Vaginal Dysbiosis with HPV
Cervical Microbiome Profiling

Clinical Interpretation Guide

Lactobacillus Species Classification and Its Association with Vaginal Dysbiosis

Optimal Protective

Lactobacillus crispatus
- 9% Dysbiotic

Protective

Lactobacillus gasseri
Lactobacillus jensenii
- 17% - 20% Dysbiotic

Transitional

Lactobacillus iners
- 66% Dysbiotic

Vaginal Microbiome Status and Its Association with High-Risk HPV Persistence

	Protective Microbiome	Transitional Microbiome	Dysbiotic Microbiome
High-Risk HPV Positive (non 16/18)	Moderate Risk	Elevated Risk	High-Risk
HPV 16/18 Positive	Elevated Risk	High-Risk	Very High-Risk

Clinical Application

Fertility & ART
- Assessing endometrial receptivity
- Improving IVF Outcomes

Pregnancy Outcomes
- Preterm Birth Risk Stratification
- Monitoring for infection/dysbiosis

STI Risk Assessment
- Increased susceptibility
- Co-infection management

Therapeutic Considerations

Treatment Approaches
- Antibiotic stewardship based on profiling
- Lifestyle modifications
- Biofilm disrupting strategies where indicated

Probiotics
- Specific strains (e.g., *L. crispatus* to restore optimal flora
- Targeted interventions for dybiosis

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The Vaginal Microbiome and HPV

How Dysbiosis Drives Acquisition, Persistence, and Cervical Disease Progression

Women with a Lactobacillus-dominant vaginal microbiome (CST I–III) maintain acidic pH (3.8–4.5), robust antiviral defenses, and intact epithelial barriers. When this ecology shifts toward dysbiosis (CST-IV)—characterized by depletion of protective Lactobacillus and overgrowth of anaerobic pathogens, the cervicovaginal environment becomes permissive to HPV infection and persistence.

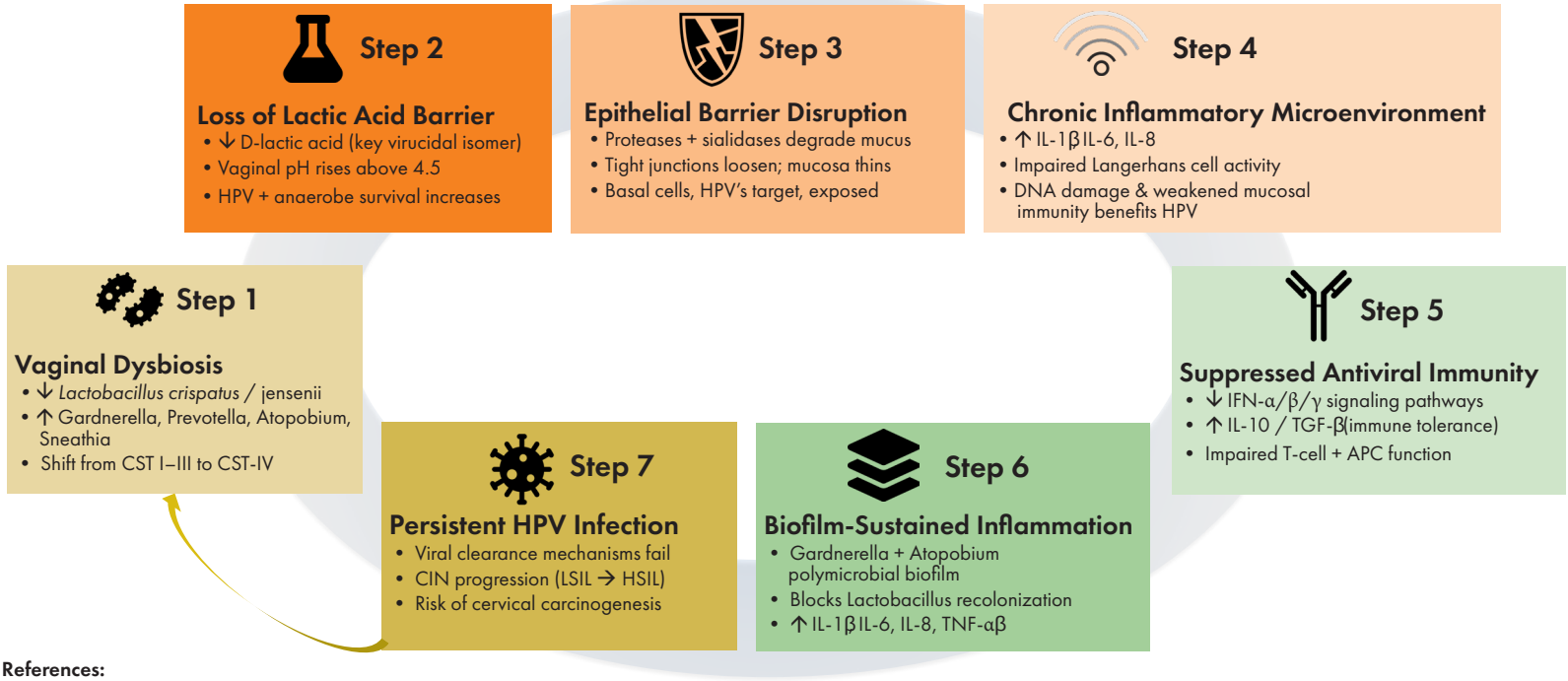
Clinical data: The rate of CST-IV microbiome doubles in LSIL, triples in HSIL, and quadruples in invasive cervical cancer, while L. crispatus dominance (CST-I) decreases with disease severity. L. iners is specifically associated with a higher tendency to shift toward dysbiotic states in HPV-positive women.

