ABSTRACT SUBMISSION GUIDELINES

GENERAL INFORMATION: Please read carefully
- Graduate students, Post-Doctoral Fellows, Residents, Clinicians and Scientists are eligible to participate in the submission of abstracts for presentation. Abstracts from members of the Department of Oncology as well as trainees in either CaRTT or the TBCRU will be given priority over all others
- PLEASE REMEMBER to compose your abstract so that it is understandable yet still data-rich.

ABSTRACT GUIDELINES:
- Abstract must be single-spaced.
- Title Text Box: Title in sentence form.
- Abstract First Line: Author list, using format shown in example below
- Second Line: List of author affiliations
- Third Line: Blank
- Fourth Line: Begin Abstract

NOTE THAT ALL ABSTRACTS NOT FORMATTED ACCORDING TO THE GUIDELINES ABOVE WILL BE RETURNED FOR REVISION.

Interaction of the HPV E7 proteins with the pCAF acetyltransferase.
Avvakumov N, Torchia J, Mymryk JS.
Department of Microbiology and Immunology, The University of Western Ontario, London Regional Cancer Centre

Most cervical carcinomas express the E6 and E7 proteins of a high-risk human papillomavirus (HPV). These proteins affect growth control by interfering with the functions of cell regulatory proteins, promoting oncogenic transformation. A key target of E7 is the tumor suppressor protein pRb, which directly interacts with E7. However, binding to additional cellular regulatory proteins is clearly required for oncogenesis, as mutants of E7 have been identified that bind to pRb, yet fail to transform efficiently. Here we demonstrate the interaction of the HPV 6, 16 and 18 E7 proteins with the PCAF acetyltransferase, which has been reported to function as a coactivator for a variety of transcription factors including p53. Mutation of a highly conserved leucine residue within the zinc finger region of HPV 16 E7 disrupts binding to pCAF and also impairs transformation and transcriptional activation. HPV 16 E7 interacts with the acetyltransferase domain of pCAF, and reduces its acetyltransferase activity in vitro. Our analysis of the interaction between the pCAF acetyltransferase and E7 provides new insight into the mechanisms by which the E7 oncoproteins can alter cellular gene expression and growth.

EXAMPLE ONLY