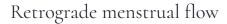
Inflammation in Endometriosis

Aetiology of endometriosis



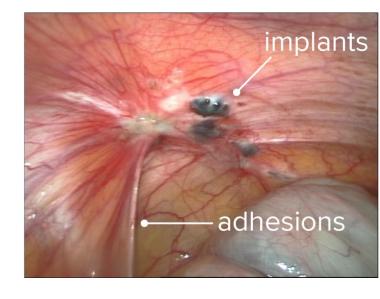
It is widely believed that endometriosis may be caused by menstrual blood flowing up the fallopian tubes and into the peritoneal cavity, causing deposits of cells that then implant on the peritoneal surfaces. This idea is now outdated.

Genetics

Endometriosis tends to run in families, so it is suspected there may be a genetic component.

Immune dysfunction

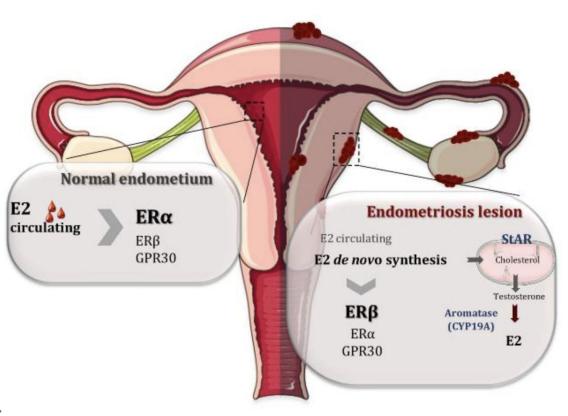
Newer research is pointing to the possibility of autoimmune dysfunction causing endometriosis. From a TCM perspective, this would fit well with endo patients who present as Yin Xu.



Oestrogen Receptors (ER)

\uparrow E2 and \downarrow P4

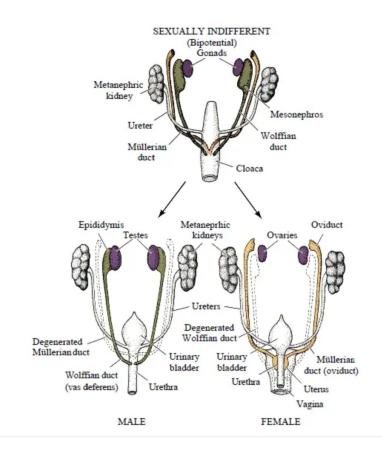
- 17β estradiol (E2) supports growth of endometrial lesions and associated inflammation and pain.
- E2 produced locally in endometriotic lesions.
- Endometrial lesions can synthesize E2 from cholesterol, whereas normal endometrium can't due to absence of CYP19A1 and StAR.
- Up regulation of E2-induced cell proliferation and inflammation increases lesions and disrupts endometrial receptivity.
- P4 normally inhibits E2-induced proliferation, however this is disrupted in endometriosis (oestrogen dominance).

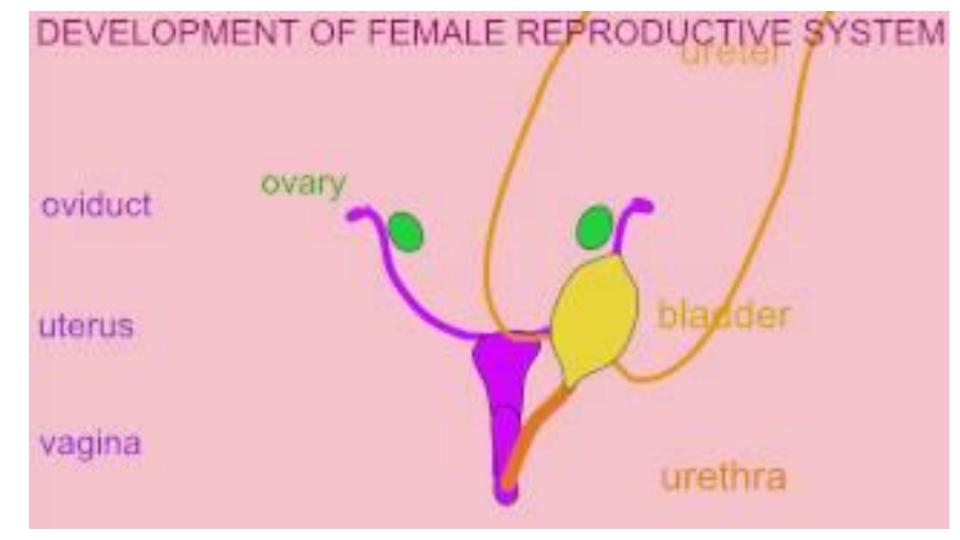


Sexual differentiation

Wolffian and Mullerian ducts

- Wolffian ducts become the male genital tract
- Mullerian ducts become the female genital tract
- Chromosomes determine which tract primarily develops
- Presence of Y chromosome causes sertoli cells to secrete
 AMH, which leads to apoptosis of the mullerian duct cells.
- Between 8 weeks and 4 months of development, cells are sensitive to AMH (important time to avoid exposure to environmental toxins and endocrine disruptors!!!)