FIVE MECHANISMS BY WHICH DYSBIOSIS PROMOTES HPV

Mechanism	What Happens	Effect on HPV
Loss of Lactic Acid Barrier	Lactobacillus depletion → reduced lactic acid → pH rises above 4.5. D-lactic acid (the key antiviral isomer) drops sharply.	Higher pH increases HPV and anaerobe survival. Loss of direct D-lactic acid virucidal activity.
Epithelial Barrier Disruption	BV-associated bacteria (Gardnerella, Prevotella, Sneathia) release proteases, sialidases, and mucin-degrading enzymes. Tight junctions loosen; mucus layer thins.	Basal epithelial cells, HPV's primary target for infection, become physically exposed to viral particles.
Chronic Inflammatory Microenvironment	BV organisms trigger elevated IL-1ß, IL-6, IL-8 and chronic low-grade inflammation. Langerhans cells are recruited but functionally impaired.	Oxidative stress causes DNA damage. HPV benefits from muted, dysregulated mucosal immunity.
Suppressed Antiviral Immunity	Dysbiosis reduces IFN-α/β/γ and antiviral pathways. Increased IL-10/TGF-β dampen HPV-targeting immunity. Impaired antigen presentation and cytotoxic T-cell recruitment.	Viral clearance mechanisms are weakened across both innate and adaptive arms. HPV persistence is directly favored.
Biofilm-sustained Inflammation	Gardnerella + Atopobium form polymicrobial biofilms on cervical epithelium. Biofilm blocks Lactobacillus recolonization and maintains chronic epithelial turnover.	Chronic turnover exposes more basal target cells. Immunosuppressed cervical niche protects HPV from clearance.

THE SELF REINFORCING CYCLE

These mechanisms create a vicious cycle: **dysbiosis → loss of lactic acid → rising pH → epithelial degradation → suppressed antiviral immunity → HPV gains access to basal cells → persistent infection → chronic inflammation → further Lactobacillus suppression**. HPV persistence correlates strongly with vaginal pH > 4.5, a hallmark of dysbiosis. Metabolic byproducts (amines, reactive oxygen species) compound epithelial DNA damage and sustain the inflammatory state that HPV exploits.



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