Endometriosis Theories and Concepts

Revised 9/5/18

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Endometriosis Theories and Concepts

"Studying endometriosis is like nailing Jell-O to a tree."
Donna Vogel, National Institute of Health, Endometriosis 2000

Introduction

This document focuses on theories and concepts that can help in understanding endometriosis. Theories date back at least to 1870 (Waldeyer) following Rokitansky's 1860 histologic description of what we now call endometriosis.

A theory may be useful at several levels including guiding research, acting as a framework for education, understanding possibilities in endometriosis, explaining why changes occur in endometriosis, and explaining why a treatment works. However, a theory does not determine if a treatment works. The success of a treatment is based on evidence, not on theory. A discussion of the *tomato effect*, or how theory can interfere with treatment, is covered later in this file.

No theory is completely adequate. It generally takes seven theories and concepts to introduce what I have seen and many more to discuss what I have read. Since theories change, this file will be periodically updated and posted at the addresses above.

The seven, non-exclusive theories and concepts needed to discuss what I have seen are:

- Retrograde menstruation with peritoneal dispersion, attachment, infiltration, and growth
- Differentiation of precursor (stem) cells into endometriotic cells with subsequent replacement of endometrial cells by endometriotic cells
- Fibrotic collagen reaction with muscular metaplasia as part of metaplasia or local reaction
- Age dependent changes in appearances and depth of infiltration
- Immunologic maturation and competence
- Surgical scar or traumatic vaginal tear implantation
- Inflammatory induction might include menstrual debris, trauma, or infection

The “Theories and Concepts” section in this document is needed to introduce what I have read and published. Those theories and concepts generally cover the source of the original cell and why, how, and when the original cell converts to endometriosis. Those include:

- The Cell of Origin
    - Dissemination / Metastasis
        - Retrograde Menstruation
        - Hematogenous Dissemination
        - Lymphatic Dissemination
        - Traumatic / Surgical Dissemination
    - Müllerian Theories
        - Müllerian Remnants (any congenital)
        - Müllerianosis (organoid)
        - Mülleriosis (non-organoid and projected to include transition)
        - Secondary Müllerian System
    - Metaplastic Theories
        - Peritoneal / Mesenchymal Stem Cells
        - Bone Marrow Stem Cells
        - Endometrial Stem Cells
- Transition from endometrium to endometriosis

  The transition involves the local environment, inflammation, epigenetic changes, genetic changes progenitor cell differentiation, biochemical changes immunologic changes, apoptosis, autophagy, reactive oxygen species, fibrosis, muscular metaplasia, macrophage migration inhibitor factor, clonality, microRNA, signaling, nerve activation, cancer-associated driver mutations, fibroblast to myofibroblast transdifferentiation, neurogenesis, angiogenesis, genetic dysregulation and more that are covered in this document

The articles listed in this review are only a small part of what is published. A recent (8/31/18) PubMed search at https://www.ncbi.nlm.nih.gov/pubmed/?term=endometriosis listed 25,538 articles that include many parts of the endometriosis story. That is an increase of 65 article since 8/14/18. The concerns include theories, results of treatment, biochemical testing, immunologic testing, inflammatory reaction, spontaneous resolution of endometriosis, stages, phenotypes, aromatase production, hormonal levels, embryology, neonatal development, genetics, epigenetics, organoid development, stromal type endometriosis, endometriosis in men, bone marrow stem cells in endometriosis, differentiated stem cells, primordial germ cells, programmed death (apoptosis) and transitions into mesenchymal cells.

The Tomato Effect (Theory-Based Medicine)

The tomato effect in medicine occurs when an effective treatment for a certain disease is ignored or rejected because it does not make sense in the light of accepted theories of disease mechanisms and treatment of these diseases. The tomato effect can interfere with the acceptance of useful remedies. (Goodwin & Goodwin 1984)

According to Goodwin & Goodwin, the only three issues that matter in picking a therapy are:

- Does it help?
- How toxic is it?
- How much does it cost?

Goodwin & Goodwin's three issues can be expanded to risks, benefits, costs, acceptability, availability, insurance coverage and other associated concerns of using a therapy.

