eJOURNAL

January / February 2010

THE PRESIDENT's MESSAGE

Need miles, will travel

Professor Hans Evers
WES President

Enigma stems from the Greek *ainigma*, which in turn comes from *ainissesthai*, meaning "to speak darkly, to speak in riddles".

Endometriosis traditionally is referred to as an enigmatic disease: a perplexing, baffling, seemingly inexplicable matter. There are a lot of frequent flyer miles to be earned in this enigma. And as long as our favourite disease remains an enigma KLM, Air France, Qantas, BA, NorthWest, Delta Airlines and Lufthansa will fare well.

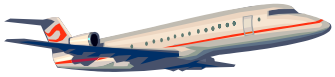
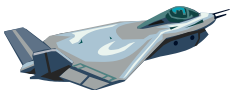
Only in the next few months the enigma will be discussed in Shiraz, Iran, at the Iranian Society of Reproductive Medicine annual meeting; in Nanning at the Chinese Reproductive Medicine congress; in Kotchi, Kerala at the Indian Society of Assisted Reproduction meeting; in Florence, Italy at the 14th World Congress on Gynaecological Endocrinology; in Buenos Aires at the Argentinean Endometriosis Society meeting, as well as in Istanbul, Frankfurt, Budapest, Milano, Orlando, Rome, Avellino, Athens and in San Francisco at the 58th Annual Clinical Meeting of the ACOG. Please consult our **Endometriosis Events Calendar** for further details.

This is a good sign; endometriosis research attracts more and more scientists and clinicians. You will learn all about the role of TECK and its receptor CCR9 in the pathogenesis of endometriosis, about Cyclooxygenase-2, the Protein Kinase-A pathway-regulated Transcriptome, Akt phosphorylation, angiogenesis and angiogenesis inhibitors, the old VEGF and the new VEGF, Beclin 1 expression, and of course the new kid on the block: the neuroendocrine cells in endometrium and endometriosis.

I have the feeling that we now have been circling the subject for quite some time but that we are not coming much closer. If so many different alterations and pathways are needed to explain one single disease, the most fundamental one apparently has not been found yet.

Therefore I would like to urge all of you: check our events calendar, book tickets, earn miles, mingle in the debate, and then go home, and start researching!

By the time of our next **World Congress on Endometriosis (4 - 7 September 2011)** we better come up with some decent answers. If not, we will at least be able to use our frequent flyer points to pay for our trip to Montpellier, France.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A WORD FROM THE EDITOR

2010

Dr Luk Rombauts
WES e-Journal Editor

As I write this introduction – and as a follow-on to the President's message – I am reminded of a quote by Professor Eric Thomas: "*Endometriosis is a disease that has allowed me to travel around the world – twice!*", which, as Hans Evers stresses, is an indication of the many challenges we still face in order to find answers to the enigma that is endometriosis.

I am therefore pleased to announce the first issue of the WES eJournal of the new decade. It is an exciting new issue for a number of reasons.

First of all, we will *not* be having a debate about what the new decade should be called (the 'teenies' ?).

Secondly, we celebrate our first book review. Associate Professsor Anusch Yazdani has read – and reviewed – the new edition of the book *Endometriosis and Pelvic Pain*, written by Dr Susan Evans and Deborah Bush.

Thirdly, we also have another stimulating Guest Editor's Digest, this time from Professor Ali Akoum, on immunity, exercise and endometriosis.

Finally, the editorial office has long been pleading to get readers to send us letters with comments or questions. We have received not one, but two such letters in response to our last issue on the link between endometriosis and ovarian cancer. So, I am delighted to announce that the forum of debate is now officially open!

I count on you all to keep it open. In particular, I seek further views and suggestions on the issues raised by our two well-known letter writers (you'll have to read the letters on pages 6 - 9 to find out who they are…).

We look forward to *your* contribution to the eJournal.

