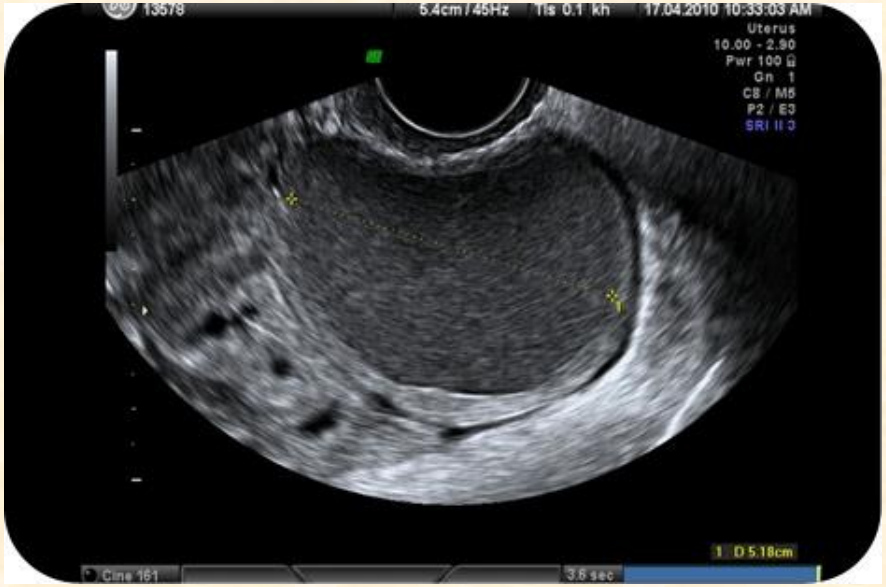
L'endométriose est-elle une lésion pré-cancéreuse ? Perspectives et implications cliniques

Pr G Chêne

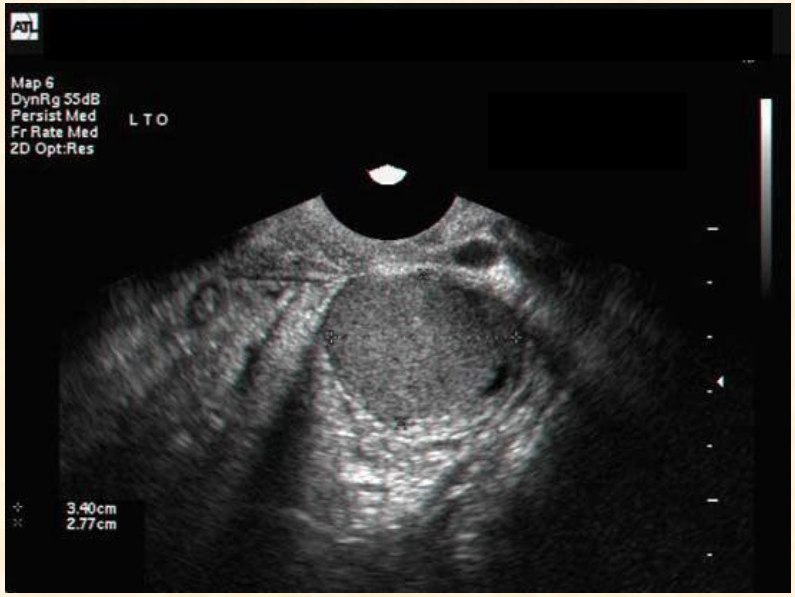
Département de Gynécologie, HFME, HCL, Lyon

06-11-2015

Quelle est la différence ?



Mme A



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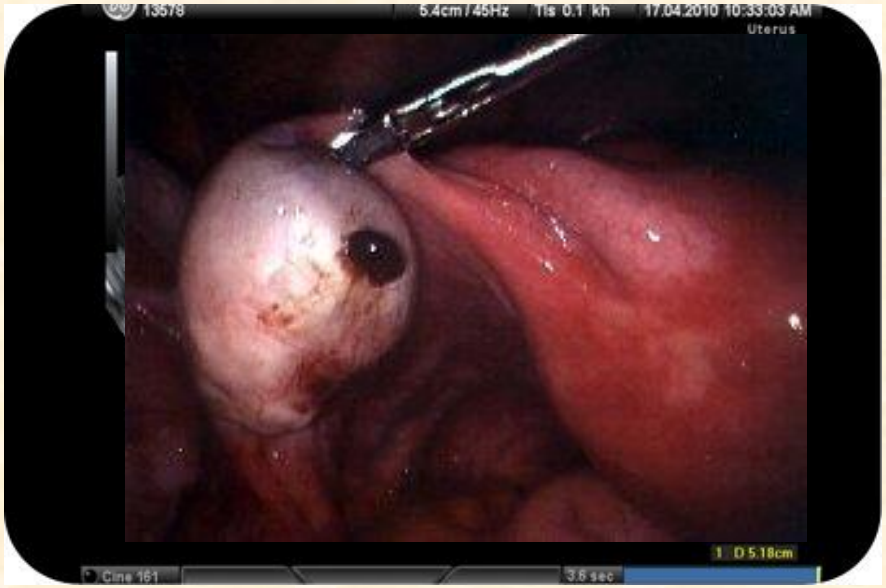

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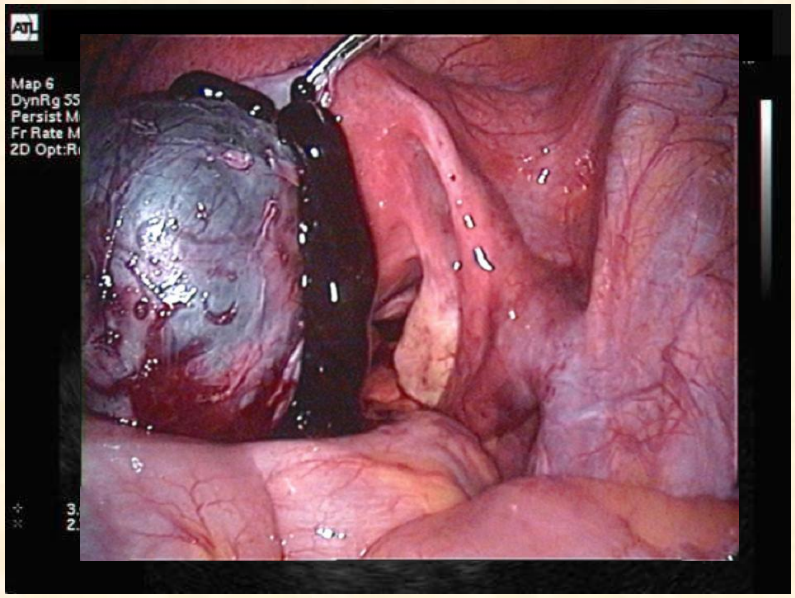

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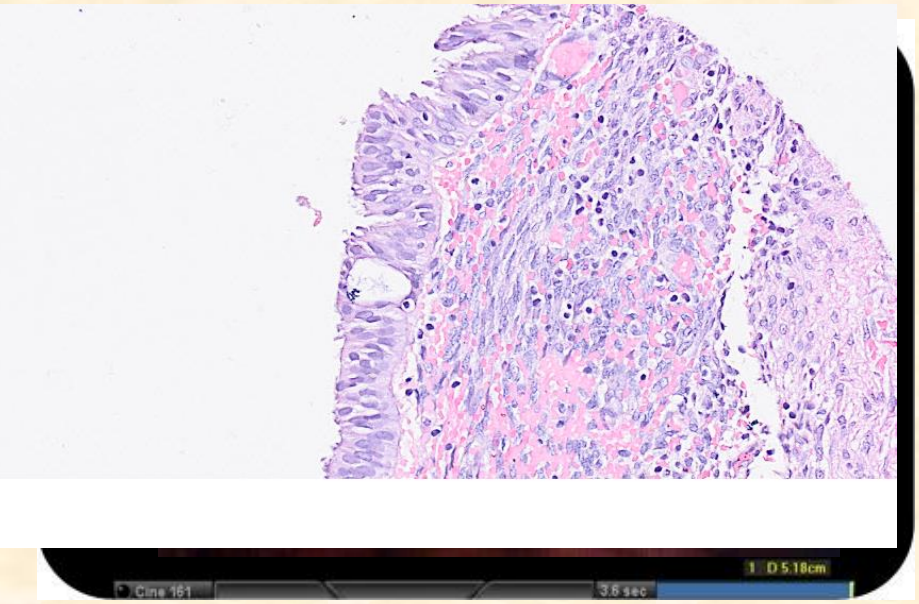

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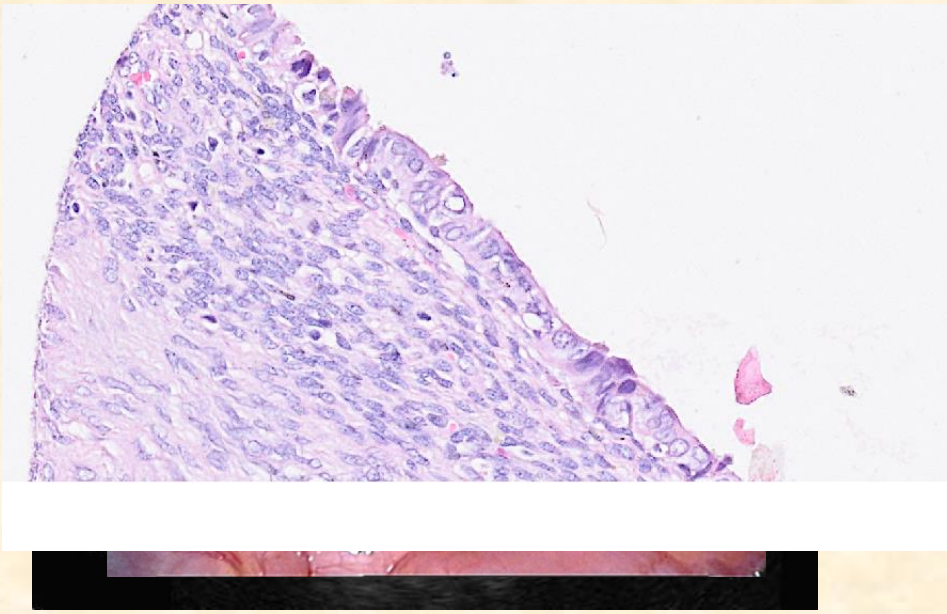

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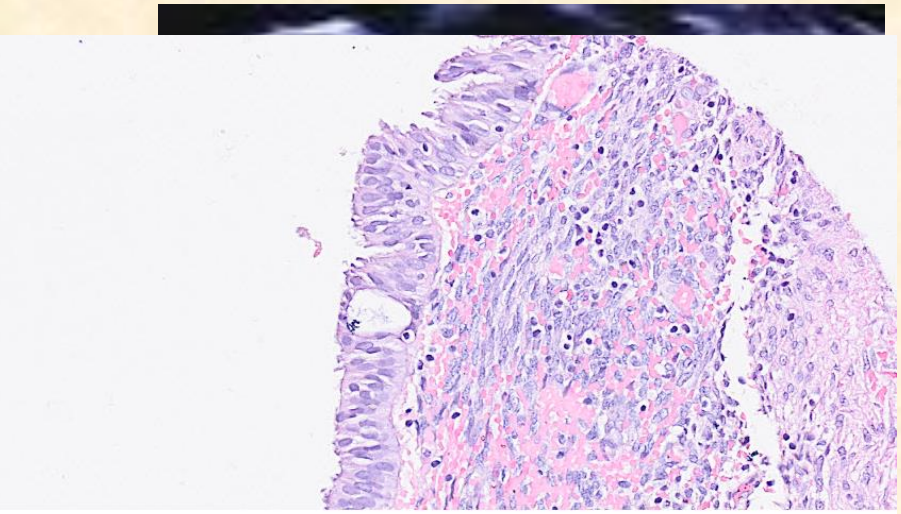

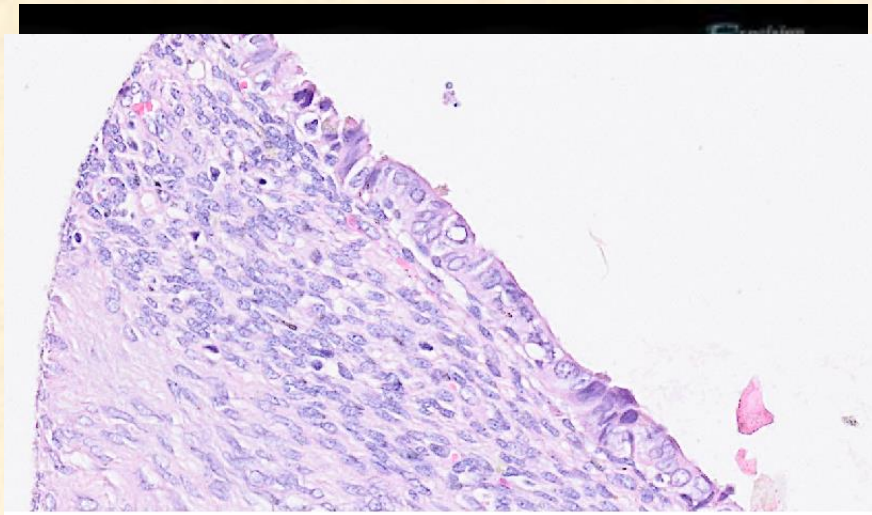

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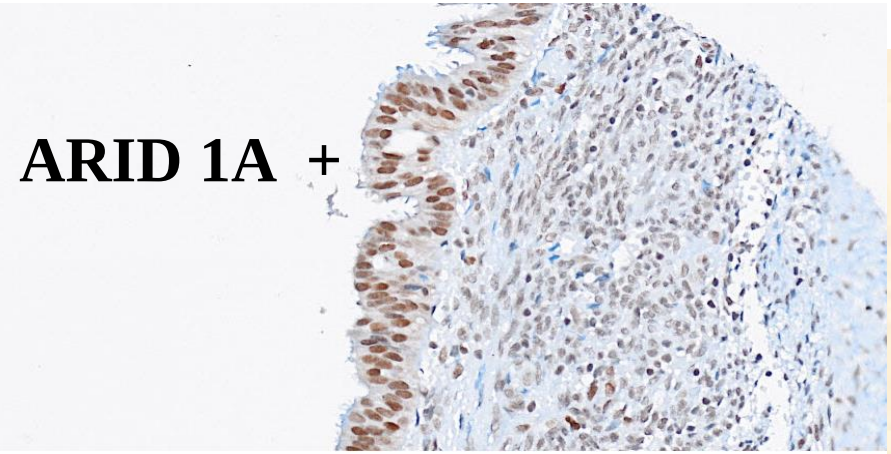
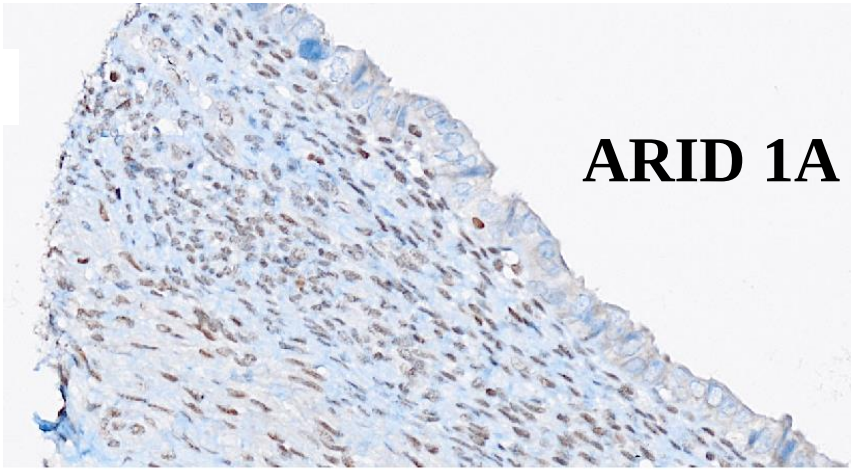
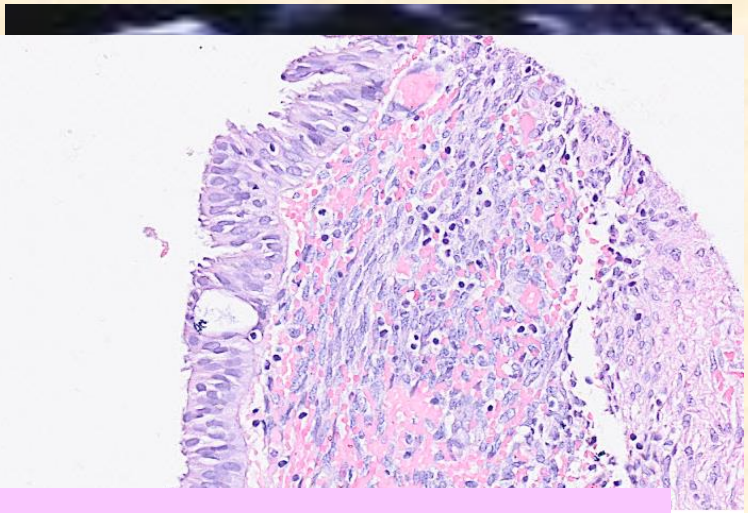


