in the text, with abbreviation following in parentheses, e.g. tumor necrosis factor (TNF). Use scientific abbreviations in accord with the usage in the American Journal of Veterinary Research.

Sample Abstract

Induction of Melanoma Cell Apoptosis by Fas Ligand.

Stacie R. Bianco¹, Kenneth Hance², Susan Fosmire¹, Amie Koenig³, Stephen J. Withrow⁴, David Matthiesen⁵, Michelle G. Ritt⁵, David M. Getzy⁶, Richard C. Duke^{2,7,8}, Donald Bellgrau^{2,7,8}, Gary R. Cutter^{1,8}, John, Wojcieszyn⁹, and Jaime F. Modiano.^{1,7,8}

¹Center for Cancer Causation and Prevention, AMC Cancer Research Center, Denver, CO, USA;

²Ceres Pharmaceuticals, Denver, CO, USA;

³Dept. of Small Animal Medicine and Surgery, College of Veterinary Medicine, Texas A&M University, College Station, TX, USA;

⁴Comparative Oncology Unit, Colorado State University, Fort Collins, CO, USA;

⁵Animal Hospital Center, Highlands Ranch, CO, USA;

⁶Idexx Veterinary Services, Broomfield, CO, USA;

⁷Dept. of Immunology, University of Colorado Health Sciences Center, Denver, CO, USA;

⁸University of Colorado Cancer Center, Denver, CO, USA;

⁹IHC Services, Smithville, TX, USA.

The long-term goal of this work is to establish optimal conditions to promote apoptosis of tumors at accessible sites, thereby providing a source of antigen to promote anti-tumor immune responses. For this study, we examined the capability to induce apoptosis of canine melanoma cells in vitro by overexpression of Fas ligand (FasL). FasL was expressed in five canine melanoma cell lines by infection with a replication-deficient adenovirus encoding murine FasL, or by cationic liposome-mediated transfection of a plasmid encoding human FasL. Four of the five cell lines underwent extensive apoptosis. One cell line was resistant to apoptosis, despite detectable levels of FasL expression as determined by its ability to kill Fas-bearing L1210-Fas target cells. Using this resistant cell line, we determined there was a log-linear relationship

between FasL expression and multiplicity of infection (m.o.i.) to >1,000 viral particles per cell. Resistance to Fas-mediated killing could thus be due to loss of Fas expression by the tumor cells, or to inhibition of signaling pathways that operate downstream of Fas. Fas mRNA was expressed by each of the four susceptible melanoma cell lines, but not by the resistant cell line. In addition, downstream signaling events may contribute to the resistant phenotype. It has been reported previously that susceptibility to FasL-mediated apoptosis requires at least one wild type copy of the PTEN tumor suppressor gene. Our results show that the resistant cell line also lacked expression of PTEN mRNA and protein, but so did two of the susceptible cell lines. However, the resistant cells also lacked mRNA for p16, and had markedly reduced levels of p53 and p21. Next, we assessed the safety of FasL administration *in vivo* to tumor-bearing dogs. FasL (600 µg), mixed with 1 mg of cationic liposomes, was administered by direct intratumoral injection to five dogs with oral or facial tumors with high metastatic potential (4 malignant melanoma, 1 osteosarcoma). No adverse events were observed over the course of 7 days, after which dogs were provided standard therapy as indicated for their tumor (surgery or radiation). Three of five tumors showed measurable regression at day 7. Cells isolated from the two tumors showing the most robust responses had detectable Fas expression, whereas those isolated from the tumor showing the weakest response, and those isolated from a tumor with no measurable response had no detectable Fas mRNA. The data suggest that overexpression of FasL is an effective means to induce apoptosis of canine melanoma cells *in vitro*, and that susceptibility may be determined by distinct genetic “fingerprints”. Moreover, intratumoral gene administration appears to provide a safe route for delivery of FasL *in vivo*.