UPCOMING MEETINGS

ESHRE Guideline for the Diagnosis and Treatment of Endometriosis
26 February 2010
Budapest, Hungary

Endometriosis 2010 - from bench to patient
18 - 20 March 2010
Milano, Italy
Abstract/video deadline: 28 February 2010

Ultrasound in deep endometriosis
24 April 2010
Rome, Italy

58th Annual Clinical Meeting of the ACOG
15 - 19 May 2010
San Francisco, USA

World Congress of Minimally Invasive Gynecologic Surgery
26 - 29 June 2010
Dubrovnik, Croatia

26th Annual Meeting of ESHRE
28 - 30 June 2010
Rome, Italy

14th World Congress of Gynaecological Endocrinology
4 - 7 March, 2010
Firenze, Italy

Annual Scientific Meeting of the SGI
24 - 27 March 2010
Orlando, USA

12th International Meeting on Gynaecological Surgery
5 - 8 May 2010
Avellino, Italy

32nd British Congress of Obstetrics and Gynaecology
2 - 3 June 2010
Belfast, United Kingdom

ESHRE pre-congress course: Endometriosis - how new technologies may help
27 June 2010
Rome, Italy

COMPLETE CONGRESS SCHEDULE

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New pathogenic pathways and therapeutic approaches

Professor Ali Akoum
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Immunity

The complexity of endometriosis as a disease is illustrated by the huge amount of data showing endometrial dysfunctions, hormonal changes, genetic mutations, local and systemic immune anomalies, disrupted immune-endocrine networks. For instance, immune cells such as peritoneal macrophages and peripheral blood monocytes are activated and instead of being able to eliminate misplaced endometrial cells, they seem to contribute to their abnormal growth. Endometriotic lesions seem to escape immunosurveillance, elude the immune system and disrupt its capability to respond appropriately. Such a phenomenon is quite well known in tumorigenesis where tumor cells are capable of modifying immune cells' phenotype and reducing the proliferative potential of T cells.

Whether endometriosis development results at least in part from abnormal immune functions and/or leads to further immunosuppression is still to be more clearly elucidated. However the existence of a positive feedback loop remains plausible.

The relationship between endometriosis and other immune-related conditions is quite interesting.

Actually, the first article I selected from the recent literature supports the existence of a possible association between endometriosis and autoimmune diseases. In a recent study published in Fertility and Sterility (Gemmill et al, 2009) showed that women with endometriosis were more likely to report physician-diagnosed cancers, endocrine diseases, and infections than women in the general population.

As indicated by the authors, the present study has certain limitations that need to be taken into account, as conditions were self-reported and it was not possible to confirm the diagnosis by laboratory tests or review of medical records. Selection bias may also exist, since the 47% of women who opted to complete the questionnaire may be different from non-respondents or other members of the Endometriosis Association; in addition, they were more educated than the general population. It is clear that this study would have been strengthened by including a similar group of women without endometriosis completing the same survey. Nevertheless, it sheds light on new aspects of endometriosis physiopathology as it describes the prevalence of coexisting diseases suspected to be common in women with endometriosis.

Cancers, infections, and endocrine diseases in women with endometriosis

Fertil Steril 2009 Nov 26 [Epub ahead of print]

Gemmill JA, Stratton P, Cleary SD, Ballweg ML, Sinaii N.

OBJECTIVE: To assess the prevalence of patient-reported, physician-diagnosed comorbid conditions in women with endometriosis. DESIGN: Cross-sectional study of self-reported survey data. SETTING: Academic research. PATIENT(S): Four thousand three hundred thirty-one Endometriosis Association (EA) members reporting surgically diagnosed endometriosis. INTERVENTION(S): None. MAIN OUTCOME MEASURE(S): Self-reported, physician-diagnosed infectious diseases, cancers, and endocrine diseases. RESULT(S): Nearly two-thirds of women reported one or more of the assessed conditions. Recurrent upper respiratory infections and recurrent vaginal infections were common and more likely in women responding to the EA survey. Melanoma was reported by 0.7% (n=29), breast cancer by 0.4% (n=16), and ovarian cancer by 0.2% (n=10). While ovarian cancer and melanoma were significantly more common than in the general population, breast cancer was surprisingly less common. Addison's disease and Cushing's syndrome were rare (0.2% and 0.1%, respectively). CONCLUSION(S): Respondents reported a higher prevalence of recurrent upper respiratory or vaginal infections, melanoma, and ovarian cancer than the general population. These findings document other potential associations related to the immune system, which may help focus future research into this disease.