Immune dysfunction in endometriosis



Gut dysbiosis

Dysbiosis disrupts immune function, leading to increased proinflammatory cytokines \rightarrow chronic inflammation \rightarrow increased adhesion and angiogenesis.



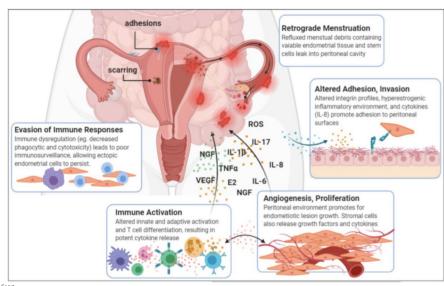
Decreased lactobacillus

Endometrial microbiota have been linked to \downarrow lactobacillus and \uparrow opportunistic pathogenic bacteria.



Mast cells

Endometriotic lesions create an environment suited to mast cell recruitment. The mast cells then release proinflammatory mediators → chronic pelvic pain and endometriosis progression.

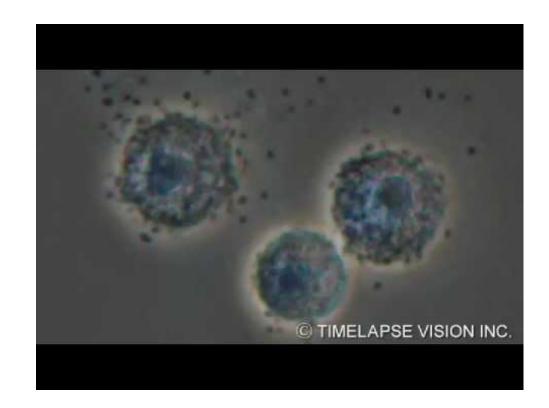


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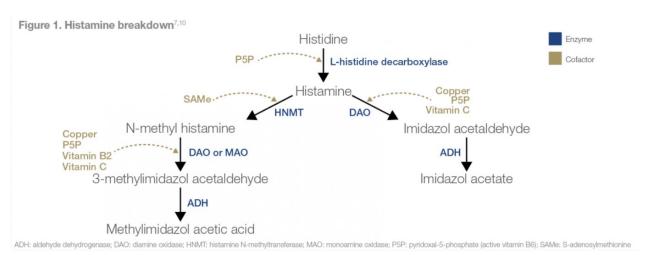
Mast Cells

Link between Endo & Mast Cells

- Mast cells present in endometrial lesions, activated and degranulated.
- Mast cell tryptase can activate protease activated receptors, found in the endometrium and endometriosis, which may contribute to angiogenesis and pain.
- Degranulation releases inflammatory mediators, attracting leukocytes, which increases the inflammatory response.



Histamine breakdown



Co-factors to support DAO & HNMT

- activated vitamin B6 (P5P)
- copper
- vitamin C
- SAMe

Standard approach to treatment

Pain medications

Non steroidal anti-inflammatory drugs such as Ibuprofen (Advil), or prostaglandin reducing drugs such as Naproxen (Naprogesic)



Laparoscopic surgery

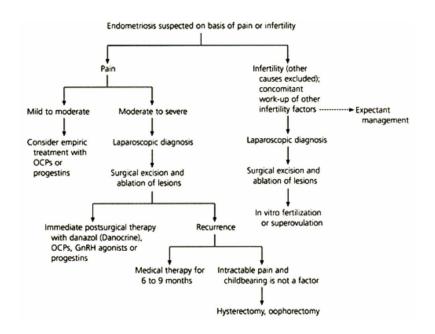
Surgery to remove endometrial lesions and adhesions, with the goal of reducing pain and increasing fertility.



Hormonal medications

OCP, progesterone, Gn-RH agonists and antagonists, aromatase inhibitors.

Treatment of Endometriosis



Holistic approach to treatment



Diet

Cruciferous vegetables to support removal of excess oestrogen.

Reduced caffeine, alcohol, energy drinks etc. Low histamine foods if needed.



Omega 3 fatty acids, PEA, Vit D, Vit C, B6.



Acupuncture

Numerous studies have shown acupuncture to be beneficial in reducing dysmenorrhea (period pain), and some studies even show it to be more effective than medications.

FOODS THAT SUPPORT **ESTROGEN METABOLISM**







broccoli

bok choy







kale



red cabbage