AAGL 26

55TH GLOBAL CONGRESS ON MIGS

BOSTON, MASSACHUSETTS
NOVEMBER 13-16, 2026

Syllabus

General Session II

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Nash S. Moawad, MD

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GS02: General Session II

Faculty: Linda Griffith, Cara R. King, Ted TM Lee, Hugh S. Taylor, Kelly Wright, Steven Young

MedTalk II - #225 - Why Excision Surgery Hurts Women

Presenter: Kelly Wright

Description: I am an excision surgeon, and excision surgery hurts women. This tongue-in-cheek MEDTalk explores the concept of hinging on a particular treatment may lead to lack of scientific progress in understanding and treating a disease. The focus on excision surgery for the past three decades has been important in teaching patients how to advocate for themselves and find a capable physician who validates their disease and suffering. However, it has hindered scientific progress through focusing on endometriosis as a single end-organ disease rather than allowing a mindset shift into endometriosis as a systemic inflammatory disease. We have seen progress in other comparative fields such as inflammatory bowel disease, inflammatory skin diseases, and even ovarian cancer. Comparing ourselves to physicians who treat these diseases, we could not imagine a colorectal surgeon who cuts out small intestine every time there's a Crohn's flare. Similarly, the focus on excision surgery has led to overuse and potential abuse of this technique, seen in the proliferation of cash pay surgeons and marketing on social media. This MEDTalk challenges the paradigm of our current treatment standards and offers hope for the future with an introduction to the current targets and treatments being studied right now - monoclonal antibodies, targeted receptor inhibitors, and even simple medications already available on the market. Let's reframe the discussion to think about what a career treating endometriosis as a chronic systemic inflammatory disease would look like, as perhaps we'll find great satisfaction in treating patients well over treating them with repeated surgeries.

Learning Objectives: *At the end of this course, the participant will be able to:* 1) Describe the concept of hinging in medicine and how it may hinder scientific progress; 2) Describe current cutting-edge research in the pathophysiology of endometriosis; and 3) Evaluate the potential role of different medications in the treatment of endometriosis.

Debate: 355 - What Drives Endometriosis?

Chair: Ted TM Lee
Moderator: Cara R. King
Faculty: Linda Griffith, Steven Young, Hugh S. Taylor

Description: Endometriosis remains one of the most complex and incompletely understood diseases in gynecology. Surgeons see and excise lesions, but the biologic forces that initiate, sustain, and amplify the disease remain deeply intertwined. Endometriosis may be driven by altered endometrial function, impaired decidualization, metabolic dysfunction, inflammation, immune dysregulation, neuroangiogenesis, estrogen dependence, progesterone resistance, and pain-system remodeling.
This 45-minute Main Stage Debate will bring together three internationally recognized scientific leaders to address one central question: What Truly Drives Endometriosis?
Rather than presenting three independent lectures, this session will be structured as a fast-paced debate among three complementary mechanistic models. Each speaker will make the strongest case for one biologic framework, followed by moderated crossfire, audience response polling, and clinical translation for the practicing endometriosis surgeon.
The goal is not to declare a single winning mechanism. The goal is to help the AAGL audience understand how disease biology should reshape surgical thinking, patient counseling, recurrence prevention, medical therapy, multidisciplinary care, and future research priorities.

Learning Objectives: *After attending this session, the participant will be able to:* 1) Compare leading mechanistic models of endometriosis pathogenesis, including metabolic/decidualization dysfunction, inflammatory and immune pathways, neuroangiogenesis, and endocrine/progesterone resistance mechanisms; 2) Explain how these mechanisms may contribute to pain, infertility, lesion persistence, recurrence, treatment response, and the mismatch between visual disease burden and patient symptoms; 3) Identify how mechanistic insights should influence surgical counseling, operative expectations, postoperative suppression, recurrence prevention, and multidisciplinary care; and 4) Recognize the limitations of a purely lesion-centered model of endometriosis and integrate biologic, inflammatory, hormonal, metabolic, neurologic, and patient-centered perspectives into comprehensive care.

