PATHOLOGY AND LABORATORY MEDICINE

RESEARCH DAY 2019

Per Department policy, **ALL** Pathology and Laboratory Medicine **residents and students** (Research-based graduate program, Pathologists' Assistant graduate program, and undergraduate program) are expected to participate. Please fill out the *exemption form* (attached to this message) and have the form signed by your *Program Director* to be exempted from submitting an abstract and participating in our research day. Faculty, postdoctoral fellows, and technical staff are also encouraged to submit an abstract.

Deadline for Abstract Submission:	**February 22, 2019**
Notification of Oral or Poster presentation:	February 28, 2019
Deadline for Submission of Posters:	March 15, 2019

Abstracts should be submitted in electronic format (see details below) by the submission deadline. Authors may indicate their *preference* either for a poster presentation or for an oral/platform presentation. The Pathology and Laboratory Medicine Research Day Committee, comprised of basic and clinical faculty, will review all abstracts (and indicated preference for poster/platform) and select abstracts for platform presentation. Notification of decisions will be sent on February 28, 2019.

We hope to see you all at the 2019 Pathology and Laboratory Medicine Research Day

**Please follow these guidelines carefully.

Abstract Submission Guidelines

- ❑ Use a text editor to compile your abstract: Title, Author(s), and Affiliation of author, Abstract text, and Keywords.
- ❑ Submit your abstract (word or plain text) electronically to Pathology.Research@schulich.uwo.ca

Abstract Sections & Formatting

Title: Provide a concise title of your abstract (15-word maximum). Only the first letters of major words should be capitalized.

Authors: Include all authors (including your supervisor and any other authors that assisted you in your research project). The authorship order usually follows the format: the presenting author (you) will be first, the corresponding/senior author (supervisor/mentor) will be last, and supporting authors will be in the middle in order of contribution level. Do not include degrees or professional titles.

Affiliation(s): Provide affiliation of all authors on the abstract.

Abstract Text: Abstract text should not be more than **300 words**. For basic and clinical science research projects, provide a structured abstract following the **IMRAD** format (a defacto standard that reflects the process of scientific discovery). The abstract should include **I**ntroduction, **M**ethods, **R**esults, **a**nd **D**iscussion. See example below.

A structured format may not be applicable to certain abstracts. Examples include case studies, viewpoints, practice innovations, laboratory efficiency reports, reports on improving patient care, team integration strategies etc. A *narrative abstract* is acceptable for these reports. There are no headings within the narrative abstract. See example below.

Keywords: At the end of the page, include 5-10 keywords that best describe your research project.

Presentation Preference: Please indicate your preference for platform or poster presentation.

Example of a scientific/structured abstract

Regulation of Vascular Endothelial Growth Factor Expression by Fibronectin in Endothelial Cells

Shali Chen[1], Rana Chakrabarti[1], Emily C. Keats[1], Megan Chen[1], and Subrata Chakrabarti[1,2]

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Introduction: Diabetic retinopathy entails proliferation of vascular endothelial cells (ECs) and unregulated angiogenesis. We have previously shown that ECs increase the expression of an embryonic variant of fibronectin, called extra domain-B fibronectin (ED-B FN) in response to high glucose. We also showed that ED-B FN regulates EC tube morphogenesis possibly through vascular endothelial growth factor (VEGF). In the present study, we have attempted to decipher the mechanisms by which ED-B FN may modulate EC phenotype. We hypothesize that ED-B FN regulates VEGF expression in ECs through interaction with selected integrin receptors.

Methods: To test this hypothesis, we first cultured ECs in high levels of glucose to investigate for any alteration. We then used integrin-specific matrix mimetic peptides, neutralizing antibodies, and RNAi to identify the integrin(s) involved in VEGF expression. Finally, we used an animal model of diabetes to study whether these *in vitro* mechanisms also take place in the retina.

Results: Our results show the exposure of ECs to high levels of glucose increases VEGF expression. ED-B FN mediated this increase since knockdown of ED-B FN completely prevented glucose-induced VEGF expression. We then identified β1 integrin as the essential receptor involved in high glucose-induced VEGF expression. We also show that diabetes increases β1 integrin and VEGF expression in the retina, which normalizes upon ED-B knockdown.

Conclusions: These findings show that high levels of glucose in diabetes increase VEGF expression in ECs through ED-B FN and β1 integrin interaction. These results provide a mechanistic basis of increased VEGF expression in diabetes.

Keywords: VEGF, endothelial cells, fibronectin, extracellular matrix, angiogenesis, diabetes

Presentation Preference: Poster or platform

Example of a case study abstract

Cytomorphologic spectrum of mixed pituitary adenoma-gangliocytomas: a report of two cases

Joanne Sy[1] and Lee-Cyn Ang[1,2]

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Introduction: Mixed pituitary adenoma-gangliocytomas are rare tumorus with a broad morphologic spectrum. Smear cytology is a useful tool for recognizing these tumours in an intraoperative setting.

Cases: The patients were 45 and 30 years old, and both presented with headache. Intraoperative smears in both cases showed a tumor composed of adenomatous and neuronal elements, in varying proportions. The first case had sheets of monotonous neuroendocrine-type cells with occasional interspersed ganglion cells. The second case, however, had a prominent fibrillary background and was predominantly neurocytic, with a mixture of large ganglion-like cells, intermediate cells, and only rare adenomatous cells.

Discussion: The diagnostic features of mixed pituitary adenoma-gangliocytomas can be recognized on intraoperative smear preparations. Smear preparations are often more useful than frozen sections because freezing artifacts may mask one of the two components of the tumour. The proportion of adenomatous and neuronal elements can vary widely from case to case. Careful search for a neuronal component should be made, especially if there is a clinical history of a pituitary adenoma showing incomplete response to hormonal therapy.

Keywords: Cytology, pituitary tumour, gangliocytoma, adenoma, tumour heterogeneity, cell morphology

Presentation Preference: Poster or platform

Example of a narrative abstract

Age-dependent protein misfolding and toxicity

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The most common and highest risk factor for neurodegenerative diseases, such as Alzheimer's disease and Parkinson's disease is advanced age. Most neurodegenerative diseases are caused by protein misfolding and protein misfolding and its ensuing toxicity are triggered and enhanced during aging. Yet the molecular mechanisms underlying aged-induced protein misfolding are mostly unclear. We employ chronologically aged yeast cells expressing misfolded polyglutamine (polyQ) expansion proteins to study this problem. PolyQ-expansion proteins are the basis for nine different neurodegenerative diseases (e.g. Huntington's disease), each of which presents a strongly age-dependent onset. In our yeast model, we observe a striking age-dependent modulation of polyQ-aggregation and polyQ-toxicity. This age-dependent toxicity and misfolding of the polyQ-expansion proteins in yeast depends on the amino acids that flank the polyQ-expansion region. Further, specific branches of the cellular protein quality control determine the aggregation status and thereby the toxicity of polyQ-expansion proteins.

Keywords: protein misfolding, neurodegeneration, cell toxicity, gene expression, aging

Presentation Preference: Poster or platform