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It has been suggested that endometriosis may increase the risk of ovarian cancer, particularly endometrioid and clear cell ovarian cancer. However, studies assessing infections in women with endometriosis are lacking and it is unknown whether immune response abnormalities and alterations noted in women with endometriosis may predispose them to have infections. Reciprocally, it is still unknown whether exogenous factors such as bacterial and viral infections may modulate the immune response and facilitate endometriosis development. Such putative pathogenic pathways remain unknown and deserve further investigations.

Fitness

It is long known that physical activity is associated with a reduced risk of endometriosis. Previous epidemiological studies showed that diet and physical activity counseling by a dietitian can significantly improve endometriosis symptoms and that women who engaged in high-intensity activity were less likely to be diagnosed with endometriosis.

Signorello et al (1997) and Cramer et al (1986) observed that vigorous exercise is an apparent protective factor against endometriosis. Dhillon and Holt (2003) reported that women with a recent, frequent and high-intensity activity had a reduced endometrioma risk compared with women who engaged in no high-intensity activity.

Intense exercise has been related to a disruption of the hypothalamo-pituitary-ovarian axis leading to hypoestrogenism (Warren and Perlroth, 2001), which would be in agreement with a reduced risk of estrogen-dependent diseases such as endometriosis endometrial cancer because of lower levels of hormonal stimuli (e.g. endogenous estrogen levels), which influence both growth and development.

A recent study from Vitonis AF et al (2010) however did not find evidence of the strong inverse association previously reported.

Using data collected from the Nurses' Health Study II, a prospective cohort study of premenopausal US nurses (102,197 premenopausal women, 996,422 person-years of follow-up with 2703 cases of laparoscopically confirmed endometriosis), the authors observed a small inverse association between adult total recreational physical activity (measured in MET hours/week) and incidence rates of laparoscopically confirmed endometriosis.

It is somehow surprising why previous studies found large protective associations and this study did not. However, it is quite plausible that the retrospective design of the previous case-control studies, compared with this prospective study may have contributed to the difference in findings.

Adult physical activity and endometriosis risk

Epidemiology 2010 Jan;21(1):16-23.

Vitonis AF, Hankinson SE, Hornstein MD, Missmer SA.

BACKGROUND: Regular exercise has been associated with a 40%-80% reduction in risk for endometriosis in several case-control studies. However, women experiencing symptoms prior to their diagnosis may be less likely to exercise than healthy controls, thus biasing the observed association. METHODS: Using data collected from the Nurses' Health Study II, a prospective cohort study of premenopausal US nurses that began in 1989, we have attempted to clarify this relation. Data are updated every 2 years with follow-up for these analyses through 2001. In 1989, 1991, and 1997 women reported average amount of time per week engaging in various physical activities. A metabolic equivalent (MET) score was assigned to each activity, and these were summed to estimate total activity. RESULTS: A total of 102,197 premenopausal women contributed 996,422 person-years of follow-up with 2703 cases of laparoscopically confirmed endometriosis. After adjusting for BMI at age 18, current BMI, smoking, parity, infertility status, oral contraceptive use, age at menarche, and menstrual cycle length and pattern in college, we observed only a slight reduction in the incidence of endometriosis, comparing the highest level of activity (>or=42 MET hours/week) to the lowest (<3 MET hours/week) (rate ratio = 0.89 [95% confidence interval = 0.77-1.03]). The association was limited to participants with no past or concurrent infertility (P = 0.002, test for heterogeneity). No associations were seen with inactivity. CONCLUSIONS: In this first prospective assessment, we did not find evidence of the strong inverse association previously reported, although we cannot rule out a modest inverse association.

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Also, controls should be representative of the source population from which the cases arose. Fertile controls may not be an appropriate control group for infertile subjects as the case in some previous studies. The proportion of healthy controls able to exercise would be higher than that of women experiencing symptoms prior to diagnosis.

This having been said, I still believe that physical activity and lifestyle change are to be taken into consideration within a global multidisciplinary therapeutic approach that meets the patient's specific needs.

