microRNA 2012
International Symposium

INSTRUCTIONS FOR ABSTRACTS

INSTRUCTIONS FOR PRESENTING THE ABSTRACT

Deadline: February 06, 2012

1) The Scientific Committee will select abstracts for Oral Presentations.

2) The abstract must be written in **English**.

3) The abstract should only be sent **electronically to microrna2012@gmail.com in the DOC/DOCX format**. We will not receive files in the **PDF** format.

4) Send only the abstract of an **original** (unpublished) paper.

5) The author presenting the abstract should be enrolled in the Congress. The submission of an abstract will only be considered after receipt of the registration.

6) An author, even though restricted to enrolment and presentation of **only one abstract**, can be the co-author of other abstracts.

7) The abstracts will be analyzed by the Scientific Committee of the Congress, and the result will be available at http://microrna2012.weebly.com website.

8) The analysis will encompass the following aspects: clearly defined relative objectives; adequate methodology; clearly presented results; pertinent conclusions.

9) The entire **responsibility** for the quality of the text (grammar, orthography and typing) falls to the author, and will be considered as a **criterion for evaluation** by the Scientific Committee.

10) Descriptions of projects, working intention, bibliographic revisions or already published papers, will **not** be accepted.

11) Enrollment with abstracts will **not** be accepted after February 6th. After this date, forms for the submission of abstracts will be blocked.

12) Selected abstracts should remain on display, in the form of panels, the duration of the Congress. The area reserved for each panel will be **1m x 1m**.

13) The poster must be presented in **ENGLISH**.

14) The poster should be accompanied by a **loop for fixing and/or Scotch tape**.

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MODEL OF THE ABSTRACT

The number of words (from the introduction to the conclusion) should remain between 200 and 450 (count with “tools” from Word).

Programmed cell death 4 loss increases tumor cell invasion and is regulated by miR-21 in oral squamous cell carcinoma

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The tumor suppressor Programmed Cell Death 4 (PDCD4) has been found to be under-expressed in several cancers and associated with disease progression and metastasis. There are no current studies characterizing PDCD4 expression and its clinical relevance in Oral Squamous Cell Carcinoma (OSCC). Since nodal metastasis is a major prognostic factor in OSCC, we focused on determining whether PDCD4 under-expression was associated with patient nodal status and had functional relevance in OSCC invasion. We also examined PDCD4 regulation by microRNA 21 (miR-21) in OSCC. PDCD4 mRNA expression levels were assessed in 50 OSCCs and 25 normal oral tissues. PDCD4 was under-expressed in 43/50 (86%) OSCCs, with significantly reduced mRNA levels in patients with nodal metastasis ($p = 0.0027$), and marginally associated with T3-T4 tumor stage ($p = 0.054$). PDCD4 protein expression was assessed, by immunohistochemistry (IHC), in 28/50 OSCCs and adjacent normal tissues; PDCD4 protein was absent/under-expressed in 25/28 (89%) OSCCs, and marginally associated with nodal metastasis ($p = 0.059$). A matrigel invasion assay showed that PDCD4 expression suppressed invasion, and siRNA-mediated PDCD4 loss was associated with increased invasive potential of oral carcinoma cells. Furthermore, we showed that miR-21 levels were increased in PDCD4-negative tumors, and that PDCD4 expression may be down-regulated in OSCC by direct binding of miR-21 to the 3'UTR PDCD4 mRNA. Our data show an association between the loss of PDCD4 expression, tumorigenesis and invasion in OSCC, and also identify a mechanism of PDCD4 down-regulation by microRNA-21 in oral carcinoma. PDCD4 association with nodal metastasis and invasion suggests that PDCD4 may be a clinically relevant biomarker with prognostic value in OSCC.
Financial Support: Ontario Institute for Cancer Research (OICR) (SKR, JI, PG, IJ, and BPO), the Galloway Fund (RG, SKR), the Canada Research Chair Program (IJ) and the CIHR Catalyst Grant #202370 (IJ).
