Background The vaginal microbiota can be clustered into six community state types (CSTs): 4 are dominated by Lactobacillus iners, L. crispatus, L. gasseri, L. jensenii, and 2 lack significant numbers of Lactobacillus spp. (termed CST IV-A and IV-B). CST IV-A is characterised by a diverse assemblage of strict anaerobes, while CST IV-B has higher proportions of the genera Atopobium, Gardnerella, among others. We sought to describe the relationship between vaginal microbiota and human papillomavirus (HPV) detection.

Methods Thirty-two reproductive-age women self-collected mid-vaginal swabs twice-weekly for 16 weeks (n=937 samples). Participants reported behaviours on daily diaries. Vaginal bacterial communities were characterised by pyrosequencing of barcoded 16S rRNA genes (V1-V2 region). Each swab was tested for 37 types of HPV using the Roche HPV Linear Array genotyping test. The effects of CSTs on the rate of transition between HPV-negative and HPV-positive states were assessed using continuous-time Markov models. Additive mixed effects logistic regression and additive mixed effects Poisson models were used to model high-risk HPV (hrHPV) and count positive states were assessed using continuous-time Markov models.

Results Participants had an average of 29 (range 25–35) samples tested for HPV, with point prevalence ranging from 58% to 77% and 16-week period prevalence of 84%. CST was significantly associated with changes in HPV status (p < 0.001). L. gasseri-dominated CSTs had the highest HPV remission rate (HR-positive to no detection) and CST IV-B had the slowest rate compared to L. crispatus-dominated CSTs (adjusted transition rate ratio (aTRR):7.58, 98% CI: 1.77–32.42 and aTRR:0.31, 98% CI: 0.09–1.05, respectively). Detection of hrHPV and count of different HPV types were highest in the middle of the menstrual cycle.

Conclusion Vaginal microbiota dominated by L. gasseri were associated with increased clearance of detectable HPV. A mid-cycle increase in HPV detection suggests a role for sex hormones in modulating latent infection.

Objective While Neisseria gonorrhoeae (GC) and Chlamydia trachomatis (CT) are known to cause PID, many women with clinical signs and symptoms of PID and histologic evidence of endometritis have neither of these pathogens. Our objective was to describe microorganisms in the upper genital tract of women with PID, and to evaluate their association with histologic endometritis.

Methods Women presenting with symptoms and meeting the CDC diagnosis of PID had an endometrial biopsy obtained by Pipelle, and the tissue was split for microbiological and histological assessment. Cultivated microorganisms were identified using phylogenetic and genotypic characteristics. Fisher’s exact tests were used to assess the association between microorganisms and endometritis (plasma cells ± neutrophils).

Results Of 156 women with clinical PID, 55 (40%) had histologic evidence of endometritis, and endometrial GC and/or CT was associated with endometritis (29% vs. 6%, P < 0.001). In addition to STIs, a broad range of bacteria representing 63 different species were recovered from 53 (39%) of the endometrial biopsy samples, including 8 novel species. The recovery of any non-GC/non-CT organisms from the endometrium was associated with histologic endometritis (53% vs. 30%, P = 0.008). Both G. vaginalis (53% vs. 16%, P = 0.01) and A. vaginae (22% vs. 3%, P < 0.001) were associated with histologic endometritis. Other anaerobic bacteria associated with bacterial vaginosis including Prevotella timonensis, P. amnii and Peptostreptococcus harei were also more frequent in the endometrium of women having endometritis (11% vs. 3%, P = 0.06) but this did not reach statistical significance. After excluding women having GC and/or CT, A. vaginae was still independently associated with endometritis (17% vs. 3%, P = 0.05).

Conclusions The recovery of non STIs from the endometrium is associated with histologic endometritis among women with clinically diagnosed PID. A. vaginae may play an etiologic role in PID and merits further evaluation for its role in nongonococal/nonchlamydial PID.