Embargoed until Tuesday, April 25th at 1:55pm ET

Date: Tuesday, April 25, 2017
Time: 1:55 – 2:15 PM
Concurrent Session G: Laboratory Leadership Service Concurrent
Room: 203 AB (3rd Floor)
Moderators: Conrad Quinn and Xin Liu

Title: Emergence of 23S Mutations Associated with Macrolide Resistance in Group B Streptococcus — Georgia, 2015

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Background: Group B *Streptococcus* (GBS) is a leading agent of newborn sepsis and a rising cause of - of severe infections in elderly and immunocompromised adults. Concurrently, resistance to first-line antibiotics for this pathogen (macrolides and clindamycin) is increasing. To characterize the GBS genetic diversity associated with this resistance, we investigated strains that showed high-level resistance to macrolides (>32) and clindamycin (≥8) by phenotypic testing but were negative for known determinants of resistance recognized by our Whole Genome Sequencing (WGS) pipeline.

Methods: In 2015, clinical isolates were obtained from 2257 invasive GBS cases. Antibiotic resistance was assayed using two simultaneous approaches: 1) phenotypic test via liquid-based Minimum Inhibitory Concentration and Etest and 2) genetic resistance determinants detection via our WGS pipeline. Isolates that exhibited antibiotic resistance but that were not identified by our WGS pipeline were characterized by manual genetic analyses.

Results: Out of 520 highly resistant isolates, we identified 3 phenotypically resistant strains without a pipeline-predicted resistance mechanism. Two contained a mutation in position 2062 of the 23S gene (A2062G). Modification at this methylation site blocks antibiotic binding, conferring resistance as previously reported in *S. pneumoniae* and other pathogens. Both strains were from the state of Georgia, and shared the same multilocus sequence type (ST8) and serotype (Ib), but they were not closely related phylogenetically. We have not identified a resistance mechanism for the third strain.

Conclusions: We report the emergence of a mutation in the 23S gene in two GBS strains, undetected by our current pipeline. To our knowledge, this mutation has not previously been detected in GBS, highlighting the value of joint phenotypic and WGS testing for timely detection of emerging resistance-associated mutations.