Course Outline

9:30 am	FAAGL - Signature Awards	
9:45 am	MedTalk II - #225 - Why Excision Surgery Hurts Women	K. Wright
	Debate: #355 - What Drives Endometriosis?	
10:00 am	Welcome, Introduction and Course Overview	T. Lee
10:05 am	Baseline Audience Poll	C. King
10:08 am	Endometriosis, Decidualization and Metabolism: An Emerging Model and Implications for Our Patients	S. Young
10:15 am	Endocrine Mechanisms: Estrogen/Progesterone Signaling and Progesterone Resistance	H. Taylor
10:22 am	Inflammation, Immunology, Pathogenesis, and Neuroangiogenesis	L. Griffith
10:29 am	Moderated Debate	T. Lee
10:39 am	Audience Re-Poll and Clinical Translation	C. King
10:42 am	Final Synthesis	T. Lee
10:45 am	Adjourn	

ADA Statement: In accordance with the Americans with Disabilities Act (ADA), please contact CMEsupport@aagl.org should you require any special assistance.

Disclosure of Relevant Financial Relationships

As an ACCME-accredited provider, AAGL is responsible for ensuring that accredited education is fair, balanced, free of commercial bias, and supports safe, effective patient care. AAGL maintains control over all aspects of educational planning, content, faculty selection, delivery, and evaluation.

All individuals in a position to control educational content are required to disclose financial relationships with ACCME-defined ineligible companies within the past 24 months. AAGL identifies and mitigates all relevant financial relationships before individuals assume their respective roles. Educational content is also subject to independent peer review by individuals without relevant financial relationships.

Disclosure information and acknowledgment of any commercial support are provided to learners before the activity to promote transparency and maintain the independence and integrity of the education.

Asterisk (*) denotes no financial relationships with ACCME-defined ineligible companies to disclose.

PLANNER DISCLOSURES

The following members of AAGL have been involved in the educational planning and/or review of this course (listed in alphabetical order by last name).

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Jamal Mourad, DO – Proctor: Intuitive Surgical
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Linda Bell, AAGL Admin Support*
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SCIENTIFIC PROGRAM COMMITTEE

Nash Moawad, MD, MS, Chairman – Consultant (ended 12/2024): Medtronic, Womed, Integra Life Sciences
Amani Harris, MD*
Ted Lee, MD – Advisory/Consultant: Johnson & Johnson, Regeneron Pharmaceuticals
Nucelio Lemos, MD, PhD – Speakers Bureau: Pfizer, Inc., Medtronic; Advisory Board: Knight Therapeutics, Vecfer Pharma
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Angela Chaudhari, MD - Consultant: Tag/Dream Medical, Johnson & Johnson; Speakers Bureau: Johnson & Johnson
Linda D. Bradley, MD, Medical Director*
Linda Michels, Executive Director, AAGL*

COURSE CHAIR DISCLOSURE:

Ted TM Lee, MD – Consultant: Anuat, Jmees, Johnson & Johnson, Regeneron

COURSE MODERATOR DISCLOSURE:

Cara R. King, DO, MS – Consultant: Medtronic

FACULTY DISCLOSURES

The following faculty have agreed to support their presentations and clinical recommendations with the best available evidence from the medical literature (in alphabetical order by last name).

Linda Griffith, MD – Consultant: EMm, RA Capital; Contracted Research: Amgen, Novo Nordisk
Cara R. King, DO, MS – Consultant: Medtronic
Ted TM Lee, MD – Consultant: Anuat, Jmees, Johnson & Johnson, Regeneron
Hugh S. Taylor, MD – Contracted Research: AbbVie, Harvard Apparatus
Kelly Wright, MD – Consultant: Karl Storz, Aqua Therapeutics
Steven Young, MD, PhD – Consultant: AbSci; Royalty: CiceroDx

MITIGATION OF RELEVANT FINANCIAL RELATIONSHIPS

All relevant financial relationships have been mitigated.