Discussions of theory are not discussions about the effectiveness of treatment. The results of surgical or medical therapy stand on their therapeutic outcomes, not on an opinion or a theory.

Since early endometriosis can be transient or stable in some, if not most cases, observation or symptomatic care, such as hormonal suppression can be reasonable. Superficial endometriosis can respond to observation (Evers 1994, Koninckx 1994, Harrison 2000), medication or coagulation. Deep endometriosis will more likely require excision (Martin 1989). Excision was successful in my practice, just as it was for Dr. David Redwine. His reoperation rate of 55%, with only 19% having histologic endometriosis, was like mine in the 1980s. (Redwine 1991)

In the later years of my practice, although the persistent pain rate after surgery remained relatively constant, I stopped doing as many repeat laparoscopies. Sutton (1994) noted that 3 of pain relief after surgery is non-specific and can be a placebo response. Performing a repeat laparoscopy for pain that occurred in the first six months after excision was not commonly useful. I focused more on their questions and concerns, helping them with expectations, considering hormonal suppression, encouraging physical therapy, considering stress therapy, deciding about judicious use of narcotics, and more.

Evidence-Based Medicine

Evidence-based medicine, like theory, is dependent on the knowledge available at the time it is applied. When knowledge changes, the approach to a disease and its treatment can also change. "Medical reversal" is a term used to describe the phenomenon when the long-established medical practice changes due to new, emerging evidence. Vinay Prasad's *Ending Medical Reversal: Improving Outcomes, Saving Lives* (2015) discusses the problems that can occur with those changes. Although evidence-based medicine is more grounded than theory-based medicine, both are subject to change over time. Both are subject to the seven stages of a medical reversal: 1) promising report, 2) adoption by providers, 3) patients and payors accept the innovation, 4) insubstantial studies that superficially support the innovation, 5) randomized controlled trials, 6) denial if the trials do not support earlier observations and finally 7) acceptance.

These problems can be compounded by delay. Balas (2000) studied the components of delay such as the time needed to do the research, have the research accepted for publication, and have the change accepted by the general medical community. He calculated that it takes an average of 17 years for research evidence to reach clinical practice.

Theories and Concepts

1. Waldeyer 1870 – Metaplasia from the germinal epithelium of an ovary. If Waldeyer considered the germinal epithelium as a precursor to ovarian serosa, this might be the first recognition of a progenitor. The germinal epithelium of an ovary had also been considered as the precursor to eggs. See Iwanoff 1898 for coelomic metaplasia and Lauchlan 1972 for metaplasia from secondary Müllerian system.
2. Cullen 1896, Russell 1899, Redwine 1988 – Müllerianosis (Mülleriosis) as a remnant or fragment of Müllerian tissue in or near the normal area of embryologic Müllerian development. See Nerune 2016 & Rei 2018 persistent Müllerian duct in men.
3. Iwanoff 1898, Meyer 1903, Sampson 1921, Matsuura 1999 – Coelomic metaplasia of ovarian serosa may be the same concept as Waldeyer's metaplasia from the germinal epithelium.
4. Cullen 1914 – Endometriosis has fibrous and muscular components like adenomyoma.
5. Hueter, 1918, Meyer, 1919 – Inflammatory metaplasia
6. Sampson 1921 – Peritoneal implantation from internally menstruating ovaries
7. Meyer 1923, Gruenwald 1942 – Coelomic metaplasia from the peritoneum
8. Halban 1925, Jerman 2015 – Lymphatic spread (metastasis) of the endometrium
9. Halban 1925 – Hematogenous vascular spread (metastasis) of the endometrium
10. Sampson 1927, Nap 2004, Nap 2012 – Retrograde menstruation theory includes:
    - Retrograde menstruation of tissue fragments
    - Peritoneal dispersion
    - Attachment
    - Inflammation
    - Infiltration
    - Growth

