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Abstract (poster session)

Characterisation of three new VIM enzymes, VIM-35, VIM-36 and VIM-37 identified in European countries: report of the SENTRY Antimicrobial Surveillance Programme

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Objective: To characterize three new VIM variants identified in P. aeruginosa (PSA) and K. oxytoca (KOX) recovered in Europe during 2011. VIM enzymes were the second type of acquired metallo-beta-lactamases identified and these enzymes are widespread in several European countries among Enterobacteriaceae and PSA isolates. Methods: Screening for carbapenemase-encoding genes was performed for carbapenem-non-susceptible Enterobacteriaceae and PSA strains collected during 2011 and submitted to the SENTRY Programme. All amplicons were sequenced. Genes encoding new VIM variants, VIM-1 and VIM-2 were cloned into PCRScript CamR(+) and transformants were susceptibility tested. Primer walking targeting integron (INT)-related structures and blaVIM were used to reveal the genetic environment of the new genes. Results: Three isolates from Poland (1 KOX and 1 PSA) and Belgium (1 PSA) carried new blaVIM variants. Isolates displayed elevated MICs against beta-lactams (including aztreonam), fluoroquinolones, amikacin and tobramycin. One PSA (Belgium) and the KOX were susceptible to gentamicin. The new VIM enzymes were named VIM-35, VIM-36 and VIM-37. VIM-36 was detected in the PSA from Belgium and displayed one amino acid (aa) alteration (Q59R) when compared to VIM-2. VIM-35 and VIM-37 were identified in KOX and PSA from Poland, respectively. VIM-35 displayed one aa alteration compared to VIM-1 (A235T; 99.6% homology) and VIM-37 showed 99.2% similarity with VIM-1, displaying two aa differences: A57S and S228R. Recombinant blaVIM plasmids carried in the same E. coli background showed that VIM-35 had similar beta-lactam resistance profile to VIM-1, whereas VIM-37 had much lower in vitro activity against cefepime and ceftazidime when compared to VIM-1 (MIC, 0.25 vs. 4 and 8 vs. >32 mg/L, respectively). VIM-36 had cefepime MIC values eight-fold lower than VIM-2, its closest ancestor. Integrons carrying blaVIM-35 and blaVIM-37 were identical and carried aacA(6')-1b, followed by blaVIM and the 3'-conserved sequence (CS). A duplication of 155-bp of blaVIM 3'-end was detected between this gene and the 3'CS. blaVIM-36 was embedded in an integron carrying aacA29 in the first position followed by this gene. Conclusions: We identified three new VIM variants that seem to have more limited activity against cefepime and/or ceftazidime when compared to ancestors. Furthermore, different genes carried in similar genetic structures were detected in the same hospital in two bacterial species.