

Conclusion *Neisseria gonorrhoeae* is able to rapidly acquire high level macrolide resistance in the presence of both DNA of AZM highly resistant NG strains and AZM.

Disclosure No significant relationships.

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CLINICALLY ISOLATED THIAMINE AUXOTROPHS OF *NEISSERIA GONORRHOEAE* INDICATE INCREASED SUSCEPTIBILITY TO HOST INNATE DEFENSES

Nelson Dozier*. *Uniformed Services University of the Health Sciences, Microbiology and Immunology, Bethesda, USA*

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Background Thiamine pyrophosphate (TPP) is an important metabolite that affects many metabolic pathways within the cell. Thiamine (thi) auxotrophs of *Neisseria gonorrhoeae* (Gc) that could not grow without TPP or other thiamine derivatives were isolated from patients in the 1970's, but the effect of thi auxotrophy on Gc pathogenesis is not known. We recently demonstrated that a genetically defined Gc mutant that cannot biosynthesize TPP is more susceptible to killing by neutrophils, cationic antimicrobial peptides (CAMPs), and reactive oxygen species (ROS) in vitro, and attenuated for experimental infection of mice that have a neutrophil response to infection. Here we investigated the susceptibility of five recently isolated TPP auxotrophs to paraquat, an inducer of ROS and CAMPs as a first step towards understanding the consequence of thi auxotrophy during human infection.

Methods Eighty-nine Gc isolates in the USUHS Gc Resistance and Reference Repository isolated between 2014 and 2017 were screened for the capacity to grow on medium without TPP. Auxotrophs were tested for susceptibility to paraquat and colistin (polymyxin E) using standard methods.

Results Five thi auxotrophs were identified among the 89 isolates tested (5.6%). Four of the auxotrophs exhibited increased susceptibility to paraquat and colistin compared to a wild-type Gc strain. Auxotroph 4097, in contrast, showed a ~2-fold greater resistance to 0.0195 mM of paraquat and colistin compared to a wild-type Gc strain, suggesting this isolate may carry a compensatory mutation(s).

Conclusion We conclude that clinically isolated thi auxotrophs are more susceptible to ROS and CAMPs. We hypothesize that these isolates may have a lesser ability to withstand oxygen-dependent and independent effectors of the host inflammatory response and that selection for compensatory mutations during infection may be one mechanism by which thi auxotrophs remain in circulation. Analysis of WGS data is underway to identify the genetic basis of thi auxotrophy and possible compensatory mutations.

Disclosure No significant relationships.

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INTERNATIONALLY DISSEMINATED CEFTRIAXONE-RESISTANT *NEISSERIA GONORRHOEAE* STRAIN FOUND IN CHINA

Shaochun Chen*, Yueping Yin. *National Center for STD Control, China CDC Institute of Dermatology Chinese Academy of Medical Sciences and Peking Union Medical College, Reference Lab, Nanjing, China*

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Background Ceftriaxone has been used to treat gonorrhoea in China for more than one decade, but an increasing level of decreased susceptibility or clinical resistance to ceftriaxone has been found. Moreover, the international spread ceftriaxone-resistant clones has been recognized as a threat to effective control of gonorrhoea. We now describe an imported ceftriaxone-resistant *N. gonorrhoeae* strain isolated in China, 2016.

Methods The isolate was collected in 2016. The antimicrobial susceptibility to ceftriaxone (CRO), cefixime (CFM), azithromycin (AZM), spectinomycin (SPT) and ciprofloxacin (CIP) was determined using the agar dilution method in reference lab at National Center for STD Control. A combination of molecular epidemiological methods including *N. gonorrhoeae* multiantigen sequence typing (NG-MAST), multi-locus sequence typing (MLST) and *N. gonorrhoeae* sequence typing for antimicrobial resistance (NG-STAR) was used to determine characteristics and resistant determinants of this isolate.

Results The strain was resistant to CRO (MIC 0.5 mg/L), CFM (MIC 1 mg/L), TET (4 mg/L) and CIP (≥ 32 mg/L), but susceptible to AZM (0.25 mg/L) and SPT (16 mg/L). The MLST type was ST1903, and NG-MAST type was ST3435. The NG-STAR type was ST233, which contains a type 60 mosaic penA allele, -35A Del in the mtrR promoter, G120K-A121D in PorB, L421P in PonA, S91F-D95A in GyrA, S87R in ParC, and wild-type 23srRNA.

Conclusion We identified a ceftriaxone-resistant *N. gonorrhoeae* strain which has sustainably transmitted worldwide for more than 3 years. The epidemiological and molecular typing data drew an integral transmission chain of this clone from Japan to China, and then disseminated globally. These findings indicate an imported risk of resistant clones in China and also call for an enhanced global gonococcal antimicrobial surveillance to track the emergence and dissemination of resistant strains for timely control the spread.

Disclosure No significant relationships.

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DISTRIBUTION OF ANTIMICROBIAL RESISTANCE IN *NEISSERIA GONORRHOEAE* – 5 YEARS OF GERMAN GONOCOCCAL RESISTANCE NETWORK (GORENET)

¹Susanne Buder, ¹Peter Kohl, ¹Eva Guhl, ¹Ingeborg Graeber, ²Thalea Tamminga, ³Sandra Dudareva, ⁴Dagmar Heuer, ⁵Viviane Bremer, ³Klaus Jansen*. ¹German Conciliar Laboratory for Gonococci, Department of Dermatology and Venerology, Vivantes Hospital, Berlin, Germany; ²Robert-Koch-Institute, Unit 34: HIV/AIDS, STI and Blood-borne Infections, Berlin, Germany; ³Robert Koch Institute, Infectious Disease Epidemiology, Berlin, Germany; ⁴Robert Koch Institute, Sexually Transmitted Bacterial Pathogens, Berlin, Germany; ⁵Robert Koch Institute, Berlin, Germany

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Background The widespread antimicrobial resistance (AMR) of *Neisseria gonorrhoeae* (NG) is a serious problem for the treatment of gonorrhoea. Because NG infections are not reportable in Germany, only limited data on disease epidemiology and antimicrobial susceptibility patterns are available. The Gonococcal Resistance Network (GORENET) monitors trends of NG AMR in Germany and links this to epidemiological data and NG multiantigen sequence typing (NG-MAST) data to guide treatment algorithms and target future prevention strategies.

Methods Between April 2014 and December 2018, NG isolates and data on patient-related information were collected from laboratories nationwide and centralized susceptibility testing using E-test was performed. Susceptibility results for