Drs. Lee, Lemos, Mikhail, and Chaudhari's planning decisions, as part of the Scientific Program Committee (SPC), were peer reviewed by other planners on the SPC who had disclosed no financial relationships with ineligible companies, and found to be fair, balanced, and free of commercial bias. Dr. Moawad's financial relationships ended 12/2024, thus mitigating them. For most of his AAGL Professional Education Committee service, Dr. Mourad had no financial relationships to disclose. He recently disclosed a new relationship as a Proctor for Intuitive Surgical. Since then, he has not participated in activities involving control or influence over accredited educational content; therefore, no additional mitigation was necessary. Dr. Lee's role as Course Chair was limited primarily to course oversight and moderation, rather than development of educational content. Any planning decisions or content under his influence were subject to independent peer review by individuals without relevant financial relationships. The moderator and debate panelists, Drs. Griffith, King, and Lee, did not develop or submit formal presentation content for review. However, all participants attested to maintaining balance, independence, objectivity, and scientific rigor; supporting clinical recommendations with the best available evidence; and ensuring discussions remain free of commercial bias.The presentation content for course faculty, Drs. Taylor, Wright, and Young, has been peer reviewed and found to be fair, balanced, and free from commercial bias.

Why excision surgery hurts women

Kelly Wright, MD, FACOG, FACS
Vice Chair, Gynecology
Division Director, Minimally Invasive Gynecologic Surgery
Program Director, CBGF
Cedars-Sinai

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Disclosures

- Financial relationships:

Consultant: Aqua Therapeutics, Karl Storz

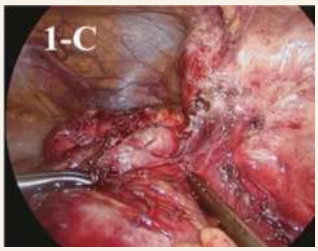
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Objectives

- 1) Describe the concept of hinging in medicine and how it may hinder scientific progress
- 2) Evaluate the current knowledge gaps in the pathophysiology of endometriosis
- 3) Develop research opportunities for progressing our field

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A tale of two conditions …





4

1940s - Breakthrough

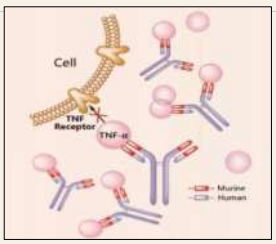


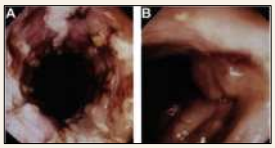
Swedish physician Nanna Svartz was treating RA patients with sulfasalazine.

Those who also had UC experienced relief of colitis symptoms.

5

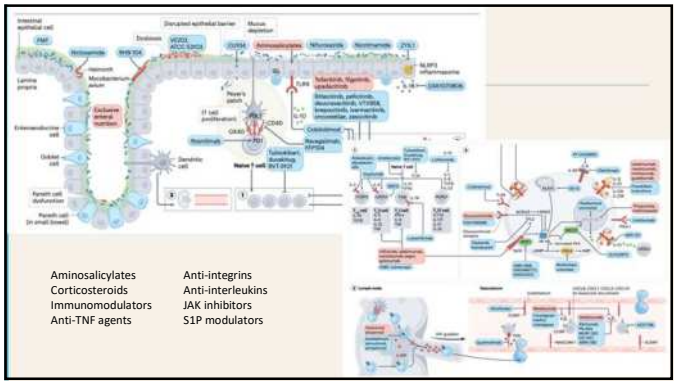
1998 - Infliximab





van Dulleman HM, van Deventer SJ, Hommes DW, et al. Gastroenterology, volume 109, issue 1, 1995
Kumar M, Murugesan S, Ibrahim N, et al. J Transl Med. 2024 Mar 16;22(1):284.

6



7

Where are they now

- EIGHT different classes of medications
- Surgery has decreased by 37% in UC and 26% CD
- Surgery remains for refractory, fibrostenotic, perianal disease



Argeny, Stanislaus & Stift, Anton & Bergmann, et al. European Surgery. 50. 177-182.

8

Precision medicine

- Biomarkers
  - predict disease complications and bowel damage (to guide step-up or top-down strategies)
  - predict the efficacy (or lack thereof) of therapies and
  - assess the risk of adverse effects, whether in the first-line of treatment or after its failure
- "Treat to target" toward a composite endpoint:
  - Mucosal healing + GI symptoms
  - Pain is considered and treated separately

9

The current gold standard for diagnosis [and treatment] does little to facilitate early diagnosis or avoid long-term sequelae.

