Canadian Association of Hospital Dentists | Association canadienne des dentistes en milieu hospitalier

ABSTRACT SUBMISSIONS

Guidelines

1) Regular IADR rules apply including, but not limited to, the following.
2) Abstract is limited to 300 words (not including title, authors, affiliations, and acknowledgements).
3) Title is limited to 10 words. Capitalize the first letter of each word except for prepositions and articles such as the, of, in, a, and, etc.
4) Projects being submitted should be original, completed research. Previously published abstracts or those presented at another meeting earlier than March 1, 2018 are not allowed. Abstracts that duplicate those to be sent to a future formal meeting are acceptable, but they should not be duplicated on the same person's CV.
5) Each person may submit only one abstract as the presenting author, but an individual may co-author multiple abstracts if so desired. **The abstract should be presented by the person who has conducted the research project.**
6) Separate author names with a comma and place an asterisk after the name of the presenting author. If the authors are from different institutions, distinguish their affiliations with superscript Arabic numerals. Please remember to obtain permission from each co-author **before** you submit your abstract.
7) Abstracts must contain a brief statement of: (a) the purpose of the investigation; (b) the experimental methods used; (c) the essential results including data and, where appropriate, statistics; (d) the conclusion; and (e) any external funding sources, including the name of the supporting agency and the grant number, if applicable.
8) Italicize scientific names of organisms. Do not underline the conclusions. Do not indent paragraphs. Do not include references.
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12) Please save your file as a Word document named "CAHD-ACDH Abstract Last name, First name" and e-mail it to Dr. Mel Schwartz at melschwartz@jgh.mgill.ca by **March 19, 2018.** Abstract acceptance notifications will be sent out by or around April 1, 2018. If you do not receive an acceptance notification, please contact Dr. Mel Schwartz.

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Sample Abstract

Stromal-Epithelial Cytokine Crosstalk in Experimentally Induced Periodontal Disease

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Objectives: Lipopolysaccharide (LPS) is a bacterial virulence factor implicated in the conversion of junctional to pocket epithelium, an early marker of periodontal disease onset. During disease progression, the epithelial barrier is compromised, allowing virulence factors to insult underlying stroma. We sought to determine if LPS-induced changes in diffusible gene products could affect signaling crosstalk between stromal and epithelial tissues, contributing to disease.

Methods: Wistar strain rats (14 male) were divided between time 0 control and 8-week treatment groups. LPS was applied daily into the gingival sulcus and histological analysis confirmed the onset of disease. Junctional epithelium and underlying stromal tissue was separately collected from healthy and diseased animals by laser-capture micro-dissection and subject to gene expression microarray analysis. Genmapp bioinformatic analysis was performed to identify gene ontology function groups of high significance ($z \geq 4$) whose protein products could potentially interact. *In vitro* validation used a chronic wound cell culture model and protein analysis by flow cytometry.

Results: LPS-altered gene ontology function grouping top-ranked the molecular binding category in both epithelia and stromal tissues. However, for stroma, the cytokine subgroup ranked near the top ($z=5.991$). Its three top-ranked stromal genes (amphiregulin, interleukin 1-β, and Fas ligand) are known to be diffusible and capable of modulating the epithelial growth factor (EGF) pathway. For epithelia, several binding subgroups associated with the EGF receptor were highly ranked, including ErbB-2 class receptor binding ($z=4.994$). Its top three altered genes (Fos ligand, mucin 4, and somatostatin receptor) were downregulated. All are reported as playing a role in normally inhibiting EGF signaling. Upregulation of all 3 stromal and downregulation of all 3 epithelial gene products was confirmed *in vitro* for up to 3 weeks with LPS treatment.

Conclusions: LPS may contribute to the onset of periodontitis by upregulating EGF pathway activity via stromal-epithelial crosstalk.

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