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Poster Session VI
Zoonotic antimicrobial resistance

EXTENDED VIRULENCE PROFILE OF ENTEROCOCCUS FAECIUM FROM SWINE (EUROPE/USA, 1995-2008)

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Objectives: Specific virulence factors (VFs) have been strongly associated with clinical *Enterococcus faecium* (Efm), but the prevalence of recently described cell wall-anchored surface proteins has been scarcely explored among animals. We aimed to determine the extended virulence profile of isolates from pig-related sources in different countries.

Methods: Representative Efm isolates (n=44) previously identified among a collection of multi-resistant strains from swine's faeces in Portugal, Denmark, Switzerland and the USA (n=18), and the animal setting in Portugal (different pig farms, n=26) were selected for this study. This collection includes isolates recovered during wide surveillance studies performed in these countries during 1995-2008, some of which are widespread among pigs of EU countries since the mid-1990s. Clonal relatedness was assessed by PFGE and MLST. Screening for 18 VFs included *esp*-Efm (enterococcal surface protein), *hyl*-Efm (hyaluronidase-like gene), IS16 (conferring genomic plasticity), and 15 predicted cell-wall anchored *E. faecium* surface proteins (Fms) with typical characteristics of MSCRACMM- (microbial surface components recognizing adhesive matrix molecules) and/or pilus-encoding genes (e.g. the collagen adhesins *acm* and *scm*, or the pilus cluster ebpABC-fm) was performed by PCR/colony hybridization. Efm strains Aus0004 and C68 were used as positive controls.

Results: Isolates clustered into CC17 (18%), CC5 (50%) or were identified as different singletons. The distribution of the 18 VFs was highly variable (0-95%), but a high number (≥7 VFs) was identified among all isolates. The *esp* gene (2%, only identified in a ST132-clone also found in Portuguese hospitals) and IS16 (9%) were confined to CC17 isolates, while *acm* was randomly distributed in different lineages except CC5 (45%). *hyl* and *fms18* were not identified. The occurrence of the remaining 13 MSCRACMM- genes ranged from 14% to 93%. The complete *ebpA*(23%)-*ebpB*(95%)-*ebpC*(93%) operon was found in 23% of isolates including all CC17. The *fms11*(14%)-*fms19*(30%)-*fms16*(30%) cluster was present in 14% of isolates, most of which belonged to CC17 (none of these genes was found in CC5). A few isolates (11%) from different lineages but CC5 carried the entire *fms14*(14%)-*fms17*(80%)-*fms13*(80%) cluster. *fms21*(91%) and *fms20*(43%) coexisted in 43% of isolates from all lineages. *scm* was predominant (93%) and *fms15* was found in 73% of isolates including all from CC5. Nine (*acm/ebpB/fms17/fms13/fms16/fms19/fms21*) and 4 (*ebpB/scm/fms15/fms21*) of the 18 genes were present in 100% of the CC17 and CC5 isolates, respectively. Complete gene clusters were not identified in CC5. The same virulence profile was established for similar PFGE types.

Conclusion: Our results demonstrate the considerable variability of VFs among Efm from swine with the lowest prevalence/diversity being observed in CC5. Complete gene clusters important in adherence/biofilm formation were predominant, but not exclusive of CC17. The exclusive association of specific VFs (esp/IS16) with swine CC17 reinforces the possibility of strain transmission between human and animal hosts.