- Lancet, 2025

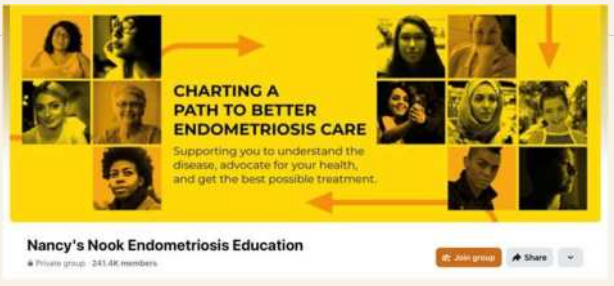
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Where are we now

- 32 y/o G1P1
  - 22 prior laparoscopies including TLH/BSO
  - Last 3 complete peritonectomies with negative pathology
  - Multiple hospital admissions for pain every year
  - MS Contin
  - Told "need cleanout every year"
- 49 y/o G2P2
  - 11 prior excision surgeries
  - TLH/BSO
  - Sigmoidectomy
  - Chole + appy
  - No surgery ever helped her pain
  - Percocet
- 42 y/o G1P1
  - Undergoing IVF, 6 cycles -> 5 euploid embryos
  - Stage 2 endometriosis and small endometrioma
  - Advised that surgery would likely not improve implantation rate
  - Pain related to PFD – started PT and had significant improvement
  - Went to another surgeon, complete peritonectomy and full excision, Lupron afterward
  - Menses never resumed

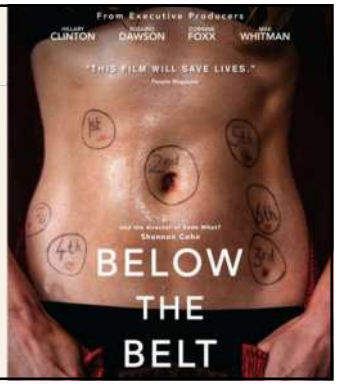
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12

Hinging

- **Hinging disease management on a categorical diagnosis or treatment slows progress because the label itself becomes an information bottleneck**
- When therapy is organized around the label, patients with mechanistically distinct disease are lumped together under one name



13

We focused on excision

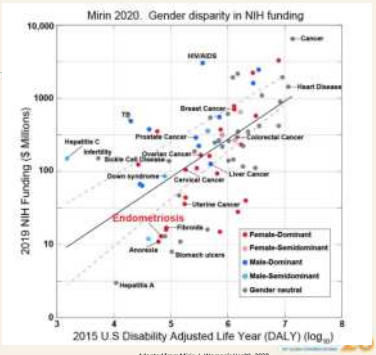




E Courtois, The Jackson Laboratory, U Conn

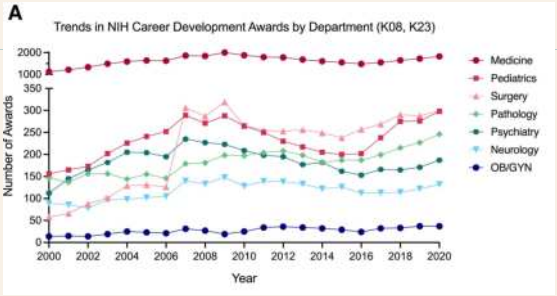
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We MUST move on



Adapted from Mirin, J. Women's Health, 2020

15



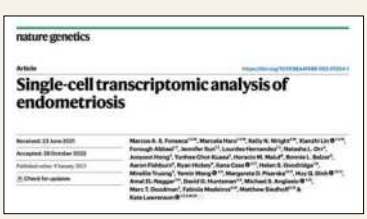
Parchem J, Townsel C, Wernimont S, et al. More than grit: growing and sustaining physician-scientists in obstetrics and gynecology. Am J Ob Gynecol. 2022.

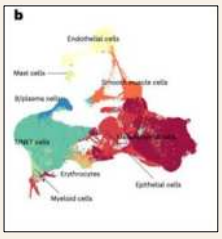
16



17

We MUST collaborate with scientists





18

We MUST move on from surgery to diagnose





19

We MUST look for new measures of progression

Assessment of endometriosis angiogenesis using ^{99m}Tc-maraciclatide imaging (DETECT): a single-centre, exploratory, open-label, non-randomised, phase 2 study

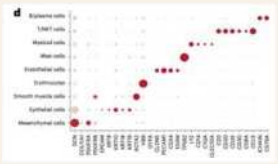


20

We MUST start targeted drug trials

- Drug trials
- Tissue studies for patients already on targeted therapies
- Study the leading invasive edge of endometriosis





- CGRP (erenumab, galcanezumab)
- TNF (adalimumab, infliximab, golimumab)
- IgE (omalizumab)
- IL-4 (dupilumab)
- IL-17 (ixekizumab)
- IL-23 (risankizumba, ustekinumab)

21

We need our own GOG



22

What can we learn from IBD?

