

**9th ANNUAL DEPARTMENT OF PATHOLOGY YOUNG INVESTIGATORS' DAY
POSTER SESSION**

**Thursday, April 5th, 2007
TURNER CONCOURSE
REGISTRATION FORM**

**E-mail COMPLETED Registration form and abstract to:
Stacey Morgan (smorgan9@jhmi.edu) on or before
Friday, March 16th, 2007**

**If you have questions or problems regarding your submission, please contact Stacey
Morgan via e-mail (smorgan9@jhmi.edu)**

Applicant's Name: **Dengfeng Cao** Degree: M.D.,Ph.D.

Applicant's Division: Gynecologic pathology

Faculty Preceptor: **Brigitte Ronnett**
(Must hold a primary appointment in Pathology)

Appointment Category: Clinical Fellow

Register for: Clinical Research

Full Poster Title * **Expression of PAX2 in endometrial hyperplasia and endometrial
Carcinomas - an immunohistochemical study of 136 cases**

Where has the work been presented? YES

Meeting Name 2007 USCAP (San Diego)

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In Preparation Yes

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***INCLUDE A ONE-PAGE ABSTRACT (including title and all authors) OF THE WORK YOU WILL BE PRESENTING**

Expression of PAX2 in endometrial hyperplasia and endometrial Carcinomas - an immunohistochemical study of 136 cases

Dengfeng Cao, Allen Gown, Russell Vang, Robert Kurman, Brigitte Ronnett

Background: PAX2 (paired box 2) is a transcription factor in the PAX family. The *PAX2* gene is essential to the development of kidney, central nerve system and ear. A recent study (Wu et al. *Nature* 438: (15): 981-987) has reported that PAX2 was activated by estrogen and tamoxifen in endometrial carcinomas but was silenced in normal endometrium, suggesting its role as an oncogene in endometrial carcinogenesis. However, the status of PAX2 in normal endometrium, hyperplastic endometrium and endometrial carcinomas has not been systematically studied.

Design: One hundred thirty-six (136) cases formed the study group including 24 with complex or simple hyperplasia without atypia (CH/SH), 31 with complex atypical hyperplasia (CAH), 17 with well-differentiated endometrioid carcinoma (FIGO grade 1 EC), 28 with moderately differentiated endometrioid carcinoma (FIGO grade 2 EC), 18 with poorly differentiated endometrioid carcinoma (FIGO grade 3 EC), and 12 with serous carcinoma or endometrial intraepithelial carcinoma (SC/EIC). Immunohistochemical staining with PAX2 antibody was performed on unstained slides prepared from archival paraffin-fixed blocks (one block per case).

Results: Pax2 staining (only nuclear staining counted) was scored as 0 (<5% lesional cells), 1+ (5-25% cells), 2+ (25-50% cells), 3+ (50-75% cells) and 4+ (>75% cells). In 42 cases in which normal endometrial glands were also present for evaluation, all normal endometrial glands showed 3+ (2 cases) or 4+ (40 cases). Staining results in endometrial hyperplasias and carcinomas were as following:

Lesion	0 (<5% cells)	1+ (5-25%)	2+ (25-50%)	3+ (50-75%)	4+ (>75%)
CH/SH (N = 22)	4 (18%)	5	3	5	5
CAH (N = 31)	17 (55%)	7	2	2	3
FIGO grade 1 EC (N = 24)	19 (79%)	1	1	0	3
FIGO grade 2 EC (N = 28)	22 (85%)	1	2	0	3
FIGO grade 3 EC (N = 19)	18 (95%)	0	0	1	0
SC/EIC (N = 12)	6 (50%)	4	0	2	0

Conclusions: PAX2 is uniformly expressed at the protein level in normal endometrial glandular cells, implicating this gene is not silenced in normal endometrium. Loss of PAX2 expression at the protein level occurs early during pathogenesis of type I endometrioid carcinoma, arguing against its oncogene role of *PAX2* in endometrial carcinomas. Loss of PAX2 expression in hyperplasias (18-55%) and type I endometrial carcinomas (79-95%), and its expression in some type II endometrial carcinomas suggests that mechanisms other than ER or tamoxifen probably regulate PAX2 in endometrial lesions.