In conclusion

It goes without saying that the disease has an enormous debilitating effect on a great number of women and a substantial economic impact.

The study of Fourquet et al (2009) adds to a rich literature on the heavy impact of the disease, the cost it generates and the importance of conducting basic and clinical research from a broader perspective and explore new pathogenic pathways and therapeutic approaches.

Patients' report on how endometriosis affects health, work, and daily life

Fertil Steril 2009 Nov 14. [Epub ahead of print]

Fourquet J, Gao X, Zavala D, Orengo JC, Abac S, Ruiz A, Laboy J, Flores I.

The objective of this study was to assess the burden of endometriosis by obtaining patient-reported outcome data describing the experience of living with this disease. Survey data from 107 women with self-reported, surgically diagnosed endometriosis showed that living with this disease may be characterized by physical limitations that disrupt health, work, and daily life.

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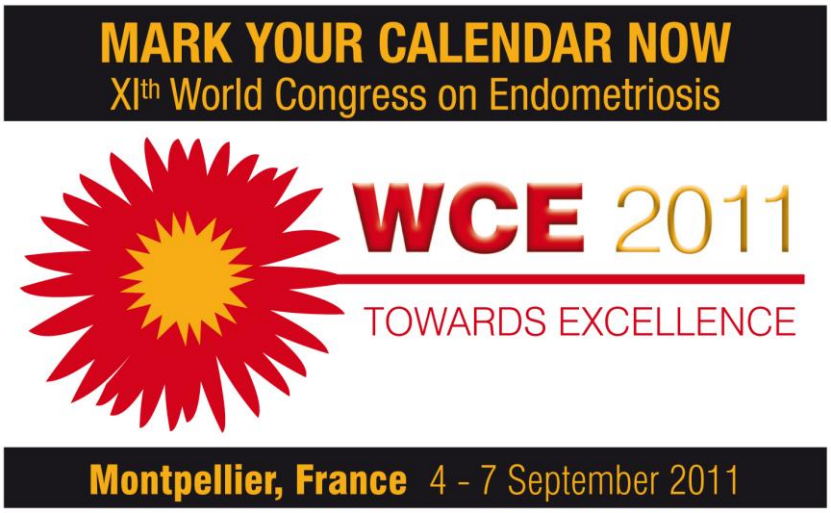

COMMENTS and DEBATE

Professor Emeritus
Ivo Brosens

Dear Editor,

Indirectly there may be a relationship with the amount of endometriotic tissue, but for a complex structure as the ovarian endometrioma, it seems simplistic to assume that the risk of cancer is related to the size of an endometrioma.

Ovarian endometriosis has multiple presentations, including chocolate cysts of various size, deep non-cystic lesions, surface pits and plaques and subtle superficial lesions.

The detailed histopathological study of twenty-nine ovaries with cystic endometriosis by Hughesdon (Hughesdon, 1957) showed that all, except three, had an essentially similar structure: the inside of a chocolate cyst is the outside of the ovary.

Hughesdon described as features of the ovarian endometrioma the presence of primordial and ripening follicles in the wall, flattening of the cortex on the bulging outside and smooth muscle metaplasia on the inside of the cortex. Using an endoscopic technique to explore the (pseudo) cystic structure Brosens et al (1994) confirmed these features and furthermore demonstrated that mucosa-type implants were predominantly located at the site of invagination and adhesions with the pelvic wall.

So, there are likely to be many factors involved, such as the amount of surface epithelium and stroma lining the cyst, metaplasia of the cortex, the fibrosis of the wall, the extent of periovarian adhesions, the presence of a communicating lutein cyst, and furthermore the use of oral contraception or progestogens, etc. that determine whether an untreated endometrioma will be 16, 8, 4 or 2 cm in diameter.

Now, for my two questions. Are data available on:

1. which percentage of cancers in ovarian endometriomas have their origin in ovarian tissue and which in endometrial tissue, and
2. whether ovarian cancers associated with endometriomas arise at the site of adhesions and invagination (where most of the endometriotic tissue is found), on the distended capsule (frequently no endometriosis on the lining) or elsewhere in the ovary?