- Trial drugs that are already used for other conditions
- Work based on mechanistic drivers of disease – we should be working together, not in silos
- Treat toward a composite endpoint
  - Disease progression
  - QoL
- Pain should be treated independently of the disease

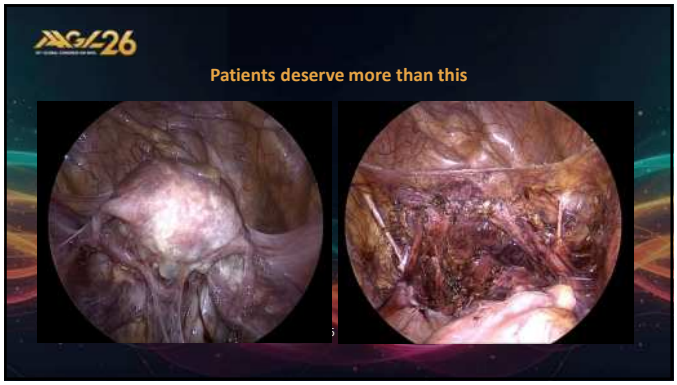
23

Toward precision medicine



Diseases should be defined by mechanism, linked directly to treatment, and continuously updated as knowledge grows

24



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26

Endometriosis Pathogenesis: A Central Role for Metabolism and Decidualization

Steven L Young MD, PhD
F. Bayard Carter Distinguished Professor
Reproductive Endocrinology and Infertility Division Director
Duke Obstetrics and Gynecology

1

Disclosures

- I am co-inventor on the intellectual property underlying the ReceptivaDX™ endometrial test for endometriosis.
- I have served on an advisory board for AbSci, regarding possible development of an endometriosis therapy.

2

Less than 7 minutes to convince you that decidualization and metabolic alterations are the central cause of endometriosis

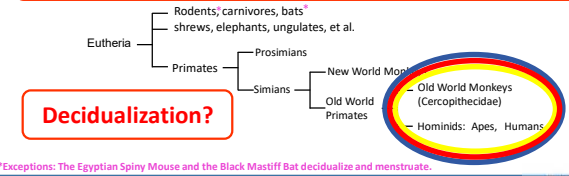
- Evolutionary argument for decidualization bolstered by correlational human data.
- Surprising monkey data, bolstered by human and mouse data, that strongly implicates altered energy metabolism as an important dysregulator of decidualization.
- Wrap-up with a working model to consider.

3

Evolutionary Biology of Endometriosis

Which placental animals (Eutheria) get endometriosis?
Which animals menstruate?
Which animals' endometrial stroma decidualizes without a pregnancy?

The set of species that Menstruate and the set that Decidualize without a pregnancy are identical, and only those species can get endometriosis!

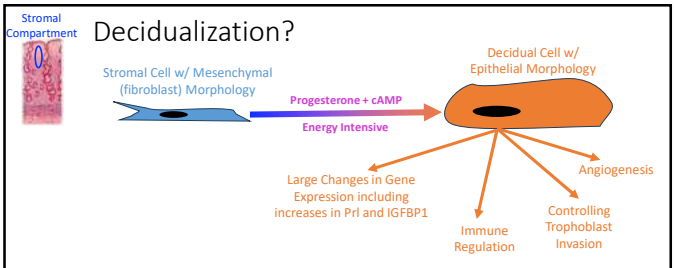


*Exceptions: The Egyptian Spiny Mouse and the Black Mastiff Bat decidualize and menstruate.

