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Commentary

Biofilm Formation in Clinical Isolates of *Enterococcus faecium*

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DESCRIPTION

Intestinal commensal *Enterococcus* is currently becoming a pathogen that is resistant to treatment. Different virulence factors are produced by it. Although the virulence factor *Enterococcus* Surface Protein (ESP) aids in adhesion, its function in biofilm development is still unclear. Additionally, a powerful biofilm producer shows multidrug resistance in many bacterial species. In order to understand the antimicrobial susceptibility pattern of *Enterococcus* spp. and to associate drug resistance with biofilm formation and the ESP gene, this study was conducted. Different clinical specimens were used to collect *enterococci* isolates. Biofilm formation was carried out using the microtiter plate method, and antibiotic susceptibility testing was carried out using disc diffusion. ESP gene detection was done using PCR. Vancomycin and High-Level Aminoglycoside Resistant (HLAR) *E. faecalis* strains were not biofilm producers and did not carry the ESP gene. However, the ESP gene was discovered to be present in additional biofilm-forming *E. faecium*, and this link was determined to be statistically significant (p=0.024). It was found that the ESP gene was not significantly associated with biofilm development in *E. faecalis*. Furthermore, neither *Enterococcus* species showed a statistically significant link between medication resistance and biofilm development. Therefore, in *Enterococcus* spp., biofilm development is not necessarily correlated with the presence or absence of the ESP gene and/or drug resistance.

The most contentious subgroup of Lactic Acid Bacteria (LAB) is *enterococci*. The most common enterococci species in human infections are *E. faecalis* and *E. faecium*, which also rank third in the frequency of multidrug-resistant nosocomial pathogen isolation from bacteremia. The Gelatinase (GelE) activity, Enterococcal Surface Protein (ESP), Aggregation factor (Agg), Hyaluronidase (Hyl), and Cytolysin (Cyl, -hemolysin) are among the virulence factors present in *enterococci*. In nosocomial infections, *enterococci's* fitness and persistence are influenced by their virulence factors. Particularly in particular environmental settings like the urinary tract and oral cavity, *enterococci* have an incredible potential to build biofilm that is highly resistant to antibiotics. However, it was discovered that the biofilm development was more closely linked to the adhesion qualities, a favourable probiotic trait, than to the virulence genes. Along with the well-known aggregators gelatinase and haemolysin, the suspected virulence factors of *enterococci* have mostly been focused on biofilm development and the existence of a gene coding for an ESP. Although other authors have shown that biofilm development may also occur without the presence of this protein, this study claimed that the ability of *Enterococcus faecalis* to generate biofilm was related to the presence of ESP. The majority of this research has only looked at the relationship between biofilm and ESP in isolates of *E. faecalis*. However, our team has also noted that *E. faecium* can create biofilms. The potential connection between biofilm and the variation ESPefm in this species has not yet been researched. Despite having discovered a two-component system, the fsr locus, that controlled the formation of biofilm through the regulation of gelatinase, whose expression has been demonstrated to be cell density-dependent, gelatinase or ESP were not associated with the production of biofilm in *E. faecalis*.

Silent gelE genes have been found in *E. faecalis* isolates from both food and clinical strains, with the latter showing a higher prevalence of silent genes. Due to this, we sought to confirm the relationship between gelatinase synthesis and biofilm development rather than the existence of the gelE gene.

The observations presented here support earlier findings that, regardless of their origin, *E. faecalis* isolates frequently possess the capacity to form biofilms. Only two isolates that colonised foreign objects and two endocarditis strains were recognised as nonbiofilm producers. Previous research has demonstrated that *E. faecalis* always has the genes bopD, locus epa, and icaA, which are currently thought to be possibly involved in biofilm production. Therefore, it's plausible that the rare strains that don't produce biofilms have genes that aren't functioning.

Biofilm generation was only found in 13 of the 45 strains of *E. faecium*, making it far less prevalent (28.8% vs. 95.2% in *E. faecalis*). The number is too little to undertake any statistical analysis when dividing the total number of isolates by the source

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of isolation. It is intriguing to observe that this feature was never present in foreign body colonisers, although 35.7% of infection isolates and 25% of environmental strains were able to generate biofilm. Such a finding might imply that the biofilm plays a different function in enterococcal infections than, say, staphylococcal foreign body infections.

Our findings support earlier research on ESP dissemination among isolates from various origins, demonstrating that the ESP gene is widESPread in *E. faecalis*, whether from infections or from a healthy environment and flora. However, some of them were found to share the same Sequence Type (ST-78, a Component of the Clonal Complex 17 [CC17]) with various *E. faecium* clinical strains isolated from other locations, both in our country and abroad. This suggests that ESPefm is almost entirely restricted to *E. faecium* from clinical samples.

In contrast to *E. faecium*, biofilm development has been observed to be a prevalent characteristic of *E. faecalis*. The association between ESP and biofilm in *E. faecium* isolated from infection compared to that from other sources may suggest a synergy between these factors as an advantage for the successful establishment of the infectious process, even though neither ESP nor gelatinase appear to be required for biofilm formation.