The importance of understanding pelvic inflammatory disease as a polymicrobial infection



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In their recent exemplary study, Alexiou and colleagues found that symptomatic- but not asymptomatic-Chlamydia trachomatis (Ct) infections were associated with pelvic inflammatory disease and tubal infertility.1 The authors suggested that differences in Ct virulence or host response may explain this. We would like to suggest that the polymicrobial nature of pelvic inflammatory disease is an alternative explanation.2 Whilst Ct and Neisseria gonorrhoeae (Ng) are clearly causally associated with pelvic inflammatory disease, detailed microbiological analyses have found them present in the fallopian tubes of a minority of patients with pelvic inflammatory disease.^{2,3} Furthermore, even when present they are found together with a range of bacterial vaginosis (BV) associated bacteria such as Atopobium, Sneathia, and Leptotrichia spp.^{2,3} Prospective studies have found that both BV and Ct are independently associated with incident pelvic inflammatory disease.4 In addition, a recent randomised controlled trial found that the addition of metronidazole (to ceftriaxone plus doxycycline) to cover BV bacteria was associated with better clinical outcomes in patients with pelvic inflammatory disease.5 These and other findings suggest that most Ct infections do not result in pelvic inflammatory disease and when they do, they do it as part of a polymicrobial infection with various BV associated bacteria. If it is the combination of Ct, Ng and BV associated bacteria that is instrumental in causing pelvic inflammatory disease

and tubal infertility, then this reduces the probability that simply screening for Ct will reduce these outcomes. This in turn provides a further justification for the recent decision by the Netherlands to stop routine screening for Ct.

Declaration of interests

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