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Absence of *Mycoplasma genitalium* in eye samples from a trachoma-endemic area of Tanzania

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In 1957, *Chlamydia trachomatis* (non-genital serovars A, B, Ba and C) was recognized as the etiological agent of trachoma.¹ *C. trachomatis* genital serovars D to K cause ophthalmia neonatorum and conjunctivitis in older children and adults. Trachoma remains endemic in many countries of sub-Saharan Africa, the Middle East and other focal areas of the developing world.² Nucleic acid amplification testing (NAAT) represented a major advance in the diagnosis of *C. trachomatis* infection among patients with trachoma, as cell culture is rarely available in endemic countries. However, even using this most sensitive technology, one fifth of patients with severe trachoma and up to one half of those with milder infections do not have *C. trachomatis* nucleic acids in eye samples.³ The higher the prevalence of trachoma, the higher the proportion of patients in whose samples *C. trachomatis* is found, but even in hyperendemic areas one third of children with trachoma have negative *C. trachomatis* NAAT.⁴ This is attributed to i) kinetics of *C. trachomatis* eye infections, with clinical signs (attributable to the host's immune response to infection) persisting after the infection has resolved; ii) factors influencing the accuracy of the test, including sampling variation, inhibition of amplification and aberrant *C. trachomatis* strains; and iii) the possible ability of other pathogens to elicit a conjunctival response resembling that produced by *C. trachomatis*.³⁻⁵

During the last few years, *Mycoplasma genitalium* has been recognized as a common sexually transmitted pathogen in sub-Saharan Africa, to a large extent because NAAT has facilitated its detection. In West Africa, *M. genitalium* is a cause of urethritis or cervicitis at least as common as *C. trachomatis*.⁶⁻⁸ As an explanation for the discrepancy between clinical trachoma and *C. trachomatis* NAAT, and given its propensity to mimic *C. trachomatis* infections in cervical, endometrial and urethral epithelial cells, we hypothesized that *M. genitalium* might also be able to infect conjunctival epithelial cells and be an aetiological agent of follicular conjunctivitis or superinfect eye epithelia previously damaged by *C. trachomatis*. Therefore, we tested baseline samples of swabs from the tarsal conjunctiva obtained from individuals living in a rural area of Rombo District, Tanzania, where overall 174 out of 956 individuals (18%) had active trachoma; among the latter, only 58 (33%) had *C. trachomatis* detected by NAAT.⁹ A sufficient quantity of sample was available for further testing for 847 participants, 155 of whom (18%) had active trachoma, including 55 (35%) with *C. trachomatis* detected by NAAT.

PCR detection of *M. genitalium* was attempted using the same primers as in our previous studies of patients with sexually transmitted infections and spiked with 8 copies of the *M. genitalium* internal control.^{6–8} Swabs of the right eye had been deposited in the Amplicor transport medium and processed as recommended for detection of *C. trachomatis* (Roche, Branchburg, NJ, USA). Extracted DNA had been stored at -80° C for six years until *M. genitalium* detection was attempted.

One specimen was inhibited. All 846 other specimens were negative for *M. genitalium* but positive for the internal control. To assess whether long-term storage had been detrimental for DNA, we retested samples that had been found to be *C. trachomatis* positive in the year 2000; 50 (91%) of them remained *C. trachomatis* positive, indicating that there had been little DNA degradation. Thus, in this remote community of Tanzania, we found no evidence that *M. genitalium* caused eye infections or contributed to the pathogenesis of trachoma.

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