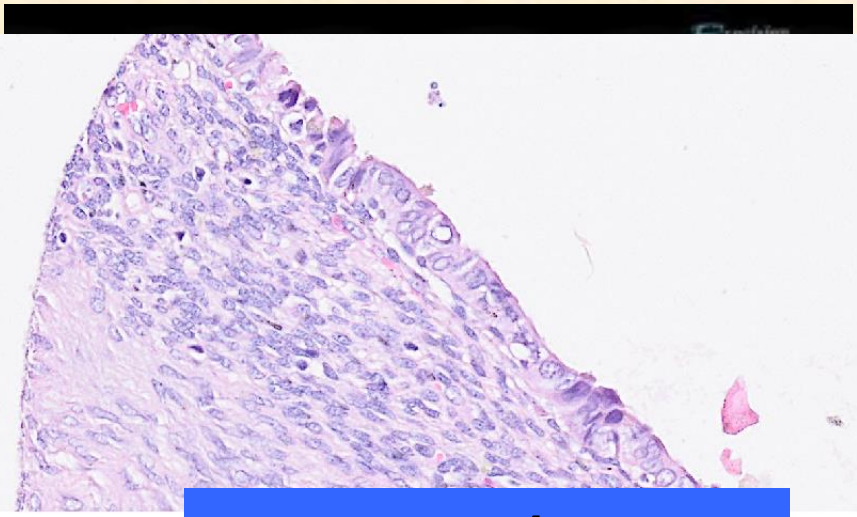


ARID 1A -

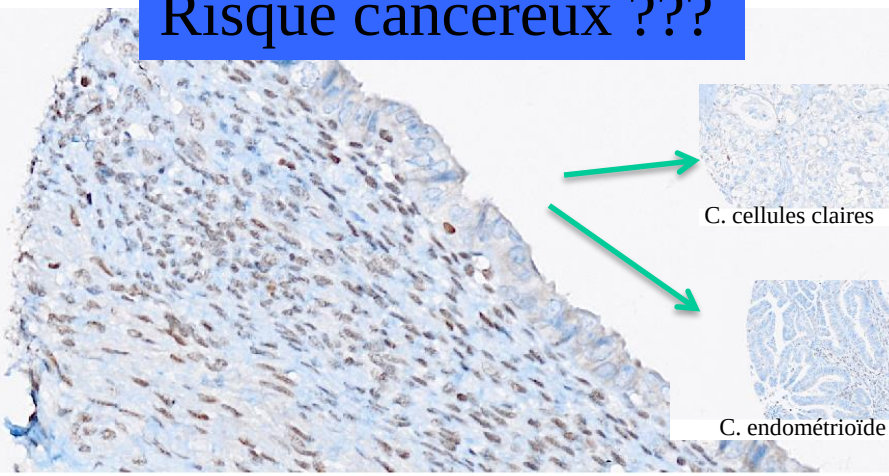
Quelle est la différence ?



Pas de risque cancéreux



Risque cancéreux ???



Association cancer ovarien et endométriose (1)

- Dr J.A. Sampson, 1925:
  - Association intime endométriose et cancer dans le même ovaire
  - Pas d'autre localisation cancéreuse primitive
  - Histologie compatible

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VOL. 10 JANUARY, 1925—IN TWO PARTS—PART 1 No. 1

ENDOMETRIAL CARCINOMA OF THE OVARY, ARISING IN ENDOMETRIAL TISSUE IN THAT ORGAN*

JOHN A. SAMPSON, M.D.

ALBANY, N. Y.

Many interesting and important problems have presented themselves to clinicians and pathologists, who have appreciated the frequency of ectopic endometrium-like tissue in the pelvis and have had an opportunity to observe the various lesions in the ovaries and other pelvic structures resulting from this tissue. One of the most interesting problems is the source of these implantation-like lesions occurring on the surface of the various pelvic structures. Are they true implantations derived primarily from uterine or tubal epithelium escaping through the tubes into the peritoneal cavity, as the study of many of these lesions in human beings indicates[1] and as Jacobson,[2] in the autotransplantation of endometrial tissue in rabbits, has experimentally proved possible, or are they derived from scattered localized metaplasias of the serosal mesothelium or from developmental inclusions of portions of the Wolffian body or Müllerian duct? This problem has not yet been definitely solved to the satisfaction of all who are interested in the subject. An exhaustive presentation of the serosal origin of these lesions has recently been published by Lauche.[3] Irrespective of their

* From the Gynecological and Pathological Departments of the Albany Hospital and the Albany Medical College. Presented in part at the Forty-Ninth Annual Meeting of the American Gynecological Society, May 17, 1924.

1. Sampson, J. A.: Intestinal Adenomas of Endometrial Type, Arch. Surg. **5**:217-280 (Sept.) 1922; The Life History of Ovarian Hematomas (Hemorrhagic Cysts) of Endometrial (Müllerian) Type, Am. J. Obst. & Gynec. **4**:451-512 (Nov.) 1922; Benign and Malignant Endometrial Implants in the Peritoneal Cavity and Their Relation to Certain Ovarian Tumors, Surg., Gynec. & Obst. **38**:287-311 (March) 1924.

2. Jacobson, V. C.: The Autotransplantation of Endometrial Tissue in the Rabbit, Arch. Surg. **5**:281-300 (Sept.) 1922; Further Studies in Autotransplantation of Endometrial Tissue in the Rabbit, Am. J. Obst. & Gynec. **6**:257-262 (Sept.) 1923.

3. Lauche, Arnold: Die Extrogenitalen heterotopen Epithelwucherungen von Bau der Uterusschleimhaut (Fibroadenomatosis Seroepithelialis), Virchow's Archiv. f. Pathologische Anatomie und Physiologie **243**:298-372 (April) 1923.

Association cancer ovarien et endométriose (2)

- Analyse de 13 études :
  - 7.911 cancers ovariens et 13.226 témoins

	Study name	Study abbreviation	Study type	Method of data collection	Ascertainment period
Asia-Pacific					
Australia	Australian Cancer Study*†, Australian Ovarian Cancer Study*†[9]	AUS	Population based	Self-completed questionnaire, checked by trained research nurse	2002–06
Europe					
Germany	German Ovarian Cancer Study†[10]	GER	Population based	Self-completed questionnaire	1992–98
Denmark	Malignant Ovarian Cancer Study[11]	MAL	Population based	In-person or phone interview	1994–99
UK	United Kingdom Ovarian Cancer Population Study[12]	UKO	Population based	Self-completed questionnaire	2006–07
USA					
CT	Connecticut Ovary Study[13]	CON	Population based	In-person interview	1999–2003
WA	Diseases of the Ovary and their Evaluation Study†[5]	DOV	Population based	In-person interview	2002–05
HI	Hawaii Ovarian Cancer Study[14]	HAW	Population based	In-person interview	1994–2007
Western PA, northeast OH, western NY	Hormones and Ovarian Cancer Prediction Study†[15]	HOP	Population based	In-person interview	2003–08
North central states (MN, SD, ND, IL, IA, WI)	Mayo Clinic Ovarian Cancer Study[16]	MAY	Clinic based	In-person interview	2000–08
NC	North Carolina Ovarian Cancer Study†[17]	NCO	Population based	In-person interview	1999–2008
NH and eastern MA	New England Case-Control Study of Ovarian Cancer†[18]	NEC	Population based	In-person interview	1999–2008
Orange County and San Diego County, CA	University of California, Irvine Ovarian Cancer Study[19]	UCI	Population based	Self-completed questionnaire	1995–2005
Los Angeles County, CA	University of Southern California, Study of Lifestyle and Women's Health†[20]	USC	Population based	In-person interview	1993–2005

*Combined for the purpose of the analysis. †Data for timing of endometriosis available.

Table 1: Description of studies included in the analysis

Association between endometriosis and risk of histological subtypes of ovarian cancer: a pooled analysis of case–control studies

Celeste Leigh Pearce, Claire Templeman, Mary Anne Rossing, Alice Lee, Aimee M Near, Penelope M Webb, Christina M Nagle, Jennifer A Doherty, Kara L Cushing-Haugen, Kristine G Wicklund, Jenny Chang-Claude, Rebecca Hein, Galina Lurie, Lynne R Wilkens, Michael E Carney, Marc T Goodman, Kirsten Moysich, Susanne K Kjaer, Estrid Hogdall, Allan Jensen, Ellen L Goode, Brooke L Fridley, Melissa C Larson, Joellen M Schildkraut, Rachel T Palmieri, Daniel W Cramer, Kathryn L Terry, Allison F Vitonis, Linda J Titus, Argyrios Ziogas, Wendy Brewster, Hoda Anton-Culver, Alexandra Gentry-Maharaj, Susan J Ramus, A Rebecca Anderson, Doerthe Brueggmann, Peter A Fasching, Simon A Gayther, David G Huntsman, Usha Menon, Roberta B Ness, Malcolm C Pike, Harvey Risch, Anna H Wu, Andrew Berchuck, on behalf of the Ovarian Cancer Association Consortium

Summary

Background Endometriosis is a risk factor for epithelial ovarian cancer; however, whether this risk extends to all invasive histological subtypes or borderline tumours is not clear. We undertook an international collaborative study to assess the association between endometriosis and histological subtypes of ovarian cancer.

Methods Data from 13 ovarian cancer case–control studies, which were part of the Ovarian Cancer Association Consortium, were pooled and logistic regression analyses were undertaken to assess the association between self-reported endometriosis and risk of ovarian cancer. Analyses of invasive cases were done with respect to histological subtypes, grade, and stage, and analyses of borderline tumours by histological subtype. Age, ethnic origin, study site, parity, and duration of oral contraceptive use were included in all analytical models.

Findings 13 226 controls and 7911 women with invasive ovarian cancer were included in this analysis. 818 and 738, respectively, reported a history of endometriosis. 1907 women with borderline ovarian cancer were also included in the analysis, and 168 of these reported a history of endometriosis. Self-reported endometriosis was associated with a significantly increased risk of clear-cell (136 [2·2%] of 674 cases *vs* 818 [6·2%] of 13 226 controls, odds ratio 3·05, 95% CI 2·43–3·84, p<0·0001), low-grade serous (31 [9·2%] of 336 cases, 2·11, 1·39–3·20, p<0·0001), and endometrioid invasive ovarian cancers (169 [13·9%] of 1220 cases, 2·04, 1·67–2·48, p<0·0001). No association was noted between endometriosis and risk of mucinous (31 [6·0%] of 516 cases, 1·02, 0·69–1·50, p=0·93) or high-grade serous invasive ovarian cancer (261 [7·1%] of 3659 cases, 1·13, 0·97–1·32, p=0·13), or borderline tumours of either subtype (serous 103 [9·0%] of 1140 cases, 1·20, 0·95–1·52, p=0·12, and mucinous 65 [8·5%] of 767 cases, 1·12, 0·84–1·48, p=0·45).

Interpretation Clinicians should be aware of the increased risk of specific subtypes of ovarian cancer in women with endometriosis. Future efforts should focus on understanding the mechanisms that might lead to malignant transformation of endometriosis so as to help identify subsets of women at increased risk of ovarian cancer.

Funding Ovarian Cancer Research Fund, National Institutes of Health, California Cancer Research Program, California Department of Health Services, Lon V Smith Foundation, European Community's Seventh Framework Programme, German Federal Ministry of Education and Research of Germany, Programme of Clinical Biomedical Research, German Cancer Research Centre, Eve Appeal, Oak Foundation, UK National Institute of Health Research, National Health and Medical Research Council of Australia, US Army Medical Research and Materiel Command, Cancer Council Tasmania, Cancer Foundation of Western Australia, Mermaid 1, Danish Cancer Society, and Roswell Park Alliance Foundation.

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See Comment page 326

Department of Preventive Medicine (C L Pearce PhD, A Lee MPH, A R Anderson BS, Prof M C Pike PhD, Prof A H Wu PhD, S J Ramus PhD, Prof S A Gayther PhD) and Department of Gynecology, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA (C Templeman MD, D Brueggmann MD); Program in Epidemiology, Fred Hutchinson Cancer Research Center, Seattle, WA, USA (Prof M A Rossing PhD, J A Doherty PhD, K L Cushing-Haugen MS, K G Wicklund PhD); Cancer Control Program, Lombardi Comprehensive Cancer Center, Georgetown University Medical Center, Washington, DC, USA (A M Near MPH); Queensland Institute of Medical Research, Brisbane, QLD, Australia (P M Webb PhD, C M Nagle PhD); Division of Cancer Epidemiology, German Cancer Research Centre (DKFZ), Heidelberg, Germany (Prof J Chang-Claude PhD, R Hein PhD); Department of

	Crude		Stratified only		Stratified and adjusted	
	OR (95% CI)	p value	OR (95% CI)*	p value	OR (95% CI)†	p value
Invasive	1·49 (1·34–1·65)	<0·0001	1·53 (1·37–1·70)	<0·0001	1·46 (1·31–1·63)	<0·0001
Clear-cell	3·73 (3·04–4·58)	<0·0001	3·44 (2·78–4·27)	<0·0001	3·05 (2·43–3·84)	<0·0001
Endometrioid	2·32 (1·94–2·78)	<0·0001	2·20 (1·82–2·66)	<0·0001	2·04 (1·67–2·48)	<0·0001
Mucinous	1·09 (0·76–1·58)	0·63	1·04 (0·71–1·51)	0·86	1·02 (0·69–1·50)	0·93
High-grade serous	1·11 (0·96–1·29)	0·16	1·16 (1·00–1·35)	0·056	1·13 (0·97–1·32)	0·13
Low-grade serous	2·02 (1·38–2·97)	<0·0001	2·22 (1·48–3·31)	<0·0001	2·11 (1·39–3·20)	<0·0001
Borderline	1·26 (1·05–1·50)	0·012	1·19 (0·99–1·43)	0·062	1·12 (0·93–1·35)	0·24
Mucinous	1·27 (0·97–1·67)	0·078	1·19 (0·90–1·57)	0·23	1·12 (0·84–1·48)	0·45
Serous	1·31 (1·05–1·63)	0·015	1·28 (1·02–1·61)	0·034	1·20 (0·95–1·52)	0·12

OR=odds ratio. *Stratified by age (5 year categories), ethnic origin (non-Hispanic white, Hispanic white, black, Asian, and other). †Stratified by age (5 year categories), ethnic origin (non-Hispanic white, Hispanic white, black, Asian, and other), and adjusted for duration of oral contraceptive use (never, <2 years, 2–4·99 years, 5–9·99 years, ≥10 years), and parity (0, 1, 2, 3, ≥4 children).

