

# *Mycoplasma genitalium* antibiotic resistance in genital and extragenital samples from MSM attending a HIV/STI Clinic in Italy

Gelinda De Grandi<sup>1,2</sup>, Angela Sandri<sup>1</sup>, Maria Carelli<sup>1</sup>, Alessandro Visentin<sup>2</sup>, Alessia Savoldi<sup>2</sup>, Massimo Mirandola<sup>2</sup>, Giulia Maria Saitta<sup>1</sup>, Maria M. Lleo<sup>1</sup>, Caterina Signoretto<sup>1</sup>, Maddalena Cordioli<sup>2</sup>

<sup>1</sup> Microbiology Section, Department of Diagnostics and Public Health, University of Verona, Verona, Italy  
<sup>2</sup> Infectious Diseases Section, Department of Diagnostics and Public Health, University of Verona, Verona, Italy  
E-mail: gelinda.degrandi@univr.it

## Background

- Mycoplasma genitalium* (MG) is one of the most warning sexually transmitted pathogens also due to its ability in developing resistance to macrolide and quinolone antibiotics.
- Resistance-guided therapy has demonstrated the best cure rates but can only be based on molecular methods, and the gap between genotypic resistance and microbiological clearance has not been fully evaluated yet.
- This study aims at finding mutations associated with MG antibiotic resistance and investigating the relationship with microbiological clearance amongst men-who-have-sex-with-men (MSM).

## Methods

Samples collection from 1040 MSM: genital (urine), extragenital (pharyngeal and anorectal swabs)

DNA extraction and MG detection by Anyplex™ II STI-7 assay (Seegene): 107 MG-positive samples from 96/1040 MSM (9.2%)

47 samples from 42 MSM were available for detection of mutations associated with macrolide resistance (23S rRNA gene) and quinolone resistance (*gyrA* and *parC* genes) by Sanger sequencing and Allplex™ MG & AziR assay (Seegene)

## Results

- 63.8% (n=30/47) of samples showed mutations in 23S rRNA gene: A2059G (n=21), A2058T (n=7), A2058G (n=4). In 2 samples, both A2059G and A2058G mutations were detected.
- 17% (n=8/47) of samples had mutations in *parC* gene: 6 presented SNPs/AA changes of known clinical relevance (G248T/S83I, G248A/S83N, G259A/D87N, G259T/D87Y).
- 4.3% (n=2/47) of samples had mutations in *gyrA* gene: only one carried a SNP/AA change of known clinical relevance (G285C/M95I).
- 17% (n=8/47) of samples had dual class (macrolide and quinolone) resistance-associated mutations in 23S rRNA and *parC* genes (n=6) or in 23S rRNA and *gyrA* genes (n=2).
- After first-line treatment with macrolide azithromycin, 64.3% (n=27/42) of MSM returned for Test of Cure (ToC): in all those resulting MG-positive (n=15) a mutation in the 23S rRNA gene had been detected; among those resulting MG-negative (n=11), most (n=10) had no mutations in the 23S rRNA gene.
- All patients undergoing second-line treatment with quinolone moxifloxacin (n=13) resulted negative at ToC, even those carrying MG strains with mutations in *parC* gene (n=6).

## Conclusions

Our observations confirm that mutations in 23S rRNA gene are associated with azithromycin treatment failure while mutations in *parC* gene alone are not always associated with phenotypic resistance to moxifloxacin.

It is fundamental to test for mutations associated with azithromycin resistance to improve antibiotic prescription and reduce antibiotic pressure on MG strains.