We have reported increases in *M. genitalium* with macrolide and fluoroquinolone resistance in Japan [3, 10]. We created Table 1 by combining the prevalence of drug resistance–associated mutations in *M. genitalium* detected in 2014 with that collected from 2011 to 2013 [3]. Further increases of *M. genitalium* with drug resistance–associated mutations were observed. In particular, the prevalence of *M. genitalium* with both macrolide resistance– and fluoroquinolone resistance–associated mutations has risen from 0% before 2013 to 16.7% in 2013 and 30.8% in 2014, indicating that multidrug-resistant *M. genitalium* is increasing. Before strains with clinically significant high-level resistance to all existing antibiotics emerge, the development of promising new antibiotic regimens for *M. genitalium* infections is imperative.

**Note**  
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**Reply to Deguchi et al**

To the Editor—We thank Deguchi and colleagues for their interest in our meta-analysis and for providing an update on the alarming levels of antimicrobial resistance of *Mycoplasma genitalium* (MG) in Japan [1]. We are also seeing increasing levels of antimicrobial resistance in Australia, with 2 studies [2, 3] reporting an increase in ParC mutations and associated treatment failure following moxifloxacin. Pristinamycin has recently been evaluated as a possible agent for MG treatment based on evidence of susceptibility in vitro [2]; however, preliminary results suggest that it may need to be administered at a high total dose of 4 g per day for 10 days, with specific mutations in the 23S ribosomal RNA (rRNA) gene associated with failure at lower doses [4]. Recent evidence from both in vitro and clinic-based studies suggests that sitafloxacin may be an effective treatment for MG [5, 6]; however, as Deguchi and colleagues point out, its use is likely to be less effective in the future with the acquisition of additional fluoroquinolone-resistant mutations [1]. The use of 2-g extended-release azithromycin in Japan has been reported to have similar pharmacokinetics to 1.5 g of non-extended-release azithromycin given over 3 days [1]; however, treatment failures have been observed with both regimens [1, 7]. The development of new antimicrobials to treat MG (or other resistant bacteria, for that matter) is urgently needed, but the reality is that the pipeline of new drugs being developed is “virtually empty” [8]. We must use existing antimicrobials more wisely and within combination therapy approaches. We need serious debate about the use of 1 g of azithromycin as first-line treatment of non-gonococcal urethritis, as its widespread use has generated resistance in other sexually transmitted infections [9, 10]. The choice of treatment must be integrated within clinical algorithms that provide concurrent diagnostic-resistance assays that allow clinicians to prescribe the most appropriate first-line organism-specific therapy. A routine test of cure must be performed within 4 weeks of treatment to ensure that infection has been effectively
cured. Multidrug-resistant MG strains are rapidly becoming a reality, and a global coordinated effort is urgently needed to examine the efficacy of novel treatment regimens using currently available drugs.

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9. Cornell JL, Bannister H, Bernal C, et al. Detectable *Trypanosoma cruzi* parasitism during pregnancy and infections with higher parasitemia levels may account for the majority of congenital infection cases seen by Kaplninski et al [9].

They also observed lower levels of parasitemia, an increase in cardiac abnormalities, and a decreased risk of congenital transmission in the group with prolonged residence in an infested house. Hence, sustained domestic vector exposure is a surrogate for chronic Chagas infection.

To decrease congenital transmission, asymptomatic acute or subacute infections should be better recognized and treated. These patients often do not display epidemiological characteristics as clear as those observed by Kaplninski et al [9] in chronically infected women. Their findings call for better screening and treating for women of childbearing age in areas of moderate or high endemicity. These approaches not only may prove useful in reducing congenital transmission rates, but they may also have an impact on vector-associated transmission.

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