Table 3: **Association between history of endometriosis and the histological subtypes of ovarian cancer**

	Crude		Stratified only		Stratified and adjusted	
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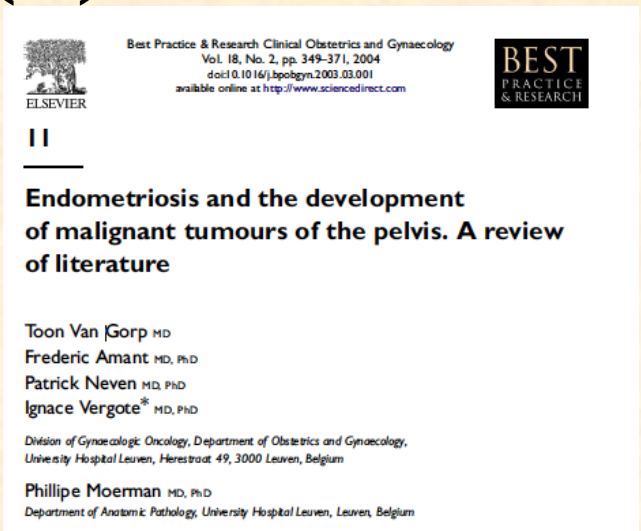
Table 3: **Association between history of endometriosis and the histological subtypes of ovarian cancer**

Association cancer ovarien et endométriose (3)

Analyse de 29 études:

- séreux de haut grade: 4.5%
- Mucineux: 1.4%
- **Endométrioïde: 19% (13-43%)**
- **Cellules Claires: 36% (11-70%)**



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BEST PRACTICE & RESEARCH

11

Endometriosis and the development of malignant tumours of the pelvis. A review of literature

Toon Van Gorp MD
Frederic Amant MD, PhD
Patrick Neven MD, PhD
Ignace Vergote* MD, PhD

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Department of Anatomic Pathology, University Hospital Leuven, Leuven, Belgium

Cancer associé à l'endométriose (EAOC)

Association cancer ovarien et endométriose (3)

Analyse de 29 études:

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- Mucineux: 1.4%
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International Journal of Gynecological Cancer:
January/February 2007 - Volume 17 - Issue 1 - p 37–43
Original Article

Risk of developing ovarian cancer among women with ovarian endometrioma: a cohort study in Shizuoka, Japan

KOBAYASHI, H.[*]; SUMIMOTO, K.[†]; MONIWA, N.[†]; IMAI, M.[‡]; TAKAKURA, K.[‡]; KUROMAKI, T.[‡]; MORIOKA, E.[‡]; ARISAWA, K.[‡]; TERAO, T.[†]

Cancer associé à l'endométriose (EAOC)

Transformation maligne: 0.7-1.6% (≈ 10 ans)

Association moléculaire cancer ovarien et endométriose (1)

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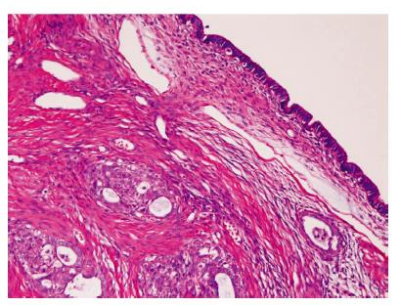
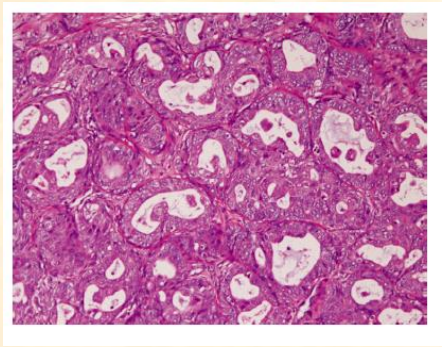


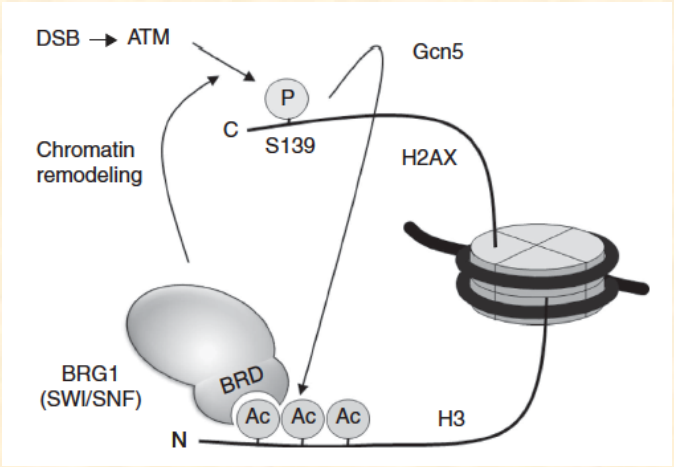


Endométrios
e « bénigne »

?????

EAOC

Association moléculaire cancer ovarien et endométriose (2)



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ARID1A Mutations in Endometriosis-Associated Ovarian Carcinomas

Kimberly C. Wiegand, B.Sc., Sohrab P. Shah, Ph.D., Osama M. Al-Agha, M.D., Yongjun Zhao, D.V.M., Kane Tse, B.Sc., Thomas Zeng, M.Sc., Janine Senz, B.Sc., Melissa K. McConechy, B.Sc., Michael S. Anglesio, Ph.D., Steve E. Kalloger, B.Sc., Winnie Yang, B.Sc., Alireza Heravi-Moussavi, Ph.D., Ryan Giuliany, B.Sc., Christine Chow, B.M.L.Sc., John Fee, B.Sc., Abdalnasser Zayed, B.Sc., Leah Prentice, Ph.D., Nataliya Melnyk, B.Sc., Gulisa Turashvili, M.D., Ph.D., Allen D. Delaney, Ph.D., Jason Madore, M.Sc., Stephen Yip, M.D., Ph.D., Andrew W. McPherson, B.A.Sc., Gavin Ha, B.Sc., Lynda Bell, R.T., Sian Fereday, B.Sc., Angela Tam, B.Sc., Laura Galletta, B.Sc., Patricia N. Tonin, Ph.D., Diane Provencher, M.D., Dianne Miller, M.D., Steven J.M. Jones, Ph.D., Richard A. Moore, Ph.D., Gregg B. Morin, Ph.D., Arusha Oloumi, Ph.D., Niki Boyd, Ph.D., Samuel A. Aparicio, B.M., B.Ch., Ph.D., Ie-Ming Shih, M.D., Ph.D., Anne-Marie Mes-Masson, Ph.D., David D. Bowtell, Ph.D., Martin Hirst, Ph.D., Blake Gilks, M.D., Marco A. Marra, Ph.D., and David G. Huntsman, M.D.

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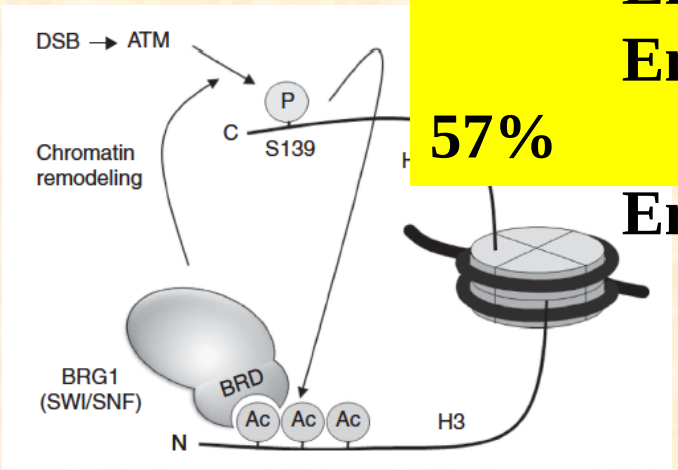
Association moléculaire cancer ovarien et endométriose (2)



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***ARID1A* Mutations in Endometriosis-Associated Ovarian Carcinomas**

Kimberly C. Wiegand, B.Sc., Sohrab P. Shah, Ph.D., Osama M. Al-Agha, M.D., Yongjun Melissa K. Winnie Yang, how, B.M.L.Sc., a Melnyk, B.Sc., Sc., Stephen Yip, , R.T., Sian in, Ph.D., Diane A. Moore, Ph.D., A. Aparicio, B.M., David D. Bowtell, David G.

M.S.A., S.E.K., G.H.), the Michael M., G.B.M., M.H., tic Pathology nd the University of British Columbia (K.C.W., D.M., G.B.M., S.A.A., B.G., M.A.M., B.G.H.) — all in Vancouver, Canada; and Simon Fraser University, Burnaby, BC (G.B.M., M.A.M.) — all in Canada; Centre de Recherche du Centre Hospitalier de l'Université de Montréal–Institut du Cancer de Montréal Hôpital Notre-Dame (J.M., D.P., A.-M.M.-M.), McGill University and the Research Institute of the McGill University Health Centre (P.N.T.), and Université de Montréal (D.P., A.-M.M.-M.) — all in Montreal; Peter MacCallum Cancer Centre (S.F., L.G., D.D.B.) and the University of Melbourne (D.D.B.) — both in Melbourne, VIC, Australia; and Johns Hopkins University School of Medicine, Baltimore (I.-M.S.)

ARID 1A Mutations

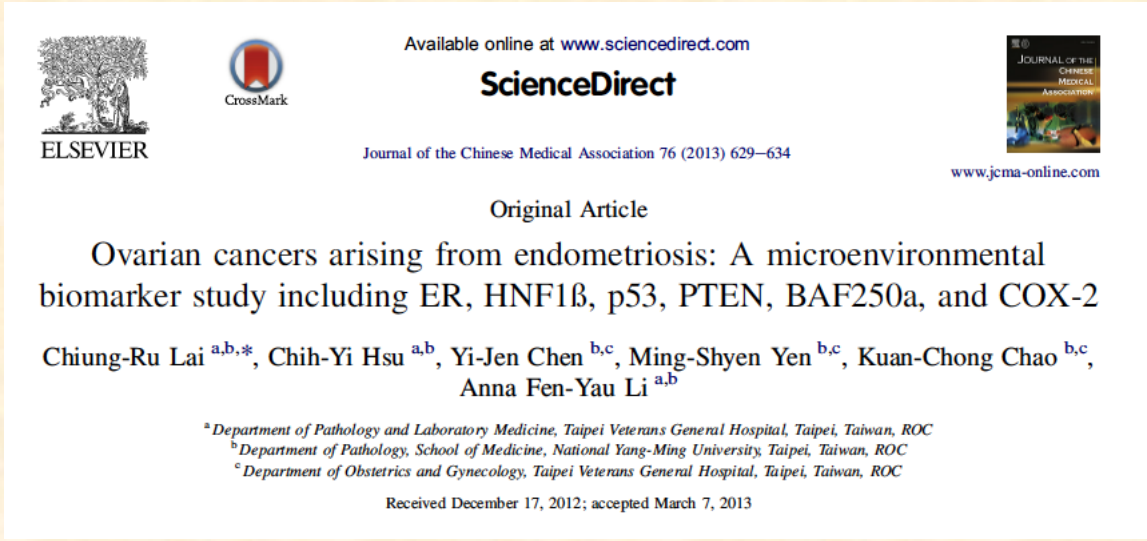
Clear cell : 41-57%

Endometrioid: 30-48%

Endométriose atypique: 38.5-57%

Endométriose: 5-15%

Association moléculaire cancer ovarien et endométriose (3)



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Original Article

Ovarian cancers arising from endometriosis: A microenvironmental biomarker study including ER, HNF1ß, p53, PTEN, BAF250a, and COX-2

Chiung-Ru Lai [a,b,*], Chih-Yi Hsu [a,b], Yi-Jen Chen [b,c], Ming-Shyen Yen [b,c], Kuan-Chong Chao [b,c], Anna Fen-Yau Li [a,b]

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Received December 17, 2012; accepted March 7, 2013

HNF-1β amplification

Clear cell : 65%

Endométriose atypique: 53%

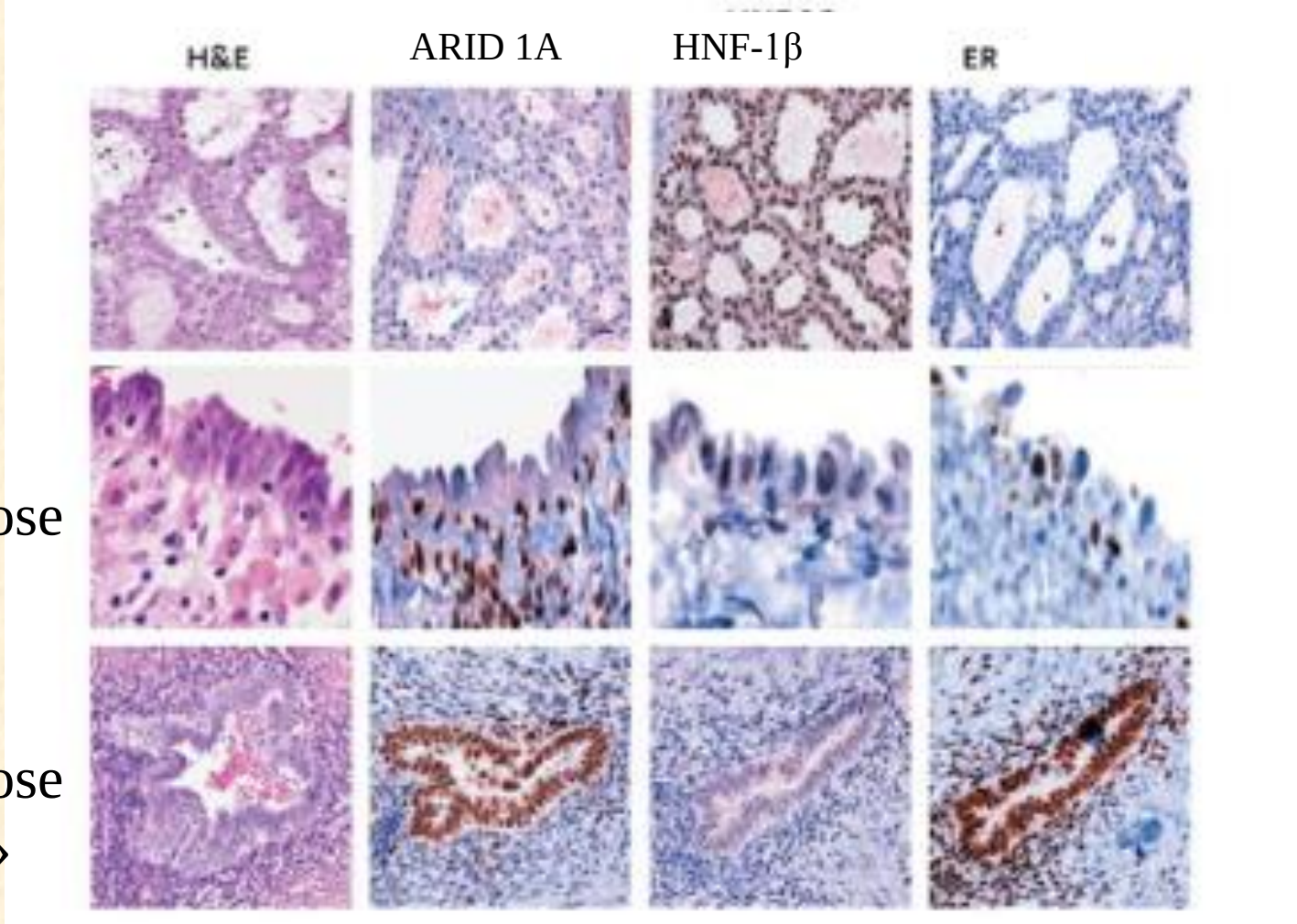
Endométriose: 33%

Association moléculaire cancer ovarien et endométriose (4)

Cellules claires

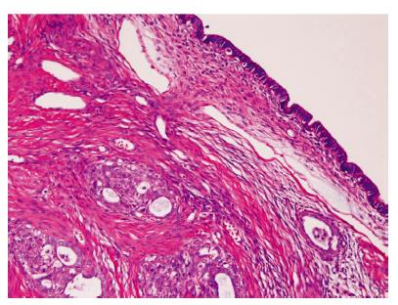
Endométriose atypique

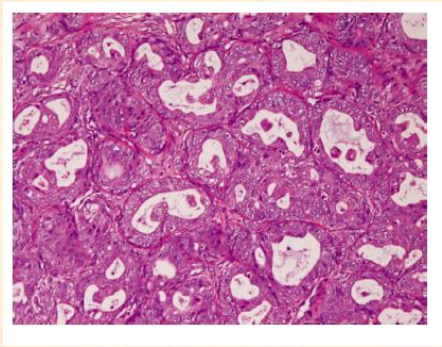
Endométriose « bénigne »



Association moléculaire cancer ovarien et endométriose (5)

Stress oxydatif,
inflammation chronique,
hypoxie…





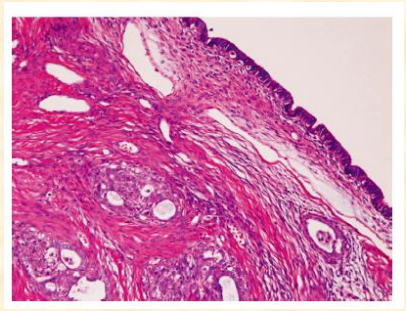
Endométrios
e « bénigne »

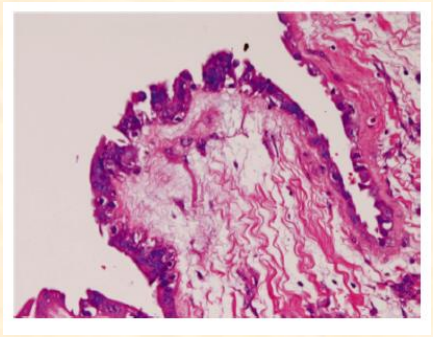
?????

