27th ECCMID Vienna, Austria 22 – 25 April 2017
The congress of ESCMID

Session: P011 Mechanisms of bacterial resistance

Category: 3d. Resistance mechanisms

22 April 2017, 15:30 - 16:30
P0221

Undetectable phenotypic VIM-1 production in an Atlantibacter hermannii clinical isolate

Delphine Girlich*[1], Remy Bonnin[1], Laurent Dortet[2], Thierry Naas[3]

[1]*Chu Bicetre, Cnr Resistance Aux Antibiotiques; Ea7361, Université Paris Sud; Service de Bacteriologie*

[2]*Imperial College London*

[3]*Hopital de Bicetre; Ea7361, Université Paris Sud; Service de Bacteriologie*

Background: We investigated the weak expression of VIM-1 in *Atlantibacter hermannii* Web-2 clinical isolate from a rectal swab from an hospitalized patient in the east of France and compared it to a previously identified VIM-1 producing *Enterobacter cloacae* from the same patient.

Material/methods: The MICs were determined by E-test, and the CARBA-NP and mass spectrometry (STAR-BL, Bruker) tests were performed as previously described to monitor carbapenemase activity. PCR assays followed by sequencing were carried out for detection and identification of carbapenemase genes. Whole-genome sequencing (WGS) was performed using MiSeq sequencer (Nextera XT, Illumina). *De novo* assembly was performed by CLC Genomics Workbench v7.0.4 (Qiagen, Les Ulis, France). The acquired antimicrobial resistance genes were identified using Resfinder server v2.1 and the genome was annotated using RAST server.

Results: *A. hermannii* Web-2 was resistant to all ß-lactams except carbapenems, with MICs values of imipenem, meropenem, and ertapenem of 0.5 mg/L, 0.19 mg/L and 0.19 mg/L respectively. It

produced an extended-spectrum ß-lactamase SHV-12 and the CARBA-NP test failed to identify any carbapenemase activity. As a VIM-1-producing *E. cloacae* Web-1 had been previously recovered from the same patient, this carbapenemase was thoroughly searched in the *A. hermannii* Web-2. The bla_{VIM-1} gene was PCR amplified and imipenem hydrolysis was detected by using the STAR-BL assay. The bla_{VIM-1} gene was located on a plasmid (ca. 150-kb) and bracketed by classical class 1 integrons features. In the *E. cloacae* Web-1 isolate, the bla_{VIM-1} gene was also integrin- and plasmid-located (ca. 200-kb). Both plasmids were of the same IncA/C incompatibility group. Plasmid extraction from both *E. cloacae* Web-1 and *A. hermannii* Web-2 isolates were electroporated in *E. coli* TOP10 and *E. cloacae* CIP 7933. Both recombinant strains expressed both plasmids with the same efficiency as assayed by CARBA-NP and RT-PCR experiments. The weak expression of the bla_{VIM-1} gene in *A. hermannii* Web-2 could then not be explained by a lack of transcription.

Conclusions: Further experiments will be necessary to understand this lack of expression in *A. hermannii* Web-2. Strains such as *A. hermannii* Web-2 expressing very weakly VIM-1 may explain screening results discrepancies between culture based methods (that remain negative due to lack of growth chromogenic selective plates) and molecular methods (that will be positive for VIM-1 DNA sequence).