PATHOLOGY AND LABORATORY MEDICINE

RESEARCH DAY 2025

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. Faculty, postdoctoral scholars, and technical staff are also encouraged to submit an abstract.

Deadline for Abstract Submission:	**February 28, 2025**
Notification of Oral or Poster presentation:	**March 7, 2025**
Deadline for Submission of Poster files:	**March 28, 2025**

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 Day 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 February March 7, 2025.

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)**

please note that IMRAD is the preferred format for submitting abstracts.

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

Shali Chen, Chakrabarti R, Keats EC, Chen M, Chakrabarti S

Pathology and Laboratory Medicine, Western University

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.

Discussion: 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

Example of a structured case study abstract

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

Joanne Sy, Ang LC

Pathology and Laboratory Medicine, Western University

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 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

Example of a narrative (free-flowing) abstract

Dysregulated transforming growth factor beta mediates bone marrow dysfunction early in diabetes

Jina JY Kum, Howlett CJ, Khan ZA

Pathology and Laboratory Medicine, Western University

Morbidity and mortality associated with diabetes are due to secondary complications that include both micro- and macro-vascular organ dysfunctions. Our recent studies show that diabetes also enhances adipogenesis in the bone marrow and reduces the number of marrow-resident vascular regenerative stem cells. In the current study, we have identified the early changes induced by diabetes in the bone marrow. Even as early as 1 month after diabetes onset in mice, changes, both structural and molecular, were evident in the marrow. These alterations occur before other target organs exhibit diabetes-induced dysfunction. Importantly, we showed that short-term diabetes enhances adipogenesis in tibiae of mice prior to stem cell depletion. This enhanced adipogenesis was associated with suppressed transforming growth factor beta (TGFB) signaling pathway. Using human bone marrow-derived mesenchymal progenitor cells (bm-MPCs), we then investigated the functional significance of TGFB signaling suppression. We show that exposure of bm-MPCs to high levels of glucose suppresses the TGFB pathway, mimicking the effect of hyperglycemia in diabetic mice. Supplementation of TGFB prevented adipogenic differentiation of bm-MPCs. Dissection of the intracellular signaling pathways revealed that TGFB1 utilizes the non-canonical TGFB-activated kinase 1 (TAK1)-mediated mechanism to inhibit adipogenesis. Transcriptome-wide gene expression profiling revealed a potential involvement of the Wnt pathway. Taken together, our studies identified enhanced bone marrow adipogenesis in diabetic mice before marrow-resident stem cell depletion and other known diabetic complications become evident. We further identified suppressed TGFB signaling pathway as a mechanism that potentially leads to deleterious adipogenesis in bones in diabetes.

Keywords: diabetes, diabetic complications, bone marrow, adipogenesis, cell differentiation, adipocytes