Chavan AR et al. Placenta 2016; Mor G et al. Ann of the NY Acad Sci 2011; Lynch VJ et al. Cell Reports 2015

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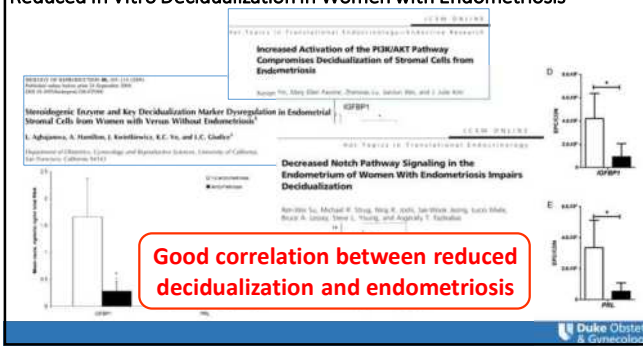
Decidualization?



If decidualization is causally linked to endometriosis, we should see altered decidualization in women with endometriosis.

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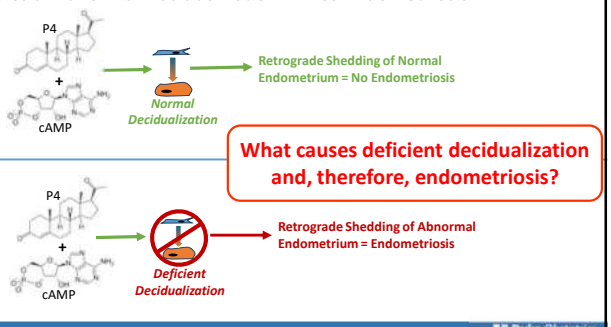
Reduced In Vitro Decidualization in Women with Endometriosis



Good correlation between reduced decidualization and endometriosis

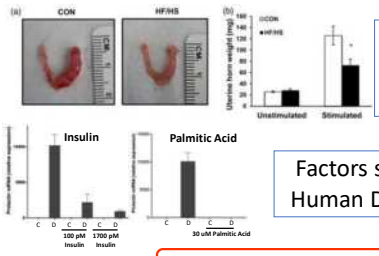
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Hypothesis: Abnormal Decidualization Drives Endometriosis



7

Obesity is Linked to Impaired Stromal Decidualization



An Obesogenic Diet Impairs Mouse Decidualization In Vivo

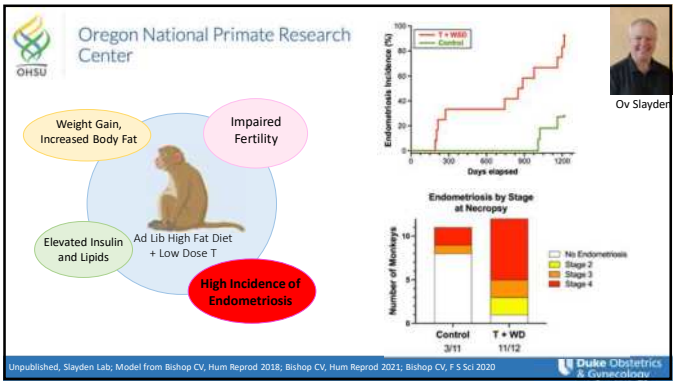
Factors seen in Obesity Impair Human Decidualization In Vitro

Can an Obesogenic Diet Cause Endometriosis?

Rhee JS, Hum Reprod 2016; Young Lab, Unpublished

8

Oregon National Primate Research Center

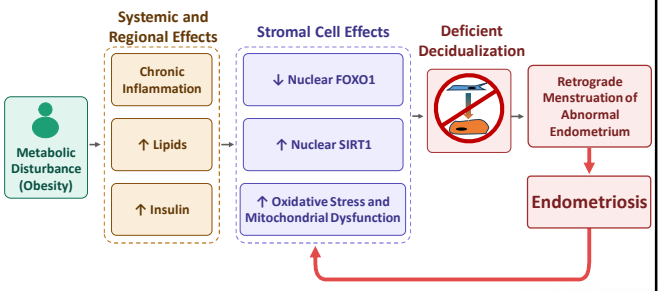


Ov Slayden

Unpublished, Slayden Lab; Model from Bishop CV, Hum Reprod 2018; Bishop CV, Hum Reprod 2021; Bishop CV, F S Sci 2020

9

Working Model



Young, Slayden, Rao, Schust, Marbrey - Unpublished

10

A wise man's question contains half the answer.

