CYREX® Laboratories

CLINICAL SPECIFICATIONS

ENTEROCOCCUS FAECALIS

Function:

Enterococcus faecalis (E. faecalis) are Gram-positive anaerobic cocci. They are ubiquitous organisms present in dairy and fermented food products, natural environments (i.e. plants, soil and water bodies), and the gastrointestinal tract of humans and other mammals. It is a common hospital-acquired infection. *E. faecalis* can survive and persist in a broad range of environments, pH, temperature, hyper- and hypotonic conditions.

Associated With:

Crohn's disease[1]
Ulcerative colitis[1]

Known Cross-Reactions:

$A\beta_{42}$ peptide[2]

Clinical Significance:

E. faecalis has been associated with blood stream infections. Factors for greater risk include: older age, male gender, liver disease, renal impairment, diabetes, hematologic transplant, malignancy, and prior treatment with antibiotics.[reviewed in 3] It accounts for approximately 97% of all infective endocarditis cases, predominantly impacting the elderly and patients with comorbidities.[reviewed in 4] Additionally, *E. faecalis* is associated with urinary tract infections, bacteraemia, meningitis, wound infections and neonatal infections, and more recently, biofilm-associated infections of artificial medical devices have been attributed to enterococci.[reviewed in 5] *E. faecalis* can colonize the oral cavity, eye, gastrointestinal tract, heart, kidney, bladder, bone and surgical sites.[6] It has been proposed that *E. faecalis* plays a role in the pathogenesis of Alzheimer's disease (AD). Rats injected with *E. faecalis* showed neurodegeneration, which suggests that pathogenic epitopes present in early AD may be influenced by *E. faecalis* infection.[7] Soscia et al.[8] found that amyloid beta ($A\beta_{42}$) peptide, a hallmark of AD, functions as an antimicrobial peptide. The group demonstrated that $A\beta_{42}$ was present at much higher levels in the temporal lobe of AD patients compared to non-AD brains.[8] These findings suggest that increased levels of $A\beta_{42}$ in the AD brain could be due to an immune response against a pathogen. Furthermore, Vojdani et al. showed that anti- $A\beta_{42}$ antibody reacted strongly with *E. faecalis*. Production of antibodies against $A\beta_{42}$ and their cross-reactivity with *Enterococcus* may induce Aβ fibrillogenesis[2] when the blood-brain barrier is breached.

References:

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5. Anderson et al. *Enterococcus faecalis* from food, clinical specimens, and oral sites: prevalence of virulence factors in association with biofilm formation. Front Microbiol, 2016; 6:1534.
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