

## **Abstract:**

Abstract Among 302 female sex workers in Nairobi, Kenya, who were followed for 17.6 +/- 11.1 months, 146 had one or more infections with *Chlamydia trachomatis*; 102 had uncomplicated cervical infection only, 23 had *C. trachomatis* pelvic inflammatory disease (PID), and 21 had combined *C. trachomatis* and *Neisseria gonorrhoeae* PID. As determined by multivariate logistic regression analysis, risk factors for *C. trachomatis* PID included repeated *C. trachomatis* infection (odds ratio [OR], 1.8; 95% confidence interval [CI], 1.3-2.4; P = .0004), antibody to *C. trachomatis* heat-shock protein 60 (OR, 3.9; CI, 1.04-14.5; P = .04), oral contraceptive use (OR, 0.28; 95% CI, 0.08-0.99; P = .048), and number of episodes of nongonococcal nonchlamydial PID (OR, 1.7; 95% CI, 1.1-2.7; P = .02). Among human immunodeficiency virus (HIV)-seropositive women, a CD4 lymphocyte count of <400/mm<sup>3</sup> was an additional independent risk factor for *C. trachomatis* PID (OR, 21.7; 95% CI, 1.2-383; P = .036); among HLA-typed women, HLA-A31 was independently associated with *C. trachomatis* PID (OR, 5.6; 95% CI, 1.1-29.4; P = .043). The results suggest an immune-mediated pathogenesis for *C. trachomatis* PID.