## Poster Presentations

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**IMMUNOHISTOCHEMICAL EVIDENCE OF ENDOMETRIAL STROMAL ALTERATION IN DYSFUNCTIONAL BLEEDING**

I Teo, MD[2], H Al-Maghrabi, MD[2], M Lamba, MD FRCPC[1], JP Veinot MD FRCPC[1], KT Mai, MD FRCPC[1]. [1]Department of Laboratory Medicine, The Ottawa Hospital and [2]Department of Pathology and Laboratory Medicine, University of Ottawa, Ottawa, Ontario, Canada.

Aims: It is commonly believed that dysfunctional bleeding (DUB) is caused by ovulation disorders resulting in hormonal disturbances, which in turn lead to disturbed angiogenesis. The role of endometrial stromal cells (ESC) in DUB is neither well understood nor emphasized in the assessment of endometrial biopsies. In normal premenopausal endometrium, calretinin reactivity is limited to the functionalis layer (FL). The FL displays zonal reactivity ranging from superficial zone of the FL in the early proliferative phase to full-thickness in the secretory phase. In the menstrual period, ESC display negative or focal reactivity. Throughout all phases, CD34 is limited in the basalis layer (BL).
Materials and Methods: Immunostains for calretinin and CD34 were performed on 50 endometrial specimens from women with DUB.
Results: Regardless of hormone exposure or histologic appearance, ESC from women with DUB often showed weaker calretinin reactivity than in normal endometrium, with focal to extensive loss of reactivity in the FL. In all cases with DUB, CD34 reactivity appeared to extend from the BL into the FL and was seen in areas with or without calretinin reactivity. In superficial secretory endometrium, endometrial glands surrounded by calretinin- or CD34+ stroma tended to be asynchronic as compared with adjacent glands
Conclusions: The altered calretinin and CD34 stromal reactivity suggests an expansion of the BL-type stroma into the FL, generating a "disordered endometrial stroma."

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**PROGESTERONE RECEPTOR REACTIVITY IN RENAL ONCOCYTOMA AND CHROMOPHOBE RENAL CELL CARCINOMA**

KT Mai, MD FRCPC[1], I Teo, MD[2]., N Roustan Delatour, MD[2], SJ Robertson, MD FRCPC[1], EC Marginean, MD FRCPC FACP[1,2]. [1]Division of Anatomical Pathology, Department of Laboratory Medicine, The Ottawa Hospital, Ottawa, Ontario; [2]Department of Pathology and Laboratory Medicine, University of Ottawa, Ottawa, Ontario, Canada

Aims: ~~Estrogen (ER) and progesterone receptors~~ (PR) display less than 1% positive reactivity in renal cell carcinoma ~~(RCC). ER and PR~~ reactivity in renal oncocytoma (RO) and chromophobe RCC (CHRCC) has not been investigated. We study the ER and PR immunohistochemical staining as potential diagnostic markers for RO and CHRCC.
Materials and Methods: 38 RO, 25 CHRCC (10 oncocytic CHRCC and 15 typical CHRCC), 20 OPRCC and 10 eosinophilic (granular) clear cell RCC were stained for ER, PR, CD117 and RCC.
Results: All RO and eosinophilic variants of CHRCC, including cases with sarcomatoid changes, displayed moderately positive nuclear reactivity for PR. The nuclear reactivity ranged from 90% to 60% in RO, and from 70% to occasional cells in eosinophilic variants of CHRCC. In CHRCC, tumour cells with eosinophilic cytoplasm had more diffuse reactivity, whereas cells with vacuolated cytoplasm, sarcomatoid changes and non-oncocytic CHRCC had more focal reactivity. Typical CHRCC were not reactive for PR. CD117 reactivity tended to be stronger in the typical CHRCC than eosinophilic CHRCC. No tumours reacted with ER.
Conclusions: PR is a highly sensitive and specific maker for RO and oncocytic CHRCC. Therefore, PR can be used in combination with CD117 and RCC in the differential diagnosis of RO and eosinophilic variant of CHRCC with other oncocytic types of RCC.