I would very much like to know your opinion.

With best wishes from a snow-white Heverlee (Belgium),
Ivo Brosens

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Dear Professor Brosens,

For a while now the journal has been encouraging readers to send us comments, so your letter is much appreciated. I expect our readers to respond to your enquiries, but I would like to start the correspondence with a few comments of my own.

You rightly mention that there are many other factors that may determine the premalignant potential of ovarian endometriomas. Unfortunately, surgical reports rarely contain the level of detail required to analyse these in epidemiological studies. Also, adding more variables as confounding factors invariably will decrease the statistical power and investigators thus often have to make hard decisions regarding which variables to enter in their models.

In my opinion, the size of the endometrioma is nothing more than a proxy for the duration of its existence and not the

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amount of endometriotic tissue within the cyst. It makes sense then that the size is correlated with malignant potential, at least if you accept that some form of oncogenic mutation has to occur before the endometriosis can develop into a malignancy.

I can't answer your second question, but there is some data available to guide us with your first question. In their review Van Gorp et al (2004) have summarised the available studies on this topic. Table 2 provides information regarding the studies that have followed the Scott classification. The Scott classification system requires that there is histological evidence of a continuous transition between benign and malignant epithelium within endometriosis and is thus considered a very strict classification system. But proof of such a histological transition may disappear with the growth of the tumour and working out what percentage of cancers in ovarian endometriomas have their origin in endometrial tissue will therefore most likely be an underestimate. This is why such reports often use a modified system, the results of which are also found in Table 2.

I sincerely hope that the eJournal will receive further insights from our readers.

With my best wishes for a fruitful 2010 from a sunny beach on the Mornington Peninsula (Australia).
Luk Rombauts

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Professor
Philippe Koninckx

Dear Editor,

The association between ovarian cancer and endometriosis has become highlighted over the last years and the last eJournal adequately details the available data. The interpretation of the data with RR ratios and confidence intervals, however, unfortunately leaves a feeling of 'How to lie with statistics'. We know the limitations of cohort studies and of associations. Since our interpretation of data as emerging from the published articles seems at least insufficiently prudent, biased and incomplete – if not incorrect – and potentially harmful, inducing fear in women with endometriosis, some discussion seems appropriate.

Biochemical similarities
Often biochemical similarities between endometriosis and cancer are described (Prowse et al, 2006; Mandai et al, 2009). The pathophysiology of endometriosis clearly is not well understood. Considering typical, cystic, and deep endometriosis as a benign tumour caused by a genomic incident (Koninckx et al, 1997) (and subtle lesions as a physiologic condition occurring intermittently in all women) remains the easiest way to explain hereditary influences and effects of dioxins and total body radiation. It, moreover, could explain the many described differences in the endometrium of women with and without endometriosis: the slight differences could point to some genetic disturbances making them more susceptible to develop endometriosis. The Sampson hypothesis seems attractive since viable endometrial cells, with implantation capacity are found in the peritoneal fluid of most women. To the best of our knowledge, however, a progression from subtle to typical to cystic or deep lesions has never been observed nor demonstrated. The Sampson hypothesis, and the metaplasia theory which is a variant, can explain implantation but should be considered as speculation since it cannot, does not even attempt to, explain progression. If, however, endometriosis is considered a benign tumour, and if the endometrial differences signal some genetic predisposition (and are not a consequence of the existing endometriosis) it is not surprising that some biochemical similarities exist between endometriosis and ovarian cancer and cancer in general.

Association statistics
The case control study of Brinton et al (1997) finds an odds ratio of 1.9 to more than 4 for developing ovarian cancer in women with endometriosis or ovarian endometriosis respectively, a conclusion based upon a Swedish hospital discharge diagnosis of endometriosis. The study of Melin et al (2006) finds an odds ratio of 1.2 equally based on a hospital discharge diagnosis of endometriosis. It can be anticipated that this group of women, in comparison with women without a diagnosis of endometriosis, had had more interventions, more pelvic pain, more medical treatments of endometriosis and probably more infertility treatments. Having been diagnosed with ovarian endometriosis means probable surgery of the ovary, very often focal coagulation leaving behind at least carbon deposits. It also means more adhesion formation etc. It is intellectually unfair to pick one aspect only –endometriosis– for the comparison of two groups of women who differ in many other aspects.

