

PATHOLOGY AND LABORATORY MEDICINE

RESEARCH DAY 2021

Abstract Submission

Per Department policy, **ALL** Pathology and Laboratory Medicine **residents** and **students** (Research-based graduate program, Pathologists' Assistant graduate program, and undergraduate programs) are expected to participate. Please fill out the *exemption form* and have the form signed by your *Program Director* to be exempted. Faculty, postdoctoral scholars, and technical staff are also encouraged to submit an abstract.

Deadline for Abstract Submission: **March 10, 2021**
Notification of Oral or Poster presentation: March 17, 2021
Deadline for Submission of Poster files: March 31, 2021

Abstracts should be submitted using the Pathology and Laboratory Medicine [Abstract Submission Form](#) by the submission deadline.

Oral or Poster Presentation Selection:

Authors may indicate their preference either for a poster presentation or for an oral/platform presentation. The Pathology and Laboratory Medicine Research Committee, comprised of basic and clinical faculty, will review all abstracts (and indicated preference for poster/platform) and select abstracts for platform/poster presentation. Notification of decisions will be sent on March 17, 2021.

Research Day Awards:

Pathology and Laboratory Medicine has created numerous awards to recognize and celebrate research excellence in our members, including our undergraduate and graduate trainees, clinical fellows and postdoctoral scholars, faculty and staff.

Selection of the awardees is based on the composite score which weighs the submitted abstract and the presentation (oral or poster). Submitted abstracts will be scored by the Research Committee or invited faculty members. A panel of judges will also score the oral or poster presentation.

Abstract Submission Guidelines

Instructions are provided on the Pathology and Laboratory Medicine [Abstract Submission Form](#) website. Examples of different abstract formats is provided below.

Example of a structured abstract (IMRAD-style)

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

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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 $\beta 1$ integrin as the essential receptor involved in high glucose-induced VEGF expression. We also show that diabetes increases $\beta 1$ integrin and VEGF expression in the retina, which normalizes upon ED-B knockdown.

Discussion: These findings show that high levels of glucose in diabetes increase VEGF expression in ECs through ED-B FN and $\beta 1$ integrin interaction. These results provide a mechanistic basis of increased VEGF expression in diabetes.

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

Example of a structured case study abstract

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

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Introduction: Mixed pituitary adenoma-gangliocytomas are rare tumours 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 of ten 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

Example of a narrative (free-flowing) 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