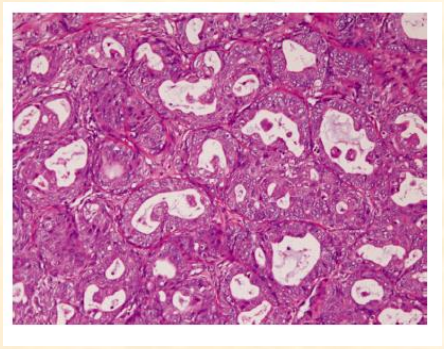
EAOC

Association moléculaire cancer ovarien et endométriose (5)

Stress oxydatif,
inflammation chronique,
hypoxie…







Endométrios
e « bénigne »

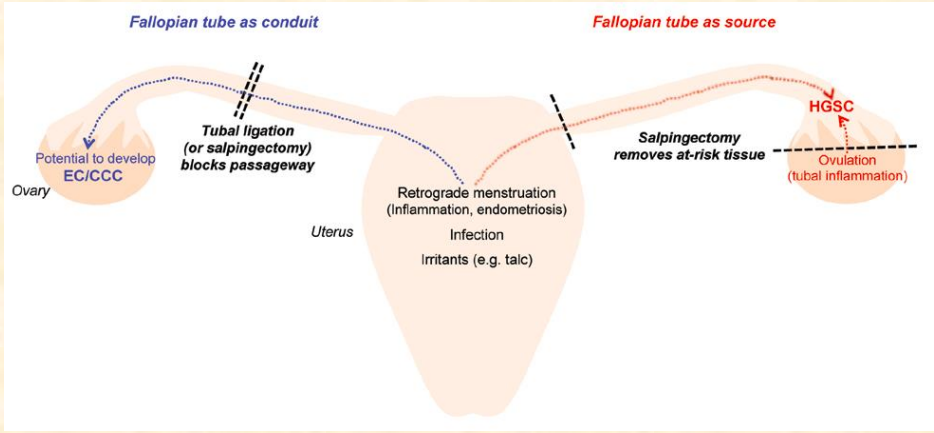
Endométriose atypique:
61-100% EAOC
1-2% endométriose « bénigne »

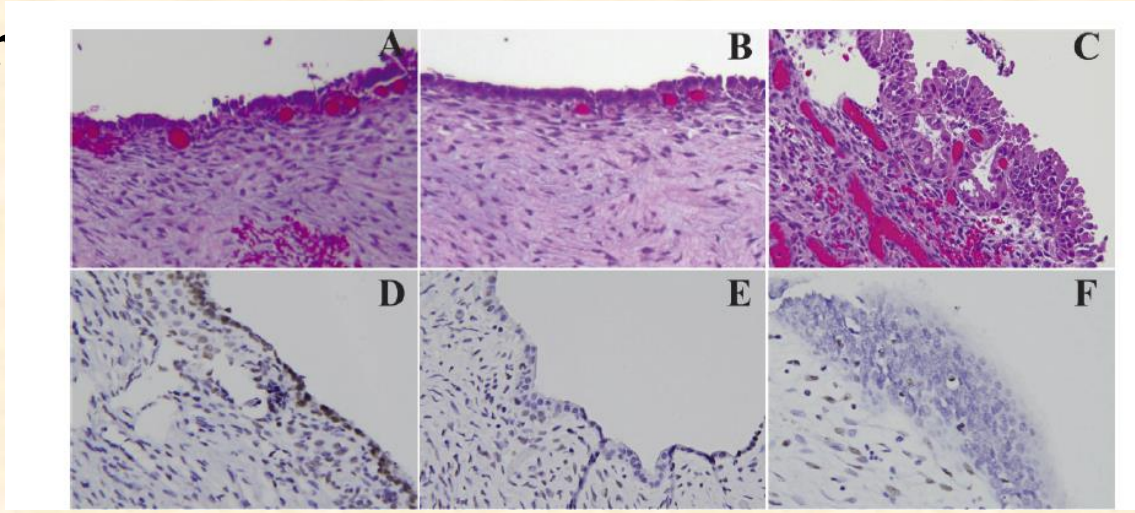
EAOC

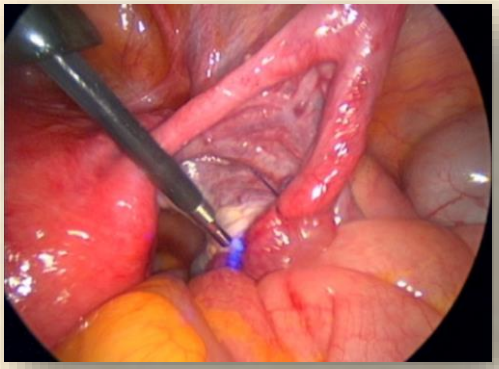
Lésion pré-cancéreuse ?

Implications et prévention

- **Contraception orale**
- **Ligature/ salpingectomie**
- **Analyse moléculaire des endométriomes ovar**
- **Biopsie optique**







Implications et prévention

- **Contraception orale**

Duration of oral contraceptive use (mean)	Cases/controls	RR and 99% FCI*
Never	14703/51908	1·00 (0·96–1·04)
Less than 1 year (0·4 years)	1492/6353	1·00 (0·91–1·10)
1–4 years (2·4 years)	2686/11329	0·78 (0·73–0·83)
5–9 years (6·8 years)	1562/7118	0·64 (0·59–0·69)
10–14 years (11·6 years)	655/3765	0·56 (0·50–0·62)
15 years or more (18·3 years)	247/1639	0·42 (0·36–0·49)

Numbers do not always add to the total, because of missing values. *Relative risks (RR) stratified by study, age, parity, and hysterectomy. Test for trend with duration of use, p<0·00001.

Table 2: Relative risk of ovarian cancer in users of oral contraceptives compared with never users, by duration of oral contraceptive use

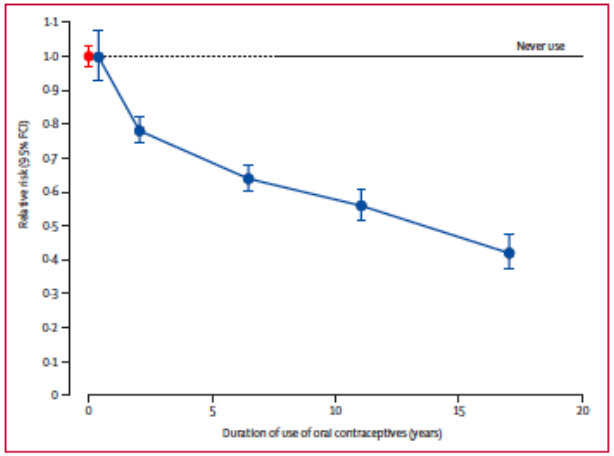


Figure 2: Relative risk* of ovarian cancer by duration of use of oral contraceptives
*Stratified by study, age, parity, and hysterectomy.

Ovarian cancer and oral contraceptives: collaborative reanalysis of data from 45 epidemiological studies including 23 257 women with ovarian cancer and 87 303 controls

*Collaborative Group on Epidemiological Studies of Ovarian Cancer**

Summary

Background Oral contraceptives were introduced almost 50 years ago, and over 100 million women currently use them. Oral contraceptives can reduce the risk of ovarian cancer, but the eventual public-health effects of this reduction will depend on how long the protection lasts after use ceases. We aimed to assess these effects.

Methods Individual data for 23 257 women with ovarian cancer (cases) and 87 303 without ovarian cancer (controls) from 45 epidemiological studies in 21 countries were checked and analysed centrally. The relative risk of ovarian cancer in relation to oral contraceptive use was estimated, stratifying by study, age, parity, and hysterectomy.

Findings Overall 7308 (31%) cases and 32 717 (37%) controls had ever used oral contraceptives, for average durations among users of 4·4 and 5·0 years, respectively. The median year of cancer diagnosis was 1993, when cases were aged an average of 56 years. The longer that women had used oral contraceptives, the greater the reduction in ovarian cancer risk ($p<0·0001$). This reduction in risk persisted for more than 30 years after oral contraceptive use had ceased but became somewhat attenuated over time—the proportional risk reductions per 5 years of use were 29% (95% CI 23–34%) for use that had ceased less than 10 years previously, 19% (14–24%) for use that had ceased 10–19 years previously, and 15% (9–21%) for use that had ceased 20–29 years previously. Use during the 1960s, 1970s, and 1980s was associated with similar proportional risk reductions, although typical oestrogen doses in the 1960s were more than double those in the 1980s. The incidence of mucinous tumours (12% of the total) seemed little affected by oral contraceptives, but otherwise the proportional risk reduction did not vary much between different histological types. In high-income countries, 10 years use of oral contraceptives was estimated to reduce ovarian cancer incidence before age 75 from 1·2 to 0·8 per 100 users and mortality from 0·7 to 0·5 per 100; for every 5000 woman-years of use, about two ovarian cancers and one death from the disease before age 75 are prevented.

Interpretation Use of oral contraceptives confers long-term protection against ovarian cancer. These findings suggest that oral contraceptives have already prevented some 200 000 ovarian cancers and 100 000 deaths from the disease, and that over the next few decades the number of cancers prevented will rise to at least 30 000 per year.

Lancet 2008; 371: 303–14

See Editorial page 275

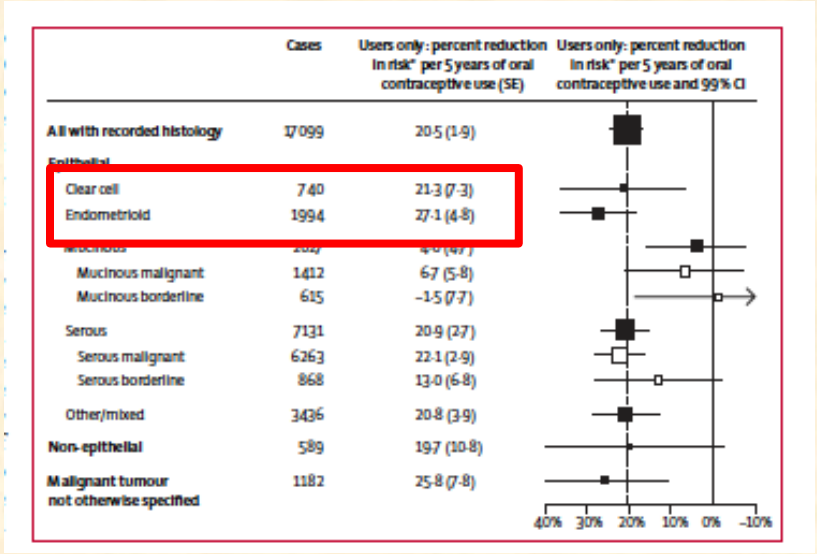
See Comment page 277

*Authors listed at end of paper

Correspondence to:
Secretariat, Cancer Research UK Epidemiology Unit, Richard Doll Building, Roosevelt Drive, Oxford OX3 7LF, UK
collaborations@ceu.ox.ac.uk

Implications et prévention

- **Contraception orale**



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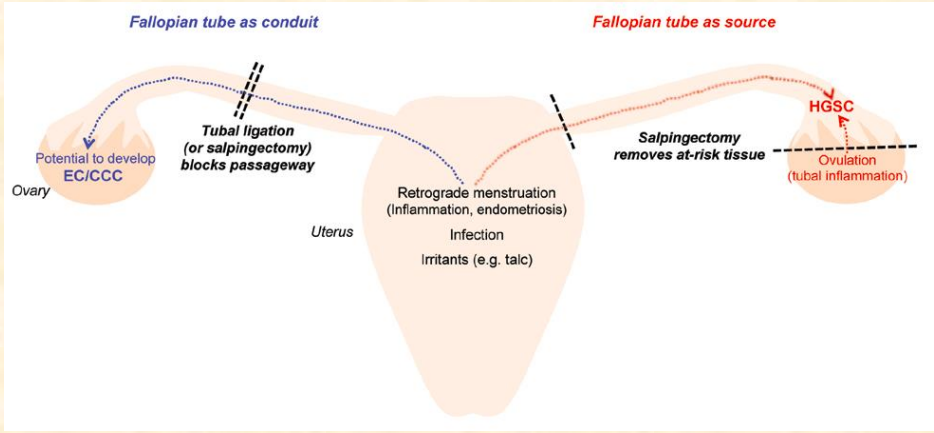
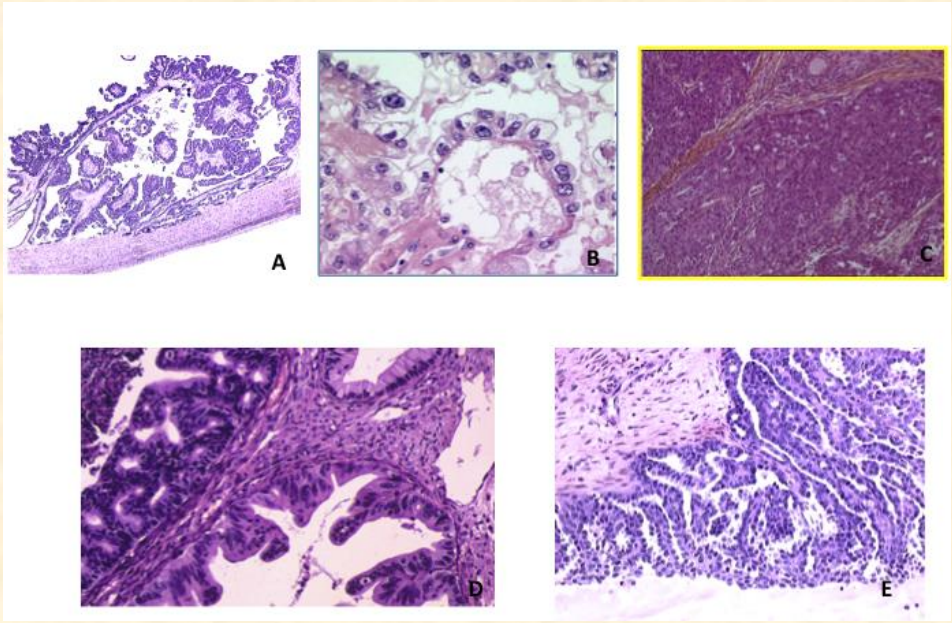
Implications et prévention

- **Ligature/ salpingectomie**



Tumeurs épithéliales de l'ovaire

- Fréquence des types histologiques
  - Séreux: 50-70%
    - Bas grade: 25%
    - Haut grade: 75%
  - Mucineux: 5%
  - **Endométrioïde: 10%**
  - **C Claires: 10%**



Les cancers de l'ovaire: une maladie hétérogène !!!