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This indeed is a fundamental problem of correlation and association statistics. Logistic regression and statistical model building were developed to find out which are independent and which are important factors. This obviously has not been done for the association of endometriosis and cancer. A second bias might have been introduced when analysing national discharge statistics. In most women ovarian endometriosis is diagnosed by ultrasound; and in a recent IOTA review (Van Holsbeke, in press) of over 2500 cysts, a risk of an (borderline) ovarian cancer was found in 1%. This could seem to support the argument, but analysing the data in detail, this is true only for women after menopause, a period of life that seems hardly associated with endometriosis. It would not be surprising if many women who did not have ovarian endometriosis by pathology ended up with a primary hospital discharge diagnosis of endometriosis.

Tubal ligation

Tubal ligation is associated with a decreased risk of ovarian cancer, a lower incidence of PID, and absence of retrograde menstruation. Tubal ligation, however, also influences in some women ovarian function and blood supply at least as evidenced by irregular cycles. Today we do not know how to explain exactly the decreased incidence of ovarian cancer in these women. The absence of retrograde menstruation does not seem logical as an explanation; indeed women with severe cystic ovarian endometriosis will have a higher incidence of infertility and thus a lower probability of undergoing tubal sterilisation.

Accuracy of diagnosis

It is surprising that the diagnosis of endometriosis is so easily accepted in the articles using hospital discharge records, knowing that the diagnosis of endometriosis is bound to have biases. Whereas for typical and deep lesions the probability of histological confirmation is very high, the rate of histological confirmation of subtle lesions is low, mainly dependent upon the expertise of the surgeon and the pathologist. Also for cystic ovarian endometriosis the histological confirmation of endometriosis, defined as stroma and glands, is not that well established and often pathology returns as 'compatible with endometriosis'; most of these women, however, will be discharged as having endometriosis.

Especially after menopause the diagnosis of endometriosis risks to be erroneous. Indeed, the occurrence of 'cystic ovarian endometriosis' or 'large cystic ovarian endometriosis' in women who did not have an existing cystic ovarian endometriosis before menopause is something rare and suspicious by definition. From the available data it is difficult to judge how strict the diagnosis of endometriosis was. Anyway, it would not be surprising, that many of these women were erroneously diagnosed as having had endometriosis. For recent prospective studies where women were followed by serial ultrasound Kobayashi et al (2009, 2008) the bias is obvious given that the ultrasound diagnosis of endometriosis has a specificity of 88% only. Moreover in the group of the 12% false positives the ovarian cancer incidence was 18% after menopause versus 0.6% before menopause (Van Holsbeke, in press). It is moreover questionable not to perform surgery in menopausal women with an ultrasound diagnosis of endometriosis.

Menopause

Unopposed oestrogen administration following menopause is associated with an increased risk of endometrial hyperplasia and adenocarcinoma. Following menopause, existing endometriosis will become inactive but will not disappear. Considering the prevalence of endometriosis in women before menopause it is very surprising that oestrogen-only therapy following menopause (in women without an endometrium) is not associated with an epidemic of adenocarcinomas originating from the endometriosis. This even looks as if endometriotic cells, although hormonally responsive, are pretty resistant to malignant transformation.

Metastatic behaviour

Especially deep endometriosis has invasive and 'metastatic' behavior because of the positive lymph nodes. In our series of over 2000 deep nodules, only one however has turned out to be malignant, and this in a postmenopausal woman (unpublished data).

In conclusion, the data describing an association between endometriosis, in particular cystic ovarian endometriosis, and ovarian cancer can be interpreted in many different ways. Today we do not consider the data sufficient to consider women with endometriosis or cystic endometriosis at risk for developing ovarian cancer, a conclusion already reached by others (Somigliana et al, 2006). It reminds us of the fact that so many ovaries have been removed prophylactically after age 50 to find out recently that women with ovaries live longer than women without ovaries (Parker et al, 2009)

'It ain't so much the things we don't know that gets us into trouble.
It's the things we do know that just ain't so'
Josh Billings

Philippe R Koninckx and *Anastasia Ussia*

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Dear Professor Koninckx and Dr Ussia

Thank you so much for your comprehensive summary and opinion on the relationship between endometriosis and cancer. We value your feedback.