    Revisions of dispersion (retrograde menstruation, lymphatic, hematogenous, traumatic, surgical), congenital (Müllerianosis (organoid), Mülleriosis (non-organoid), secondary Müllerian system) and metaplasia theories have been expanded to include the role of stem cells, replacement of endometrial cells by endometriotic cells, differentiation of stem cells into endometriotic cells, and other concerns reviewed in the references that follow.
11. Novak 1931 – Metaplasia due to hormonal stimulation
12. Fallon 1950 – Endometriosis can be colorless and amenorrheic
13. Levander 1955, Merrill 1966, Lauchlan 1972 – Induction of endometriosis due to activation of mesenchymal cell metaplasia by degenerating endometrium that arrives in the pelvis.
14. Nora 1956, Steck 1965, Kaunotz 1979 – Direct implantation in surgical scars, amniocentesis needle tract or traumatic vaginal tears
15. Karnaky 1969, Jansen 1869 – Diagnosed endometriosis in the absence of hemosiderin
16. Karnaky 1969, Redwine 1987, Davis 1988, Koninckx 1991 – Based on age distributions, there is a 4 to 20-year progression from an initial water blister lesion (clear papule) to red to hemorrhage to scar to scar with blue dome cysts (black only appearance) to deep infiltrating endometriosis.

17. Karnaky 1969 – Endometrium and endometriosis respond differently to antiestrogen therapy. He further notes that the differences in humans were not seen in monkeys and questions if monkey research might be on normal transplanted endometrium and not endometriosis. He felt this supported the theory of coelomic metaplasia.
18. Lauchlan 1972 – Differentiation of tissue in a secondary Müllerian system may be responsible for endometriosis outside the normal Müllerian developmental area. He felt that pelvic endometriosis was most compatible with retrograde while distal, non-abdominal sites might be hematogenous dissemination or metaplasia. He also noted that endometriosis is histologically different than endometrium with a mixture of cell types. See Cullen 1914 for fibrous and muscular components.
    Author's Note: Subtle, superficial peritoneal and small typical endometriosis are generally outside the normal Müllerian area. Deep infiltrating retrocervical, rectovaginal and anterior cervical endometriosis may start in the normal Müllerian area. Ileum, appendix, cecum, lateral gutters, and diaphragm are intra-abdominal, but not in the secondary Müllerian area.
19. Mettler 1979 – Reported on ovarian cyst resection but concluded that more than "*coagulation of endometriotic foci cannot be performed via the laparoscope*."
20. Goldstein 1980 – Endometriosis in adolescents. Karnaky 1969 discussed young girls.
21. Goldstein 1980, Redwine 1988 – A close-up or near-contact view is better for recognizing subtle appearances of endometriosis.
22. Simpson 1980 – Genetic predisposition is generally seen as an observation, not a theory.
23. Semm 1980 – Laparoscopic partial excision needs to precede coagulation with large nodules.
24. Martin 1983, 1985, 1986, 1987, 1988, 1989 – The CO2 laser can be used laparoscopically for excision of deep endometriosis.
25. Semm 1984 (German), Semm 1987 (English) – "*The surgical excision of endometriosis implants is still considered the optimal treatment of pelvic endometriosis."*
26. Malinak 1984 – Recurrence rates are likely higher than published due to asymptomatic recurrence.
27. Vernon 1986 – There are differences in prostaglandin production in the four (4) surface phenotypes examined. "*Petechial implants may be more pathologically influential than older implants*." "*A patient who presents with severe, progressive dysmenorrhea but is shown at laparoscopy to have minimal disease may have exaggerated pain symptoms as a result of the presence of the more biochemically active, petechial implants, whereas a patient with extensive disease may have minimal pain symptoms due to the presence of primarily inactive, powder-burn implants.*" See Davis 1993
28. Redwine 1987, Martin 1989, Albee 2008 – Any abnormality of the pelvic peritoneum, no matter how small, how subtle, or what color, may be endometriosis.
29. Halme 1988, Hill 1992, Northick 2016 – Lack of immunologic competence results in an inadequate response of the peritoneal defense system to the normal retrograde flow that is present in most women. The inadequate response results in evasion of apoptosis of endometrial cells, and endometriosis continues to live.
30. Martin 1989 – 13 of the 20 laparoscopic surface appearances of endometriosis were phenotypic. In 2018, we do not know if only some or all these have similar or contrasting characteristics. Vernon (1986) used four other descriptive superficial phenotypes.