	Séreux de bas grade	Cellules claires	Endométrioïde	Mucineux	Séreux de haut grade
Signatures moléculaires	BRAF KRAS HER2	ARID1A HNF 1B PI3KCA PTEN	ARID1A PTEN B-catenine KRAS	KRAS HER2	TP53 BRCA
Instabilité génomique	faible	faible	faible	Faible	forte
Fréquence	5%	10%	10%	3%	70%
Précurseurs	Borderline séreux	Endometriose Adénofibrome	Endométriose Adénofibrome	Séquence: Adenome/ Borderline	-Trompe de Fallope (STIC) -Ovaire (dysplasia)
Pronostic	Favorable	Intermédiaire	Favorable	Favorable	Mauvais
Réponse initiale à la chimiothérapie à base de sels de platine	Intermédiaire (20-30%)	Chimiorésistance (15%)	Bonne	Chimiorésistance (15-20%)	Bonne (80%)
Cibles thérapeutiques potentielles	Inhibiteurs de BRAF Inhibiteurs de MEK	Inhibiteurs de PI3K	Inhibiteurs de mTOR	Inhibiteurs de MEK	Inhibiteurs de PARP Inhibiteurs du cycle cellulaire

Synthèse
General review

Bulletin du Cancer Volume 100 • N° 7-8 • juillet-août 2013
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L'odyssée de la trompe de Fallope : de l'ovaire à la trompe. À propos du cancer séreux de haut grade de l'ovaire

The Fallopian tube odyssey: from the ovary to the tube. About high-grade serous ovarian carcinoma

Gautier Chêne[1,2,3], Jacques Dauplat[2], Nina Radosevic-Robin[1], Anne Cayre[1], Frédérique Penault-Llorca[1]
[1] Centre Jean-Perrin, département d'anatomie et de cytologie pathologiques, 58, rue Montalembert, 63000 Clermont-Ferrand, France
[2] Centre Jean-Perrin, département de chirurgie, 58, rue Montalembert, 63000 Clermont-Ferrand, France
[3] CHU de Saint-Étienne, département de gynécologie, 42000 Saint-Etienne France
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Tirés à part : G. Chêne

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Critical Reviews in Oncology/Hematology xxx (2013) xxx–xxx

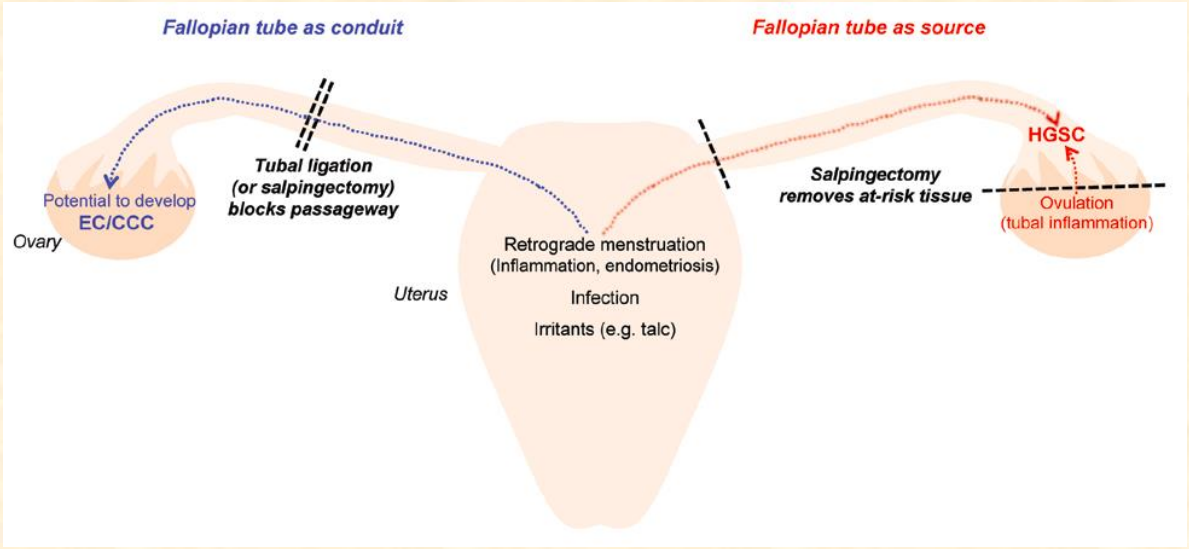
CRITICAL REVIEWS IN Oncology Hematology Incorporating Geriatric Oncology

www.elsevier.com/locate/critrevonc

Tu-be or not tu-be: That is the question. . . About serous ovarian carcinogenesis

G. Chene[a,b,c,*], J. Dauplat[b], N. Radosevic-Robin[a], A. Cayre[a], F. Penault-Llorca[a]
[a] Department of Histopathology, Centre Jean Perrin, Clermont-Ferrand, France
[b] Department of Surgery, Centre Jean Perrin, Clermont-Ferrand, France
[c] Department of Obstetrics and Gynecology, CHU St Etienne, France

Accepted 6 March 2013



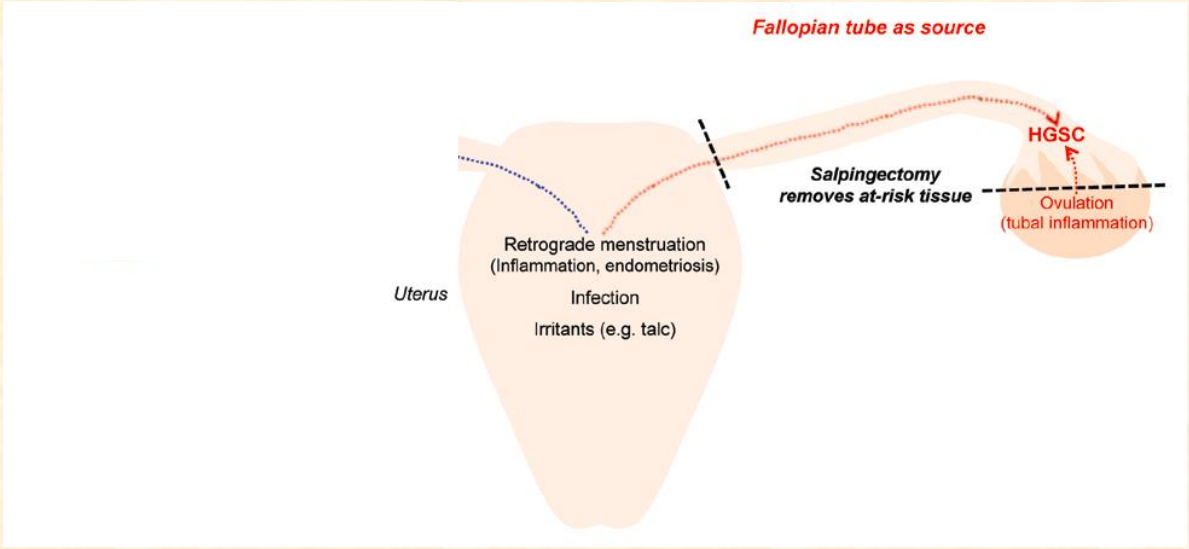
DÉCLARATION DE LA GOC

CONCERNANT LA SALPINGECTOMIE ET LA PRÉVENTION DU CANCER DE L'OVAIRE

Recommandations de la GOC :

- **En raison de son utilisation possible en tant que méthode de prévention du cancer, on recommande aux médecins de discuter des risques et des avantages de la salpingectomie bilatérale avec les patientes devant subir une hystérectomie ou faisant la demande d'une méthode contraceptive permanente et irréversible.**

- **Étant donné que l'ensemble des avantages et des risques de ce changement de pratique n'a pas encore été déterminé, la GOC s'est donné comme priorité la mise en place d'une étude nationale sur la prévention du cancer de l'ovaire, mettant l'accent sur l'ablation des trompes de Fallope.**



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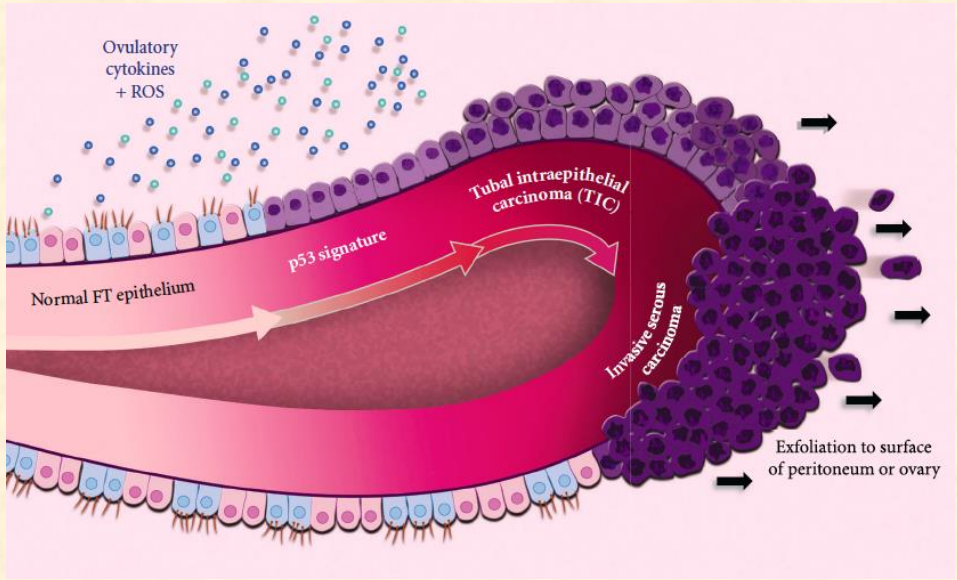
JMIG The Journal of Minimally Invasive Gynecology

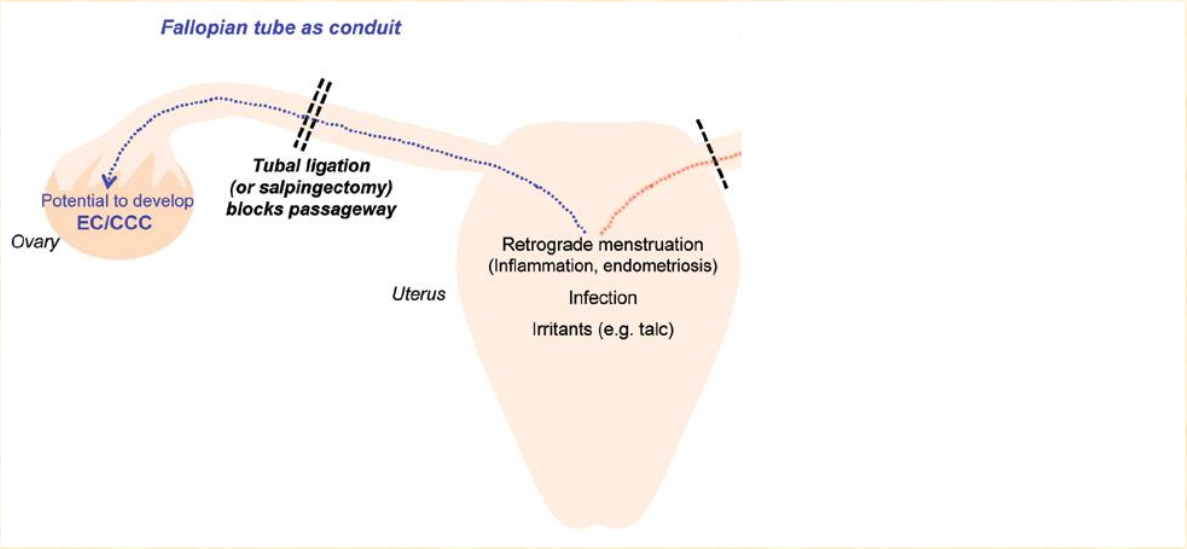
Review Article

Surgical Implications of the Potential New Tubal Pathway for Ovarian Carcinogenesis

Gautier Chene, MD*, Kourosh Rahimi, MD, Ann-Marie Mes-Masson, MD, and Diane Provencher, MD

From the Research Centre of the University of Montreal Hospital Centre (CRCHUM), Montreal Cancer Institute (Drs. Chene, Mes-Masson, and Provencher), and Department of Pathology, CHU Montreal (Dr. Rahimi), Montreal, Quebec, Canada.





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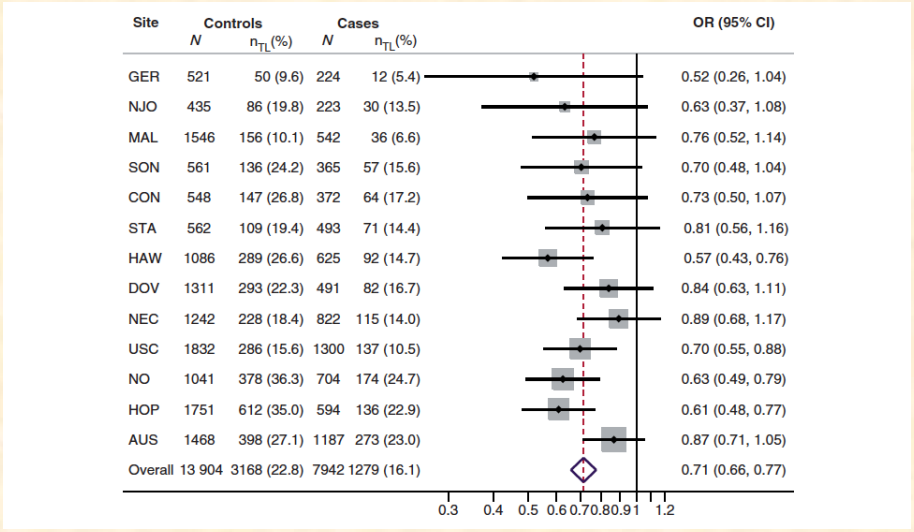
International Journal of Epidemiology 2013;**42**:579–589
doi:10.1093/ije/dyt042

OTHER ORIGINAL ARTICLES

Tubal ligation and risk of ovarian cancer subtypes: a pooled analysis of case-control studies

Weiva Sieh,[1]* Shannon Salvador,[2] Valerie McGuire,[1] Rachel Palmieri Weber,[3] Kathryn L Terry,[4] Mary Anne Rossing,[5] Harvey Risch,[6] Anna H Wu,[7] Penelope M Webb,[8] Kirsten Moysich,[9] Jennifer A Doherty,[10] Anna Felberg,[1] Dianne Miller,[2] Susan J Jordan,[8] Australian Cancer Study (Ovarian Cancer), Australian Ovarian Cancer Study Group, Marc T Goodman,[11] Galina Lurie,[11] Jenny Chang-Claude,[12] Anja Rudolph,[12] Susanne Krüger Kjær,[13,14] Allan Jensen,[13] Estrid Høgdall,[13,15] Elisa V Bandera,[16] Sara H Olson,[17] Melony G King,[16] Lorna Rodriguez-Rodriguez,[16] Lambertus A Kiemeney,[18] Tamara Marees,[18] Leon F Massuger,[19] Anne M van Altena,[19] Roberta B Ness,[20] Daniel W Cramer,[4] Malcolm C Pike,[7,17] Celeste Leigh Pearce,[7] Andrew Berchuck,[21] Joellen M Schildkraut[3] and Alice S Whittemore[1] on behalf of the Ovarian Cancer Association Consortium

Ligature tubaire (1)



