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P1399 Prevalence of type VI and type III secretion system effectors in clinical isolates of Pseudomonas aeruginosa

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Background: *Pseudomonas aeruginosa* is able to secrete a wide range of proteins involved in virulence. It manipulates eukaryotic host cells using secreted effectors delivered by the type III or the type IV Secretion Systems (T3SS and T6SS). The T3SS allows the injection of bacterial effectors (Exo toxins) into eukaryotic cell. *P. aeruginosa*, encodes three T6SSs, H1-, H2- and H3-T6SS, of which the H1-T6SS is best characterized and is mainly involved in delivering toxins to kill bacterial competitors. Recently, two T6SS-secreted phospholipases D, PldA (H2-T6SS) and PldB (H3-T6SS), were identified as trans-kingdom virulence effectors, triggering both killing of bacterial competitors and internalization into non-phagocytic cells. Here, we decipher the prevalence of T3SS and T6SS effectors in *P. aeruginosa* isolates responsible for human infections and from environmental strains.

Materials/methods: *P. aeruginosa* clinical isolates responsible for sepsis (n=46), urinary tract infection (n=32), pulmonary infection (n=58), chronic infections in CF patient (n=49), 47 environmental strains and 33 multidrug resistant isolates were tested by PCR for the presence of T3SS effectors (*exoS*, *exoT*, *exoY* and *exoU*), H1-T6SS (*tse1*, *tse2*, and *tse3)*, H2-T6SS *(pldA)* and H3-T6SS (*pldB*) effectors Antimicrobial susceptibility testing was performed by disc diffusion method and interpreted according to EUCAST breakpoints. The prevalence of these effectors was also checked on 95 *P. aeruginosa* strain for which complete genomes have been deposited in NCBI database.

Results: The *pldA* prevalence was found to be low (18% to 43%) in all clinical isolates except those responsible for of pulmonary infection (50%). In addition, we found that the *pldA* prevalence increased with the resistance level to reach 100% in all carbapenemase producers of IMP-type. In contrast, the *pldB* prevalence was high (93-100%) in all isolates. Regarding T3SS effectors, *exoT* and *exoY* are present in nearly all isolates while *exoS* and exoU were found to be exclusive (only one of the two toxins is present) with and higher prevalence of *exoU* in strains responsible for pulmonary infections.

Conclusions: Our results suggest that *pldA* (H2-T6SS) might have a role during pulmonary infections and might have been co-selected in multidrug-resistant isolates, and particularly in IMP-producing isolates.