31. Martin 1989, Davis 1993 – The type of procedure should consider the depth of infiltration. The definition of deep decreased from 5 mm in 1989 to less than 3 mm in 1993. Clinically, this definition was not overly useful as it could only be determined if the lesions were excised and processed for specific depth measurements. The concept then changed over several years to peritoneal and infiltrating lesions. Infiltration and pain were generally associated with fibrosis and depth. (Ripps 1991, Ripps 1992, Khare 1996, Vigano 2017, and Liu 2017). Furthermore, even superficial appearance could be associated with infiltration to 4 mm. (Koninckx 1991)
32. Cornillie 1990 – In-phase cyclic changes are different in deep (≥5 mm), intermediate (2 to 4 mm), and superficial (<1 mm) endometriosis
33. Cornillie 1991 – The presence of endometrial protein PP14 positivity varies in deep (≥5 mm), intermediate (2 to 4 mm), and superficial (≤1 mm) endometriosis.
34. Koninckx 1991, Gordts 2017 – Deep endometriosis is endometriotic disease. Superficial endometriosis is either stopped by the immune system or converted into endometriotic disease.
35. Portz 1991, Vitale 2018 – Reactive oxygen species (ROS) or free radicals may increase growth and adhesion of endometrial cells in the peritoneal cavity, promoting endometriosis and infertility
36. Ripps 1991 – Pain and tenderness are associated with fibrosis (scarring) of implants.
37. Ripps 1992 – Pain and tenderness are related to the depth and volume of implants.
38. Koninckx 1992 – Deep endometriosis has 3 phenotypes: superficial (<3 cm), intermediate3 to 5 cm) and deep (0.5 cm or deeper)
39. Rier 1993 – Environmental toxins such as dioxin may increase the risk of endometriosis by modulating the immune response of altering tissue-specific responses to hormones.
40. Haney 1993 – Endometriosis is associated with a localized sterile inflammatory process, growth factors, cytokines, and activated macrophages in the peritoneal fluid.
41. Davis 1993, Vercellini 1991 – Adolescents with functional pain, cyclic pain, abdominal pain, nausea, constipation, and diarrhea during menses have the largest proportion of red lesions. See Vernon 1986.
42. Evers 1994, Koninckx 1994, Harrison 2000, Nap 2004 – Endometriosis in its superficial form is generally self-limiting and transient.
43. Wild 1994, Nisolle 2000, Witz 2002 – Endometrial stromal cells and epithelial cells can attach to the peritoneum within one hour and the mesothelium can be replaced by 24 hours. These observations were in research animals. Research as this level in humans will likely continue be unethical without a major paradigm shift in technology.
44. Sutton 1994 – Pain relief at three months is not significantly different between patient who had endometriosis removed and those who had a diagnostic laparoscopy only. At six months the placebo response had resolved and pain recurred in the diagnostic only group.
45. Perper 1995 – Menstrual cramps (dysmenorrhea) are related to the number of implants.
46. Fernandez 1995 – Bone marrow-derived cells are found in endometriosis.
47. Khare 1996 – Differences in collagen types suggest that ovarian endometriosis may be metastatic while pelvic wall-infiltrating endometriosis is metaplastic.
48. ASRM 1997 – Eight laparoscopic phenotypes