As usual your comments are food for thought. I find myself very tempted to respond, but instead I invite our readers to continue the discussion. So WES members, let us have your feedback on this opinion-paper!

With kind regards,
Luk Rombauts

JOB OPPORTUNITIES

- **Postdoctoral position available at the Netherlands Metabolomics Centre**
- **Call for applications (from Argentina) for scholarships to the University of Leuven**

CASE STUDIES WANTED

The ASRM EndoSIG is soliciting suggestions for endometriosis clinical case scenarios for inclusion in the new ASRM Case-of-the-Month surveys. These clinical vignettes on diagnosis and management will utilize a multiple choice question format and will help the CME Committee identify professional practice gaps.

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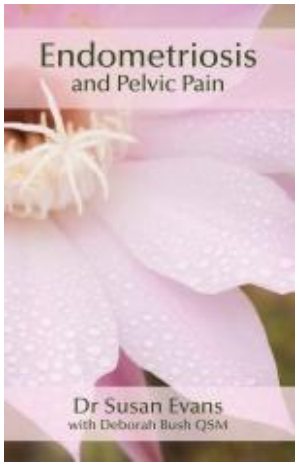

Endometriosis and pelvic pain (second edition)

Author: Susan Evans
Publisher: Susan Evans
Publication Year: 2009
Target Audience: Consumer

Rating

Content:
Readability:
Interest:
Overall:

Reviewed by: Anusch Yazdani
Associate Professor, University of Queensland and Director of Research and Development, QFG Research Foundation, Australia

Unfortunately, only too infrequently does a book come along that leads a sensitive and comprehensive approach to the diagnosis and management of chronic pain. *Endometriosis and Pelvic Pain*, written by Dr Susan Evans, is such a book.

Originally published in 2005, the first edition has enjoyed deserved success as the definitive guide for women with endometriosis or chronic pelvic pain. Written by and from the perspective of a respected and experienced specialist in the field, Dr Evans explores endometriosis in the greater context of chronic pain.

In its most fundamental form, the book provides a very good introduction to female anatomy and the gynaecological consultation and then covers the aetiology of endometriosis, symptoms, diagnosis, medical and surgical treatment options.

Excellent chapters cover frequently asked questions ('*Will my endometriosis come back?*' "*Will I be able to have children*?" or '*Do I need a hysterectomy*'). Most importantly, however, the author provides a comprehensive overview of other causes of pain and/or associated conditions, including adenomyosis, irritable bowel syndrome or musculoskeletal pain. Complementary and dietary therapies are appropriately addressed.

In 2009, the book was extensively rewritten and reorganised. Most noticeably, the chapters have been reorganised by *symptoms* in a more consumer focussed approach, rather than addressing individual *conditions* - an organisational structure that appeals mainly to health professionals.

The current edition adds excellent chapters by Deborah Bush on living positively with chronic pain and endometriosis, clearly empowering women to re-establish a locus of control, rather than allowing the diagnosis of endometriosis to rule. Throughout the book, case vignettes illustrate specific issues and problems.

Unfortunately, while the first edition was widely obtainable, the current edition is only available by mail order (**www.drsusanevans.com**) or Endometriosis New Zealand (**www.nzendo.co.nz**). Furthermore, the somewhat anachronistic approach of limited illustrations and sparse graphics is less likely to appeal to a generation that expects the sensory barrage of YouTube, the authority of Wikipedia and the immediacy of Twitter. While this does not detract from the book, it is a relatively heavy read for generation Y. The combination of limited access and readability may unfortunately diminish the impact of this otherwise excellent book.

Overall, however, *Endometriosis and Pelvic Pain* remains essential reading for women affected by endometriosis and chronic pain, their families, loved ones and carers.

DEADLINE for contributions to the April/May eJournal is 25 March 2010

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