- Solomon Ibn Gabirol (aka Solomon ben Judah)
Andalusian Jewish Poet and Philosopher (1021 – 1070)

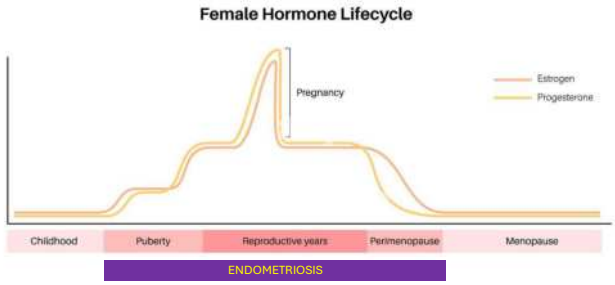


11

Endocrine Mechanisms: Estrogen/Progesterone Signaling and Progesterone Resistance

Hugh S. Taylor, MD
Anita O'Keeffe Young Professor
Chair Department of Obstetrics, Gynecology and Reproductive Sciences
Yale School of Medicine

1

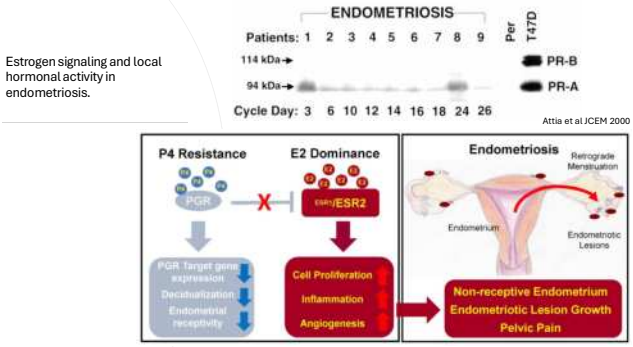


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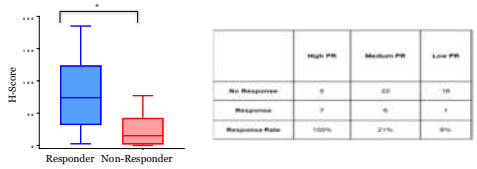
3

Estrogen signaling and local hormonal activity in endometriosis.



4

Progesterone Resistance Mediated by Altered PR Expression

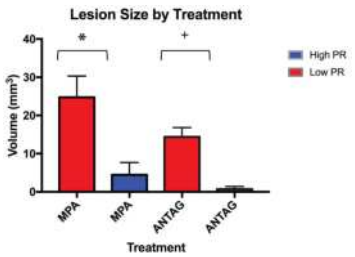


	High PR	Medium PR	Low PR
No Response	0	22	16
Response	7	6	1
Response Rate	100%	21%	6%

Flores VA et al . Progesterone Receptor Status Predicts Response to Progestin Therapy in Endometriosis. JCEM. 2018

5

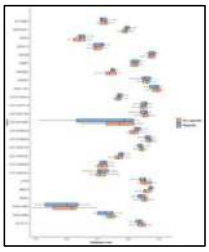
Progesterone resistance and its relevance to lesion persistence, medical therapy, and treatment response.

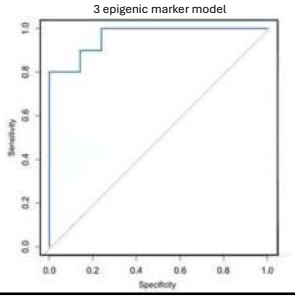


Flores V et al **Progesterone receptor status predicts aggressiveness of human endometriotic lesions in murine avatars** FS Sci. 2025;6(1):65-72.

6

Epigenetic Biomarkers of Progesterone Resistance in Peripheral Blood



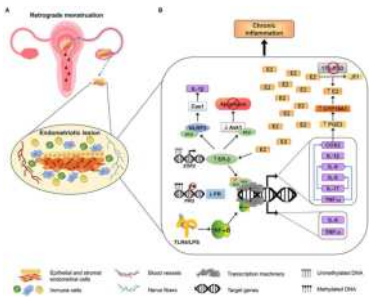


Cevik et al Biomark Res. 2026 Mar 18;14(1):32.

7

How endocrine mechanisms interact with inflammation, immune dysfunction, and pain biology.