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**BUILDING CANADIAN IMMUNOHISTOCHEMISTRY QUALITY CONTROL (CIQC)**

S. Bahzad[1], B. Gilks[2], S. Klassen, and E. Torlakovic[1]. Departments of Pathology and Laboratory Medicine, Royal University Hospital, University of Saskatchewan, Saskatoon, SK[1], and Vancouver General Hospital, The University of British Columbia, Vancouver, BC.

Immunohistochemistry is an integral part of anatomic pathology and hematopathology practice and its outcome is the basis of an expanding number of diagnoses and also treatment choices. Despite its wide and routine clinical use, its standardization still lags behind. External quality control/assurance allows comparison of performance and results, serves as an early warning system for problems, identifies systematic kit problems, provides objective evidence of laboratory quality, serves as an indicator of where to direct improvement efforts, and identifies training needs. No such national program is established in Canada so far. Fifteen Canadian clinical immunohistochemistry laboratories were invited to stain tissue microarrays (TMA) slides that contained 76 tissue cores to represent lesions with various expressions of tested epitopes. Selected were tests that are in daily use for evaluation of undifferentatiated tumours: pancytokeratin, low molecular weight cytokeratin (LMWCK), vimentin, S-100, and HMB-45. In appropriate setting, these markers enable distinction between carcinoma, melanoma, and sarcoma. The stains were scored on the scale of 0 to 3+ with separate scores for pathological or predominant cell population as appropriate and background non-specific staining. Pancytokeratin staining produced from 0 to 30% false negative rate, with similarly significant differences between the laboratories also for LMWCK, vimentin, and S-100. While most laboratories employed similar detection methods, the differences appeared to be secondary to variations in antigen retrieval procedures or dilution of the primary antibodies. The results are in more detail posted for viewing and virtual microscopy at www.ciqc.ca. We conclude that Canadian clinical immunohistochemistry laboratories produce variable results even with most commonly used test and that an external QC programs would probably help to achieve standardization in immunohistochemistry. also false positive

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**CYCLOPHILIN C-ASSOCIATED PROTEIN (CyCAP) NULL MICE: A MODEL OF EXPERIMENTAL MUCOSAL HYPERPLASIA OF THE COLON**

C. Wang[1], E. Torlakovic[1], Vicki Keeler[2], T. Benerjee[1], S. Laferté, Ph.D.[2]. Departments of Pathology, Royal University Hospital[1], and Biochemistry[2], University of Saskatchewan, Saskatoon, Canada.

The discovery of a "serrated neoplasia pathway" has shifted the attention from classical adenomas to serrated/hyperplastic mucosa as the significant precursor of colorectal carcinoma. In mice, hyperplasia of the colonic mucosa is a regular phenomenon after a challenge with colonic carcinogens. CyCAP, murine orthologue of the tumour-associated antigen 90K (TAA90K)/Mac-2 BP, is a widely expressed secreted glycoprotein that modulates the host response to bacterial endotoxin.
Wild-type (WT) and CyCAP-knock out (KO) mice were treated by azoxymethane (AZ). Five animals were included in each group (WT, WT+AZ, KO, KO+AZ). The number and the size of colonic tumours was recorded/ The crypt depth was measured in at least 60 perfectly oriented crypts per animal and the number of colonocytes was counted in the same number of crypts.
KO+AZ animals had more mucosal hyperplasia than WT+AZ animals ($p=0.005$, Independent Sample T-test). In both groups, the crypt depth ($r=0.723$, $p=0.018$, Spearman Correlation) and colonocyte number ($r=0.863$, $p=0.001$, Spearman Correlation) were positively associated with total numbers of tumours and total tumour size. KO mice had larger numbers of tumours ($p=0.003$, Linear Regression) and overall larger tumour mass ($p=0.016$, Linear Regression). In fact, KO mice spontaneously developed colonic mucosal hyperplasia early in life ($p<0.0001$, Independent Sample T-test).
KO mice represent the first model of spontaneous colonic mucosal hyperplasia and highlight the potential role of CyCAP as a tumour suppressor during early stages of colonic carcinogenesis. Studies of KO mice should provide novel insights about the possible function of TAA90K in early stages of colon carcinogenesis.