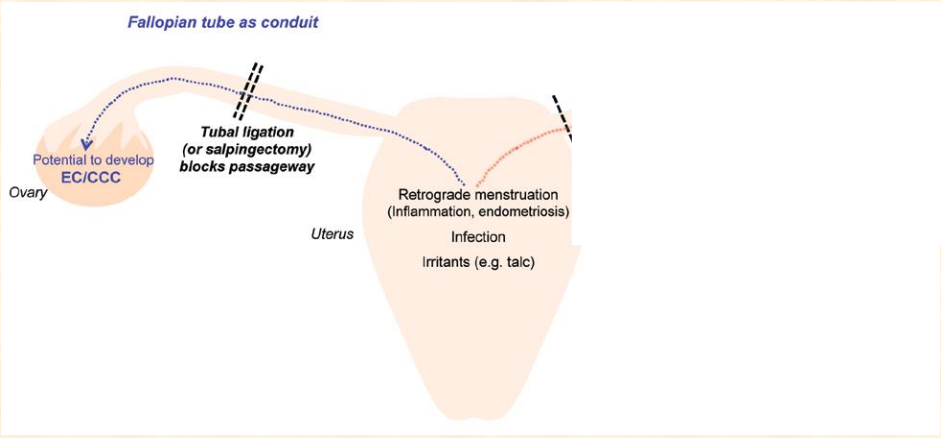


Table 3 Subtype-specific risk of invasive and borderline ovarian tumours associated with tubal ligation

Tumour behaviour, histology	Cases *N*	Prior tubal ligation *N* (%)	OR[a] (95% CI)
Invasive			
Serous	4777	893 (18.7)	0.81 (0.74-0.89)
High-grade[b]	3791	729 (19.2)	0.80 (0.73-0.89)
Low-grade[b]	361	58 (16.1)	0.89 (0.65-1.22)
Mucinous	574	77 (13.4)	0.68 (0.52-0.89)
Endometrioid	1273	138 (10.8)	0.48 (0.40-0.59)
Clear cell	737	81 (11.0)	0.52 (0.40-0.67)
Borderline[c]			
Serous	1309	233 (17.8)	0.98 (0.83-1.16)
Mucinous	906	161 (17.8)	1.01 (0.83-1.23)
Serous/mucinous	2215	394 (17.8)	0.98 (0.86-1.12)

Ligature tubaire (2)

Human Reproduction Update, Vol.17, No.1 pp. 55–67, 2011
Advanced Access publication on July 15, 2010 doi:10.1093/humupd/dmq030

human reproduction update

Tubal ligation and the risk of ovarian cancer: review and meta-analysis

D. Cibula [1,*], M. Widschwendter [2], O. Májek [3], and L. Dusek [3]

[1]Oncogynecological Centre, Department of Obstetrics and Gynecology, General Teaching Hospital, First Medical School, Charles University, Prague, Czech Republic [2]Department of Gynecological Oncology, Institute for Women's Health, University College London, London, UK [3]Institute of Biostatistics and Analyses, Masaryk University, Brno, Czech Republic

*Correspondence address. Oncogynecological Centre, Department of Obstetrics and Gynecology, General Teaching Hospital in Prague, First Medical School, Charles University, Apolinarska 18, Prague 2, Czech Republic. Tel: +420-603547055; Fax: +420-224967451, E-mail: david.cibula@iol.cz

Submitted on February 7, 2010; resubmitted on May 10, 2010; accepted on June 16, 2010

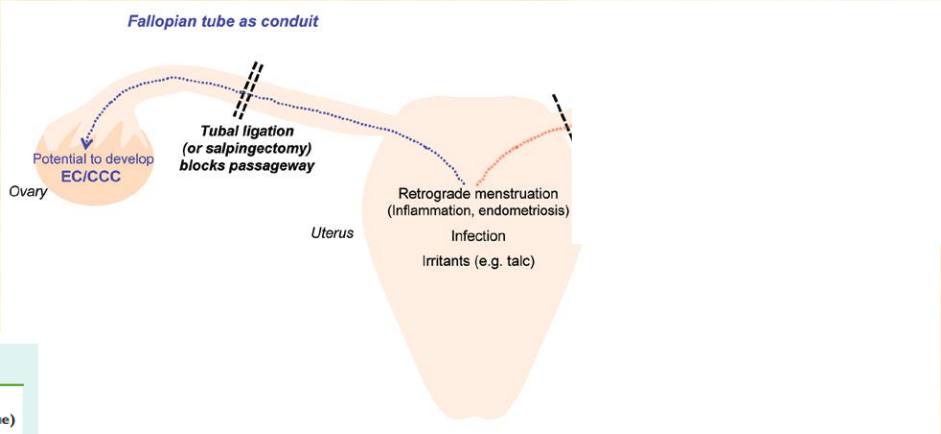
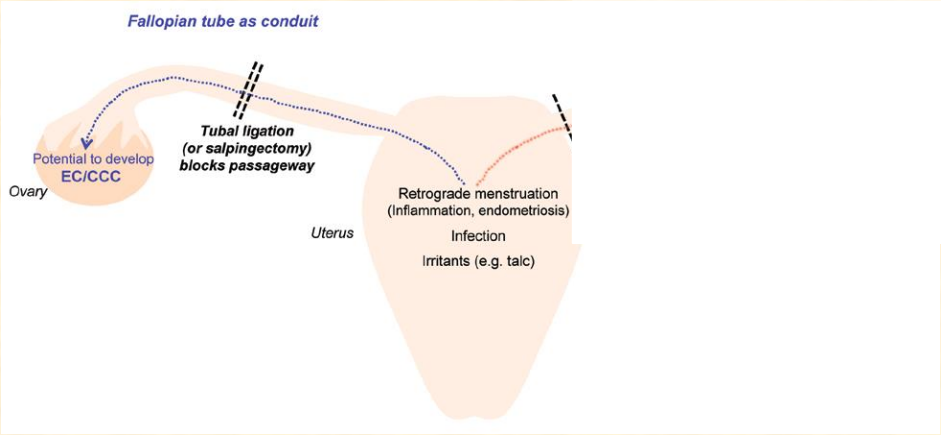


Table VI Risk of OC after TL: final outcomes of performed meta-analyzes.

Analysis/outcome	Outcome of meta-analysis RR: relative risk			Comparison of subgroups (*P*-value)
	RR (95% CI)	*P*-value	Heterogeneity test: *P*-value	
Main analysis				
OC after TL/all studies/	0.69 (0.64–0.75)	<0.001	0.039	-
OC after TL/strict selection/	0.66 (0.60–0.73)	<0.001	0.317	0.125[2]
OC after TL/extended selection/	0.74 (0.66–0.84)	<0.001	0.023	
Subgroups				
Years since TL				
0–4	0.69 (0.51–0.93)	0.015	0.524	0.590
5–9	0.82 (0.65–1.05)	0.113	0.074	
10–14	0.65 (0.44–0.97)	0.034	0.815	
15–19	0.88 (0.58–1.35)	0.573	0.245	
Tumor type				
Invasive	0.68 (0.61–0.75)	<0.001	0.460	0.096
Borderline	0.86 (0.67–1.10)	0.227	0.679	
Histology of invasive OC				
Serous	0.73 (0.63–0.85)	<0.001	0.133	<0.001[2]
Mucinous	0.92 (0.66–1.30)	0.653	0.012	
Endometrioid	0.40 (0.30–0.53)	<0.001	0.412*	
BRCA1/2 mutation				
BRCA1	0.69 (0.53–0.89)	0.004	0.098	0.849[2]
BRCA2	0.73 (0.42–1.24)	0.243	0.333	

[1]CI: confidence interval.
[2]Statistically significant heterogeneity was observed within one of subgroups.
*RR for endometrioid tumors is significantly lower ($P < 0.001$) than RR for serous and mucinous tumors.

Ligature tubaire ou salpingectomie prophylactique ?



human reproduction OPINION

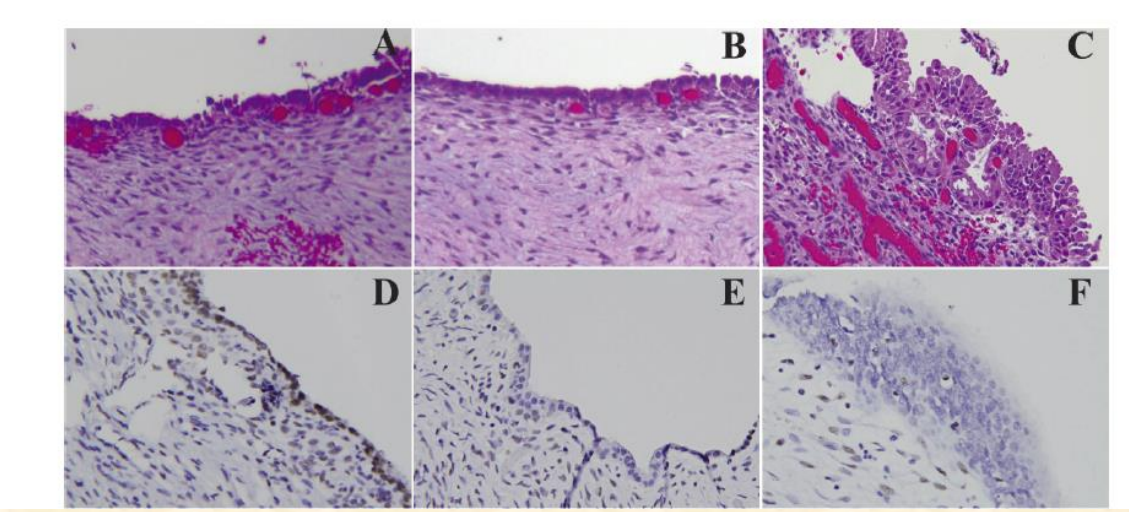
The 'incessant menstruation' hypothesis: a mechanistic ovarian cancer model with implications for prevention

Paolo Vercellini[1,2,*], Piergiorgio Crosignani[1], Edgardo Somigliana[1,2], Paola Viganò[2,3], Laura Buggio[1], Giorgio Bolis[1], and Luigi Fedele[1]

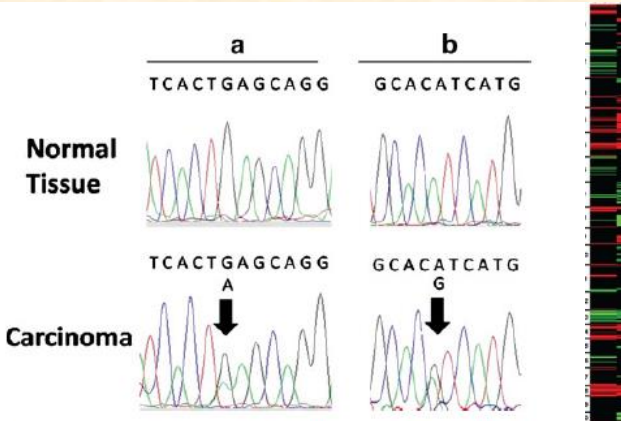
[1]Clinica Ostetrica e Ginecologica, Istituto 'Luigi Mangiagalli', Università Statale di Milano, Fondazione IRCCS 'Ca' Granda'—Ospedale Maggiore Policlinico, Via Commenda, 12, 20122 Milan, Italy [2]Center for Research in Obstetrics and Gynaecology (C.R.O.G.), Milan, Italy [3]Division of Gynaecology, Ospedale San Raffaele, Milan, Italy

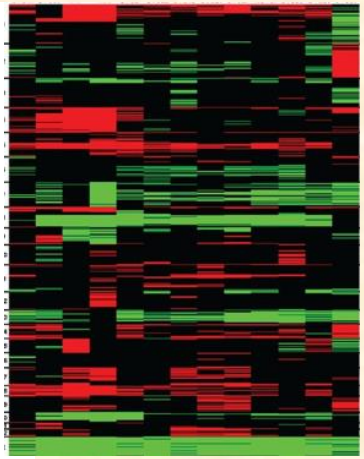
Implications et prévention

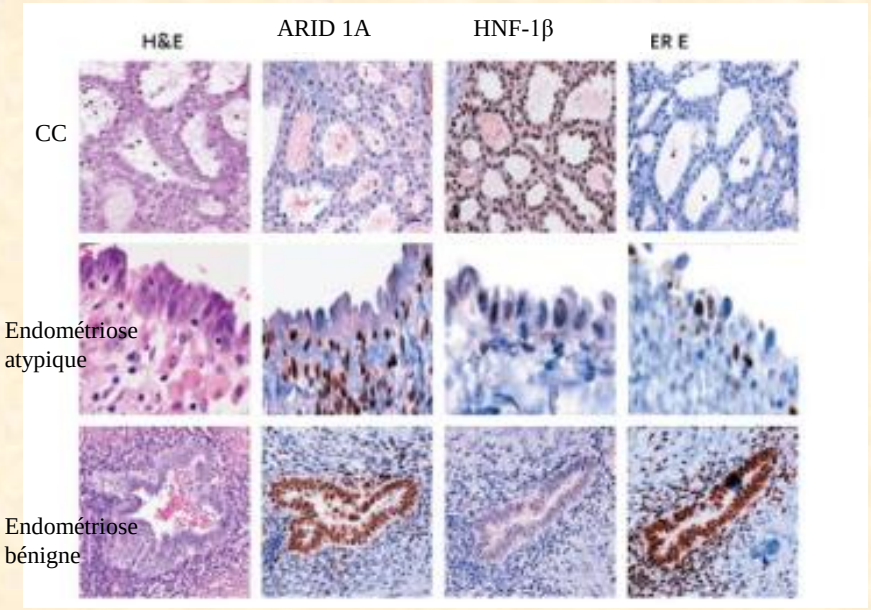
- **Analyse moléculaire des endométriomes ovariens**