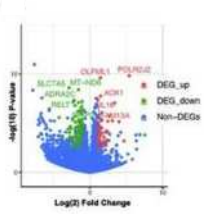
- Loss of Immunosuppression by PR
- Increase ER beta mediated inflammation
- Painful Lesions are more Inflammatory



8

Endometriosis pain: genes linked to inflammation distinguish symptomatic from asymptomatic disease

- Mol Hum Reprod 2026;32(4):gaag055.





9

What surgeons should understand about postoperative hormonal suppression and recurrence risk.

- The disease that you see is enriched in P resistant subtype. All endo is partially P resistant- varies in severity.
- P resistant disease is not just resistant to P- more aggressive disease.
- More inflamed and more painful.
- More prone to recurrence.

10

Whether surgery alone can ever be considered biologically complete treatment.

- No- high recurrence risk.
- Stop menstruation (IUD, continuous OC, GnRH antag)
- Long term suppression is key. Manage as if progestin resistant.

11

"If this model is correct, surgeons should…"

- Consider hormonal therapy as adjuvant to surgery- not as two mutually exclusive options.
- All endometriosis is at least partially P resistant. May need to use high doses of progestins (limited by side effects) or GnRH antagonists.
- Reduce growth of microscopic lesions as well as inflammation and systemic effects.
- Be aggressive with post operative therapy.

12

CULTURAL AND LINGUISTIC COMPETENCY & IMPLICIT BIAS

The California Medical Association (CMA) outlines standards for Cultural Linguistic Competency and Implicit Bias in CME. The goal of the standards is to support the role of accredited CME in advancing diversity, health equity, and inclusion in healthcare. These standards are relevant to ACCME-accredited, CMA-accredited, and jointly accredited providers located in California. AAGL is ACCME-accredited and headquartered in California.

CMA developed the standards in response to California legislation (Business and Professions (B&P) Code Section 2190.1), which directs CMA to draft a set of standards for the inclusion of cultural and linguistic competency (CLC) and implicit bias (IB) in accredited CME.

The standards are intended to support CME providers in meeting the expectations of the legislation. CME provider organizations physically located in California and accredited by CMA CME or ACCME, as well as jointly accredited providers whose target audience includes physicians, are expected to meet these expectations which began on January 1, 2022. AAGL has been proactively adopting processes that meet and often exceed the required expectations of the legislation.

CMA CME offers a variety of resources and tools to help providers meet the standards and successfully incorporate CLC & IB into their CME activities, including FAQ, definitions, a planning worksheet, and best practices. These resources are available on the CLC and IB standards page on the CMA website.

Important Definitions:

Cultural and Linguistic Competency (CLC) – The ability and readiness of health care providers and organizations to humbly and respectfully demonstrate, effectively communicate, and tailor delivery of care to patients with diverse values, beliefs, identities and behaviors, in order to meet social, cultural and linguistic needs as they relate to patient health.

Implicit Bias (IB) – The attitudes, stereotypes and feelings, either positive or negative, that affect our understanding, actions and decisions without conscious knowledge or control. Implicit bias is a universal phenomenon. When negative, implicit bias often contributes to unequal treatment and disparities in diagnosis, treatment decisions, levels of care and health care outcomes of people based on race, ethnicity, gender identity, sexual orientation, age, disability and other characteristics.

Diversity – Having many different forms, types or ideas; showing variety. Demographic diversity can mean a group composed of people of different genders, races/ethnicities, cultures, religions, physical abilities, sexual orientations or preferences, ages, etc.

Direct links to AB1195 (CLC), AB241 (IB), and the B&P Code 2190.1:

Bill Text – AB-1195 Continuing education: cultural and linguistic competency.
Bill Text – AB-241 Implicit bias: continuing education: requirements.
Business and Professions (B&P) Code Section 2190.1

CLC & IB Online Resources:

Diversity-Wheel-as-used-at-Johns-Hopkins-University-12.png (850×839) (researchgate.net)
Implicit Bias, Microaggressions, and Stereotypes Resources | NEA
Act, Communicating, Implicit Bias (racialequitytools.org)
https://www.contemporaryobgyn.net/view/overcoming-racism-and-unconscious-bias-in-ob-gyn
https://pubmed.ncbi.nlm.nih.gov/34016820/
https://hr.ucdavis.edu/departments/learning/certificate/implicit-bias
https://pmc.ncbi.nlm.nih.gov/articles/PMC7011228/