49. Nisolle 1997 – Peritoneal, ovarian, and rectovaginal nodules are three different entities
50. Gaetje 1997 – Invasion based on E-cad- epithelial cells
51. Leyendecker 1998 – Intrauterine tissue injury and repair at the endometrial-muscularis interface (TIAR) due to intrauterine trauma.
52. Starzinski-Powitz 2001, 2003 – Differentiation of stem cells into endometriotic cells
53. Redwine 2002 – 38 differences between endometriosis and eutopic endometrium in humans
54. Gazvani 2002 – The peritoneal environment can influence the development of endometriosis.
55. Kats 2002 – Macrophage migration inhibitory factor is higher in early (subtle red) than in late (blue, black, or white) lesion appearances.
56. Bulun 2004 – Inflammatory reaction exponentially increases local aromatase activity
57. Chan 2004 – Endometriosis is clonal
58. Marsh & Laufer 2005 and Cabana et al. 2010 – Inflammation may be a precursor, facilitator or early presentation.
59. Klemmt 2006, Akoum 2006, Klemmt 2007, Grümmer 2012, Klemmt 2018 – Changes in the eutopic (within the uterus in the normal location) endometrium can be associated with changes in ectopic endometrium (endometriosis).
60. Batt 2007 – Choristoma is a neoplastic Müllerian tissue in non-Müllerian areas
61. Ohlsson Teague 2009 – MicroRNA-regulated pathways associated with endometriosis
62. Acién 2012 – Accessory and cavitated uterine masses are non-inflammatory, organoid examples of how Müllerian remnants can appear.
63. Batt 2013, Laganà 2017 – Müllerianosis as an organoid remnant of Müllerian tissue in the normal area of embryologic Müllerian development. Organoid remnants are not the what is more commonly called endometriosis.
64. Batt 2013 – Hamartoma is a neoplastic Müllerian growth in the normal Müllerian area.
65. Brosens 2013 – Endometriosis is a progressive disease
66. Raposo 2013 – Extracellular vesicles involved in intercellular communication (signaling)
67. Becker 2014 – Six surgical phenotypes: clear, red, white, blue/black, brown, and vascular.
68. Kobayashi 2014 – Infectious precursors or infectious induction of endometriosis.
69. Gargett et al. 2014, Brosens 2015 – Perinatal retrograde dissemination is like Sampson but suggests an earlier occurrence shortly after birth.
70. Forte 2014 – Chromosomal anomalies and instability can alter gene expression
71. Khan 2014 – Three patterns of occult microscopic endometriosis (OME) based on patterns of Ber-EP4 (epithelial cell marker), CD10 (stromal cell marker), Calretinin (mesothelial cell marker), estrogen/progesterone receptors (ER/PR) and Ki-67 (cell proliferation marker).
72. Guo 2015 – Repeated tissue injury and repair (ReTIAR) due to cyclic bleeding in endometriosis.
73. Laux-Biehlmann 2015 – Pain due to activation of peripheral nerve endings in response to retrograde and extra-uterine menstruation
74. Koninckx 2016 – There are four phenotypic types of endometriosis: subtle, typical, cystic ovarian, and deep.
75. Nerune 2016 – Persistent Müllerian Duct Syndrome (PMDS) in men.

76. Laganà 2017 – "Unus pro omnibus, omnes pro uno" is a combination of many concepts into a process that begins during embryogenesis. Components include Hox (homeobox) genes, Wnt (wingless) genes, Müllerian derivatives and remnants, genital ridge leakage during organogenesis, human embryonic stem cells (hEmSC), endometrial stem progenitor cells (hESP), stem/progenitor cells residing in adult uterus, mesenchymal stem cells from bone marrow, and embryonic ectopic implantation.
77. Gordts 2017 – Whether the original cell comes from the endometrium, endometrial pale cells, other stem cells, bone marrow cells, embryonic cells, neonatal cells, adult cells or another source of endometrial or potentially endometrial cells is not important as the genetic and epigenetic changes that are associated with the specific phenotypes of endometriosis.
78. Vigano 2017 – Fibrotic condition with endometrial stroma and epithelium.
79. Liu 2017 – Epithelial-mesenchymal transition, fibroblast-to-myofibroblast transdifferentiation, smooth muscle metaplasia, fibrosis, vascularity, hormonal receptors, and proteins involved in epigenetic modifications. Differences may result from the different lesional microenvironments.
80. Makiyan 2017 – Congenital primordial germ cells remnants can be the source.
81. Anglesio 2017 – Cancer-associated driver mutations can be present in deep infiltrating endometriosis.
82. Klemmt 2018 – Other stem cell concerns include lack of apoptosis, evasion of immunosurveillance, angiogenesis, neurogenesis, exosomes, plasticity, stem cell signaling, aberrantly activated signaling pathways, stem cell migration, immunogenicity, peritoneal cavity homeostasis, dysregulation of Wnt and Hox genes, phenotype and microRNA analysis.
83. Brosens 2018 – Progression requires active neo-angiogenesis.
84. Panir 2018 – Non-coding RNA is associated with endometriosis.
85. Foster 2018, Luo 2018 – Endometrial implant survival, growth, evasion from apoptosis, and immune dysregulation are estrogen-dependent processes. Either autophagy or apoptosis can be a cause of cell death.
86. Baranov 2018 – A genetic program governs the origin of stem cells, transition into mesenchymal stem cells, invasion of the peritoneum and progression to endometriotic lesions. Baranov discusses the possibility that the stem cells could be disseminated during organogenesis or from the endometrium during retrograde menstruation.