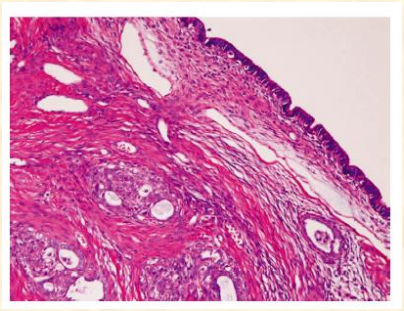
Analyse moléculaire

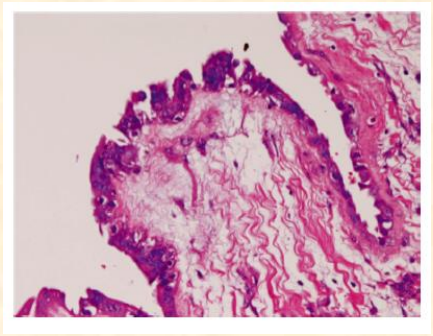


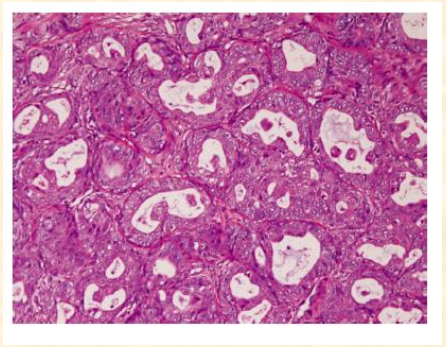




PTEN, PI3KCA, MET, ARID1A mutations
Amplification HNF-1β
LOH…





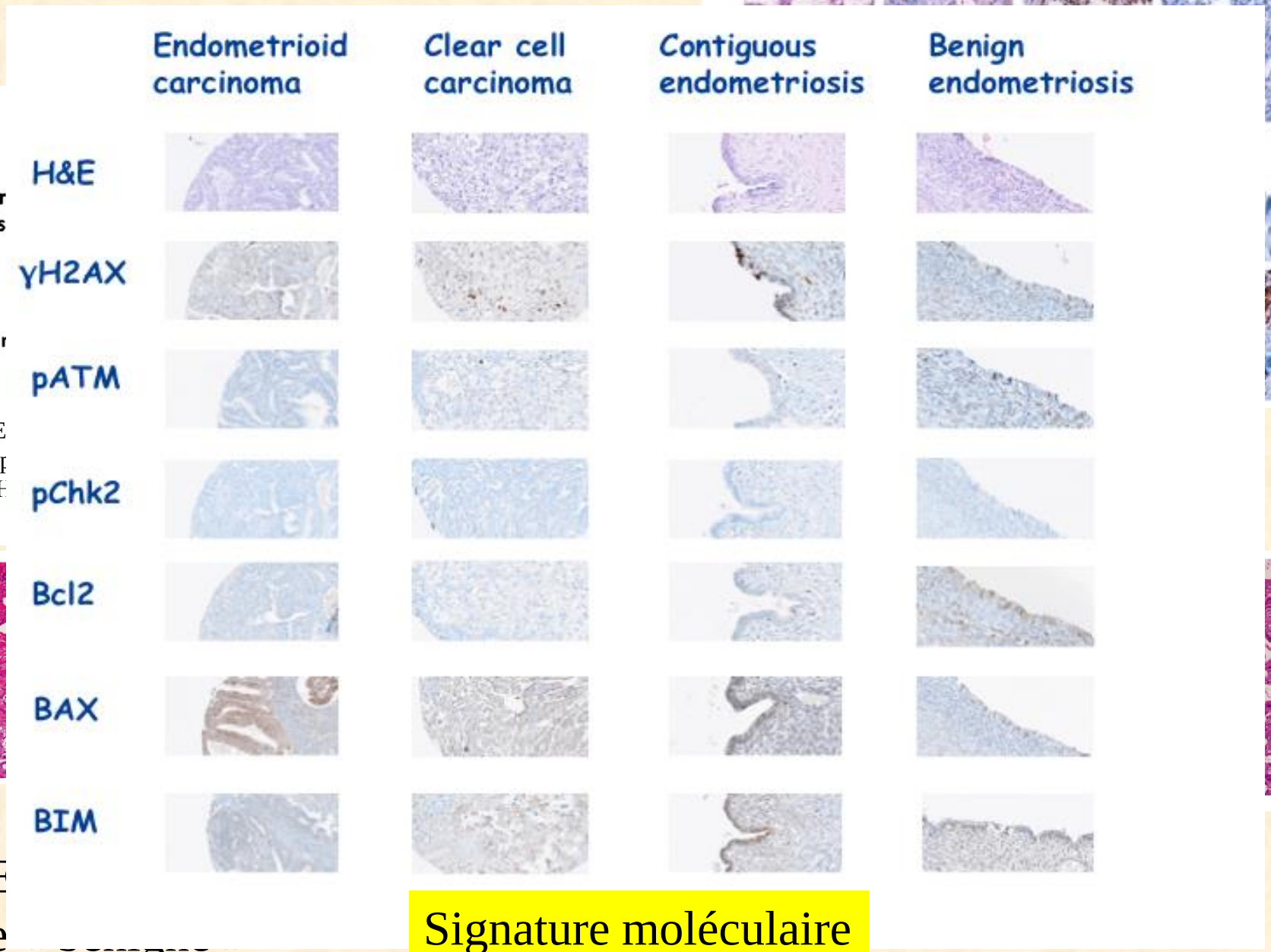


Endométrios e « bénigne »

Endométriose atypique

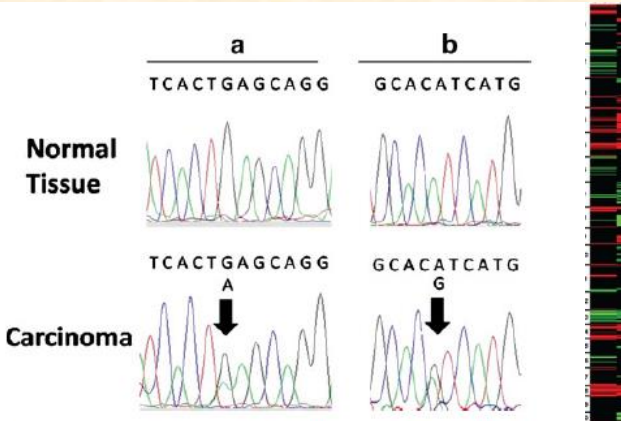
EAOC

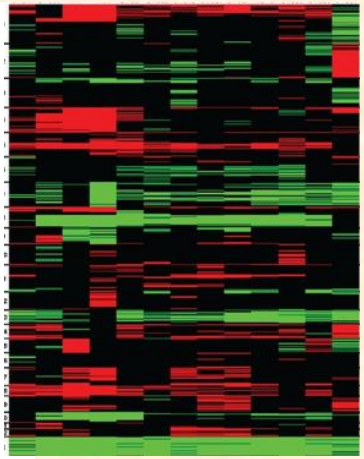
Analyse moléculaire

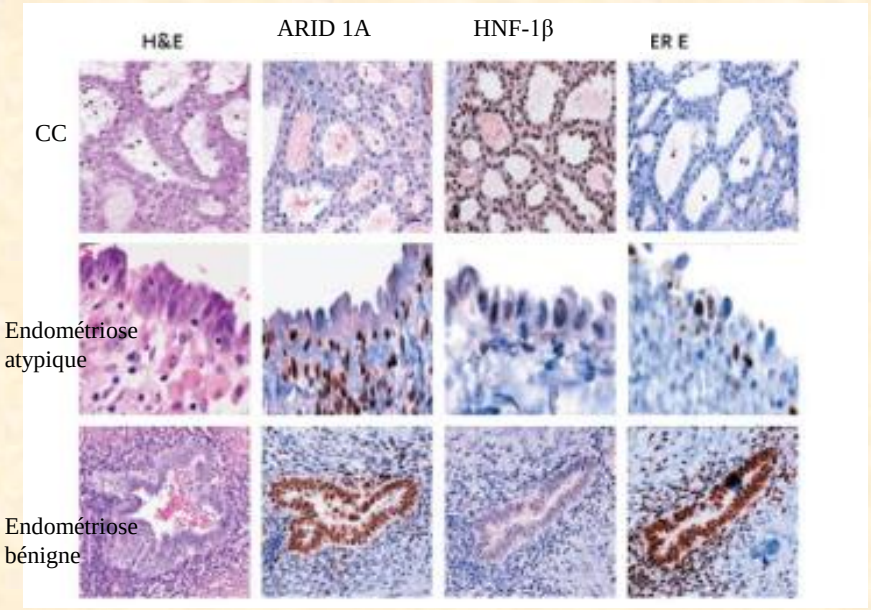


Signature moléculaire

Analyse moléculaire







PTEN, PI3KCA, MET, ARID1A mutations
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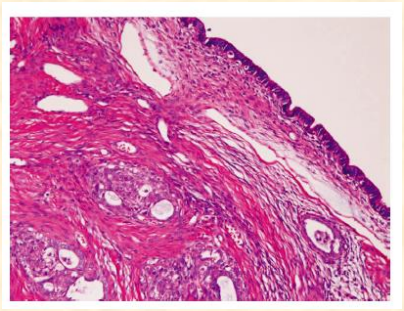
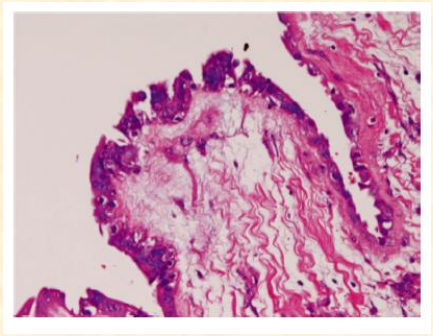
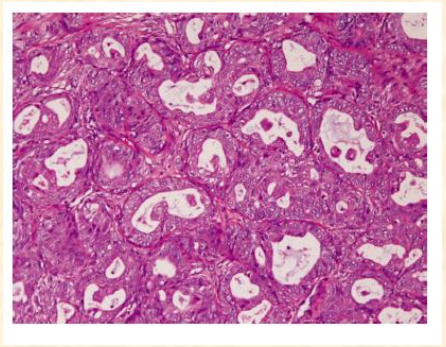






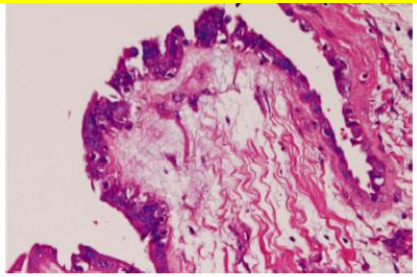
Endométrios e « bénigne »

Endométriose atypique

EAOC

Analyse moléculaire

Lésion à risque ???? Quelle surveillance ????



Endométriose
atypique

Analyse moléculaire

MODERN PATHOLOGY (2012) 25, 885–892

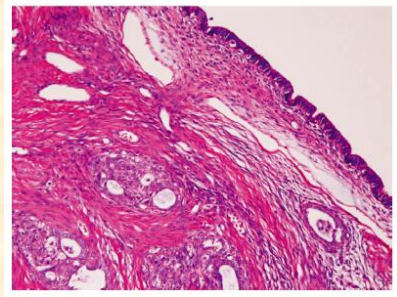
Loss of *ARID1A*/BAF250a-expression in endometriosis: a biomarker for risk of carcinogenic transformation?

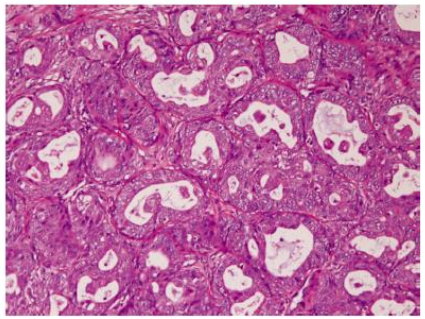
Eleftherios P Samartzis[1], Nicolas Samartzis[1], Aurelia Noske[2], André Fedier[1], Rosmarie Caduff[2], Konstantin J Dedes[1], Daniel Fink[1] and Patrick Imesch[1]

[1]*Department of Gynecology, University Hospital Zurich, Zurich, Switzerland and* [2]*Department of Pathology, University Hospital Zurich, Zurich, Switzerland*

Mutations of the tumor-suppressor gene *ARID1A* result in the loss of protein expression of the BRG-associated factor 250a (BAF250a), a large subunit of transcription-regulating Human SWI/SNF complexes, which have an important role in the control of cell proliferation and tumor suppression. *ARID1A* mutations are particularly frequent in endometriosis-associated ovarian clear cell and endometrioid carcinomas, and were recently described as a possible key mechanism and early step in the transformation of endometriosis into cancer. Here, we examined the immunohistochemical expression pattern of BAF250a in a tissue microarray including 74 endometriosis and 30 endometrium samples. Ovarian cancer samples ($n=136$) served as a control. Epithelial BAF250a expression was assessable in 90/104 (87%) and stromal BAF250a expression in 95/104 (91%) of the endometriosis, and endometrium cases due to lack of adequate tissue in some spots. Complete lack of BAF250a expression was observed in three endometriomas ($n=3/20$, 15%) and one deep-infiltrating endometriosis sample ($n=1/22$, 5%), but in none of the peritoneal endometriosis ($n=0/16$) and eutopic endometrium samples ($n=0/30$). A comparison of the mean immunoreactivity scores revealed a significantly lower expression rate of BAF250a in endometriomas compared with normal endometrium ($P<0.0005$), as well as peritoneal ($P=0.003$) and deep-infiltrating endometriosis ($P=0.02$). Our data demonstrates that a complete loss of BAF250a expression is observable in some endometriotic lesions, especially in endometriomas. In addition, we report that a partial loss of BAF250a expression is occurring in the form of cell clusters indicating a clonal loss of BAF250a expression in these cells. The loss of expression of the tumor-suppressor protein BAF250a in some endometriomas possibly indicates a risk of

Modern Pathology (2012) **25**, 885–892; doi:10.1038/m





Endométrios
e « bénigne »

Stress oxydatif,
inflammation chronique,
hypoxie…

EAOC

Journal of Pathology
J Pathol 2015; **236:** 201–209
Published online 23 March 2015 in Wiley Online Library
(wileyonlinelibrary.com) DOI: 10.1002/path.4516

ORIGINAL PAPER

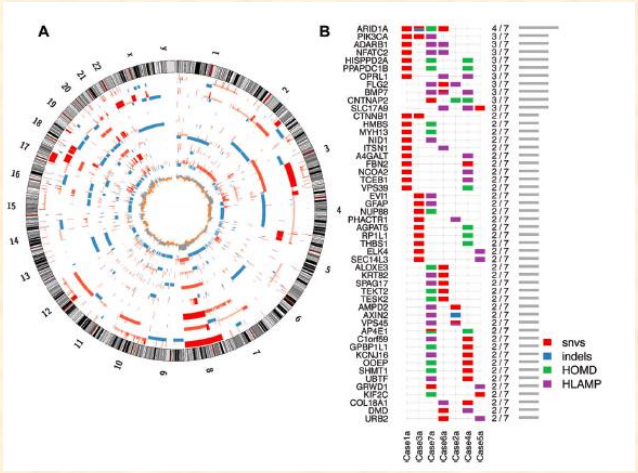
Multifocal endometriotic lesions associated with cancer are clonal and carry a high mutation burden

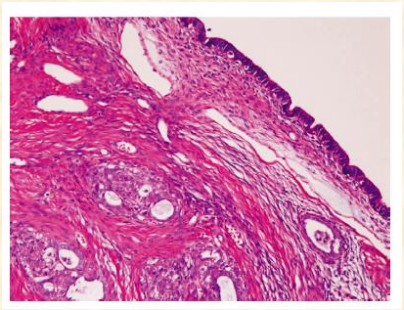
Michael S Anglesio,[1,†] Ali Bashashati,[2,†] Yi Kan Wang,[2,†] Janine Senz,[1] Gavin Ha,[2] Winnie Yang,[2] Mohamed R Aniba,[2] Leah M Prentice,[2] Hossein Farahani,[2] Hector Li Chang,[1] Anthony N Karnezis,[1] Marco A Marra,[3] Paul J Yong,[4] Martin Hirst,[3,5] Blake Gilks,[1,6] Sohrab P Shah[1,2] and David G Huntsman[1,2,4*]

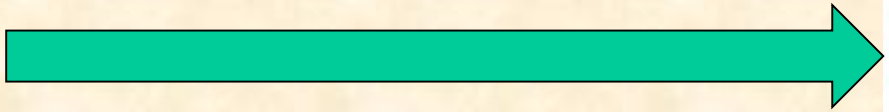
[1] *Department of Pathology and Laboratory Medicine, University of British Columbia, Vancouver, Canada*
[2] *Department of Molecular Oncology, British Columbia Cancer Agency Research Center, Vancouver, Canada*
[3] *Michael Smith Genome Science Centre, British Columbia Cancer Agency Research Center, Vancouver, Canada*
[4] *Department of Obstetrics and Gynaecology, University of British Columbia, Vancouver, Canada*
[5] *Department of Microbiology and Immunology, University of British Columbia, Vancouver, Canada*
[6] *Department of Anatomical Pathology, Vancouver General Hospital, Vancouver, Canada*

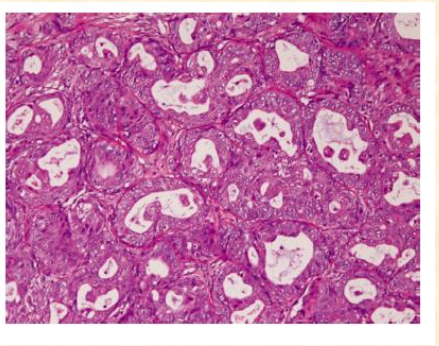
Analyse moléculaire:

➔Plusieurs types moléculaires d'endométriose





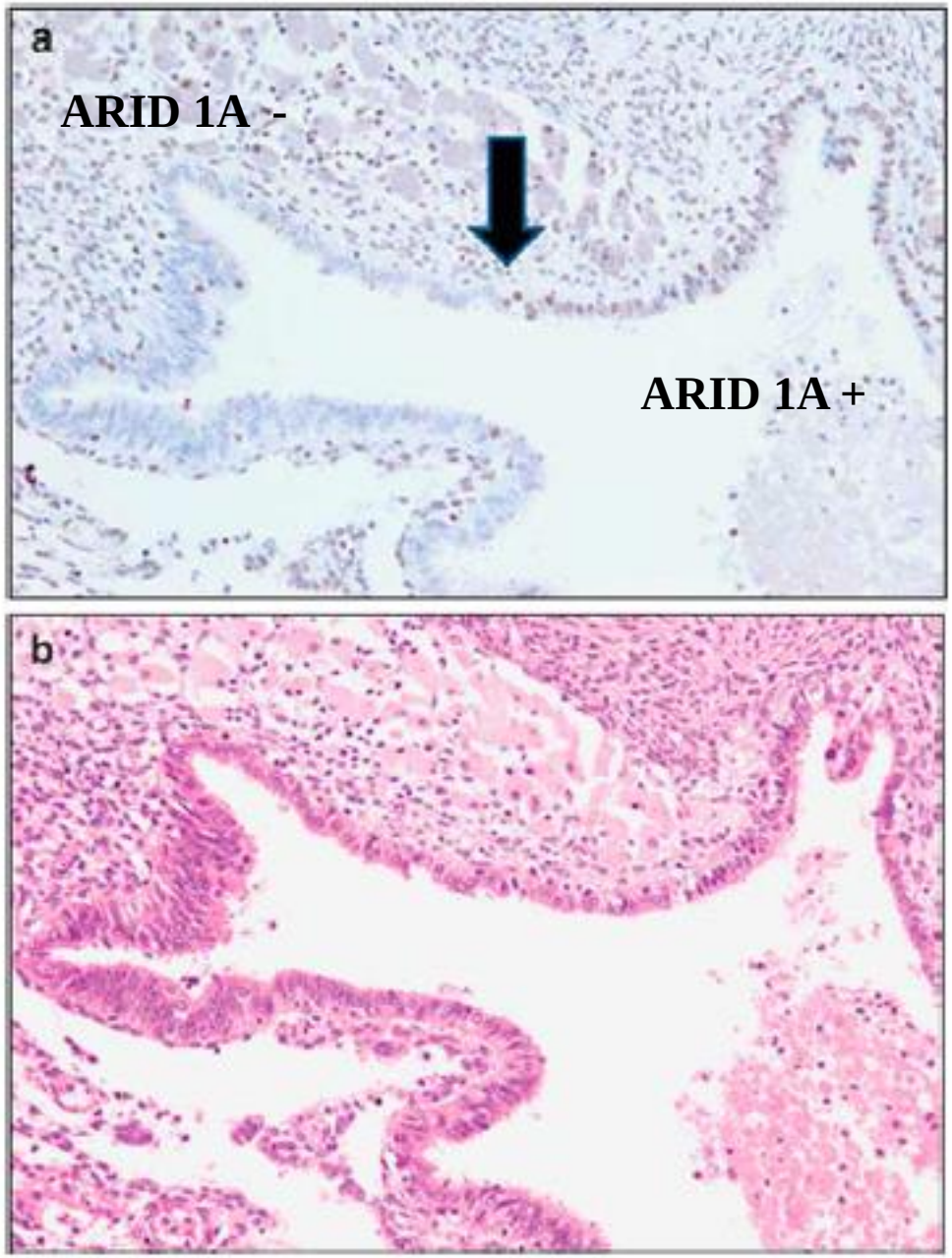


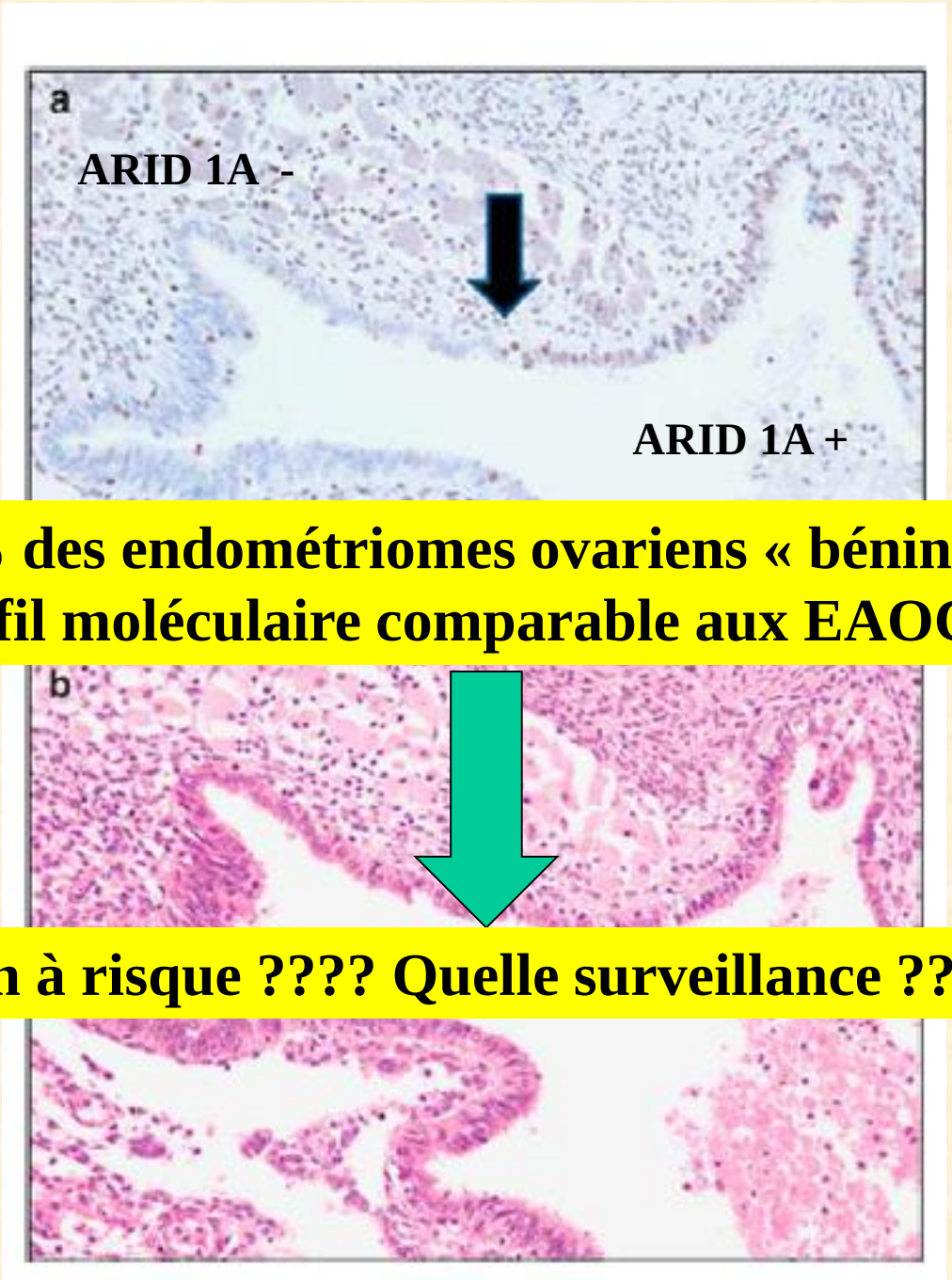


Endométrios e « bénigne »

Stress oxydatif, inflammation chronique, hypoxie…

EAOC


a
ARID 1A -
ARID 1A +
b



5-15 % des endométriomes ovariens « bénins » ont un profil moléculaire comparable aux EAOC

Lésion à risque ???? Quelle surveillance ????

Analyse moléculaire:

➔Thérapies ciblées

Therapeutics, Targets, and Chemical Biology

Cancer Research

A Synthetic Lethality–Based Strategy to Treat Cancers Harboring a Genetic Deficiency in the Chromatin Remodeling Factor BRG1

Takahiro Oike[1,2,6], Hideaki Ogiwara[1], Yuichi Tominaga[3], Kentaro Ito[3], Osamu Ando[3], Koji Tsuta[4], Tatsuji Mizukami[1,6], Yoko Shimada[1], Hisanori Isomura[1,2], Mayumi Komachi[6], Koh Furuta[4], Shun-Ichi Watanabe[5], Takashi Nakano[6], Jun Yokota[2], and Takashi Kohno[1]

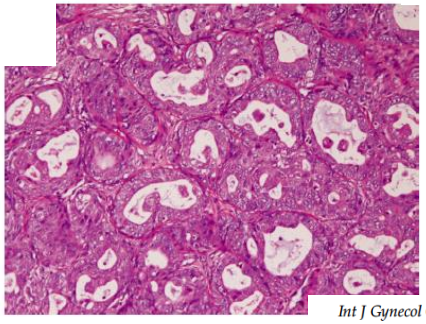
Curr Cancer Drug Targets. 2008 December ; 8(8): 733–740.

The *PIK3CA* Gene as a Mutated Target for Cancer Therapy

John P. Gustin[*,†], **David P. Cosgrove**[*], and **Ben Ho Park**[*,†,‡]

[*] Sidney Kimmel Comprehensive Cancer Center, The Johns Hopkins University School of Medicine, Baltimore, MD 21231

[†] Johns Hopkins University, Department of Chemical and Biomolecular Engineering, Baltimore, MD 21218



Int J Gynecol Cancer 2008, 18, 976–984

EAOC

Effect of a c-Met-specific, ATP-competitive small-molecule inhibitor SU11274 on human ovarian carcinoma cell growth, motility, and invasion

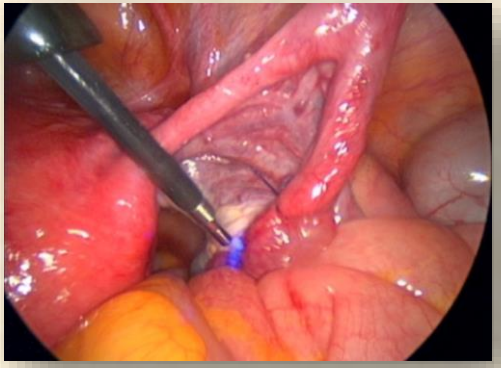
E.C. KOON*†, P.C. MA‡, R. SALGIA§, W.R. WELCH†‖, J.G. CHRISTENSEN¶, R.S. BERKOWITZ*† & S.C. MOK*†

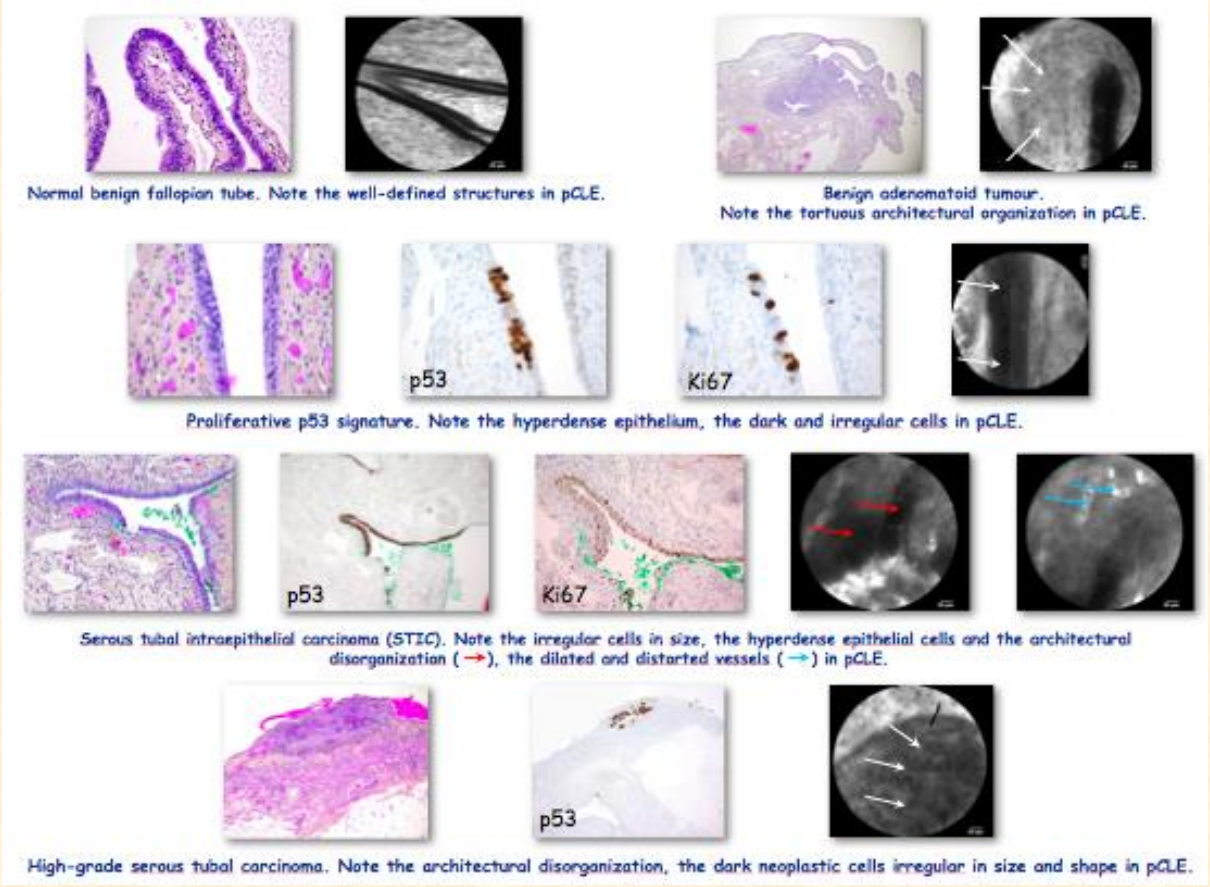
*Division of Gynecologic Oncology, Department of Obstetrics, Gynecology and Reproductive Biology, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts; †Dana-Farber Harvard Cancer Center, Dana-Farber Cancer Institute, Harvard Medical School, Boston, Massachusetts; ‡Division of Hematology/Oncology, University Hospitals Case Medical Center and Ireland Cancer Center, Case Comprehensive Cancer Center, Cleveland, Ohio; §Section of Hematology/Oncology, Department of Medicine, University of Chicago, Pritzker School of Medicine, University of Chicago Cancer Research Center, Chicago, Illinois; ‖Department of Pathology, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts; and ¶Pfizer Global Research and Development, San Diego, California

Implications et prévention

- **Biopsie optique**

La biopsie optique





Normal benign fallopian tube. Note the well-defined structures in pCLE.

Benign adenomatoid tumour.
Note the tortuous architectural organization in pCLE.

Proliferative p53 signature. Note the hyperdense epithelium, the dark and irregular cells in pCLE.

Serous tubal intraepithelial carcinoma (STIC). Note the irregular cells in size, the hyperdense epithelial cells and the architectural disorganization (→), the dilated and distorted vessels (→) in pCLE.

High-grade serous tubal carcinoma. Note the architectural disorganization, the dark neoplastic cells irregular in size and shape in pCLE.

Conclusions

- Endométriose: 5-15%
- Transformation maligne: 0.7-1.6% (≈ 10 ans)
  - Rare mais Association significative endométriose-cancer endométrioïde/ cellules claires
- Endométriose atypique: méfiance !
- Surveillance à long terme de toute endométriose

EXPERT REVIEWS ajog.org

GYNECOLOGY

New insights in the pathophysiology of ovarian cancer and implications for screening and prevention

Farr R. Nezhat, MD; Radu Apostol, DO; Camran Nezhat, MD; Tanja Pejovic, MD, PhD

- Surveillance clinique et radiologique (écho/IRM) prolongée:
  - endométriose apparue à un âge jeune
  - longue durée d'évolution de l'endométriose
  - volumineux endométriome ovarien
  - modifications radiologiques (échographiques/IRM) de l'aspect de l'endométriome
  - présence d'endométriose atypique à l'analyse anatomopathologique

Conclusions

- Endométriose: 5-15%
- Transformation maligne: 0.7-1.6% (≈ 10 ans)
  - Rare mais Association significative endométriose-cancer endométrioïde/ cellules claires
- Endométriose atypique: méfiance !
- Surveillance à long terme de toute endométriose
- Marqueurs prometteurs: ARID 1A, HNF-1β…
- Place de la ligature tubaire ? Salpingectomie prophylactique ?

Merci de votre attention…