Müllerian Remnant, Dispersion & Unus Pro Omnibus, Omnes Pro Uno.

Congenital (Müllerianosis (organoid), Mülleriosis (non-organoid), & secondary Müllerian system), dispersion (retrograde reflux, lymphatic dissemination, & hematogenous dissemination), and progenitor cell theories are all incomplete. Müllerian theory can explain origin in Müllerian areas but not the general distribution of endometriosis without relying on a congenital concept that could parallel metaplasia concepts in Lauchlan's (1972) secondary Mullerian system. Both Müllerian and secondary Müllerian can explain endometriosis in men. But, Mullerian theory does not explain why a congenital remnant would cause an inflammatory reaction. In contrast, congenital, non-midline, Müllerian remnants such as accessory and

cavitated uterine masses (Acién 2012) are non-inflammatory, organoid examples of how Müllerian remnants can appear. Also, Ron Batt considers Müllerian abnormalities to have eight forms. Four of those are congenital, and four are acquired. (Batt 2013) His presentation on this at the Endometriosis Foundation of America is at https://player.vimeo.com/video/125963026

Theories of the 19th & early 20th centuries did not investigate the intricate interactions that control or fail to control persistence, infiltration, and growth, in addition to the histologic, biochemical, and immunologic differences between endometrial and endometriotic lesions. Antonio Laganà's "Unus pro omnibus, omnes pro uno" (Med Hypotheses, 2017, 103:10-20) is a combination of many of those concepts into a process that begins during embryogenesis. Components include Hox (homeobox) genes, Wnt (wingless) genes, Müllerian derivatives and remnants, genital ridge leakage during organogenesis, human embryonic stem cells (hEmSC), endometrial stem progenitor cells (hESP), stem/progenitor cells residing in adult uterus, mesenchymal stem cells from bone marrow, and embryonic ectopic implantation.

Klemmt (2017) reviewed the molecular and cellular pathogenesis of endometriosis. A free, download is at https://www.researchgate.net/publication/316840373_Molecular_and_Cellular_Pathogenesis_of_Endometriosis

Infection

Marsh and Laufer (Fertil Steril 2005, 83:758-760) and Cabana et al. (Contemp Ped 2010, 27:22-27) did not exclude infection as the source of the inflammation in their publications. See "Subtle Inflammatory Lesions" below.

Gazvani et al. (J Endometriosis Pelvic Pain Disorders, 2013, 5:2-9) suggested that C. albicans may contribute to the pathogenesis of endometriosis by modulating cytokine production.

Kobayashi et al. (Mol Med Rep, 2014, 9, 9-15. DOI:10.3892/mmr.2013.1755) concluded that infection and sterile inflammation are involved in endometriosis development.

Khan et al. (J Endometriosis Pelvic Pain Disorders 2016, 8:2-7) found higher intrauterine microbial colonization with endometriosis.

Cicinelli et al. (Fertil Steril 2017, DOI 10.1016/j.fertnstert.2017.05.016) concluded that chronic endometritis might represent a facilitating factor in the development of endometriosis.

Canis et al. (J Gynecol Obstet Hum Reprod. 2017, 46(3):219-227) considered "occult pelvic inflammatory disease" as a potential initiating event for endometriosis."

But, none of these of clarified why infection did not result in overt pelvic inflammatory disease.

Subtle Inflammatory Lesions

An additional concern is raised by inflammatory lesions suggestive of endometriosis in adolescents and children. (Marsh and Laufer 2005, Cabana et al. 2010) If these are infectious, then antibiotics can treat active infection and potentially decrease long-term morbidity.

But, if these are sterile inflammatory lesions or if bacteria are present but part of a healthy microbiome, then antibiotics may interfere with the microbiome.

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