

The oral mucosa is continuously exposed to commensal and pathogenic bacteria [1]. Symptoms: – Symptoms can develop rapidly (acute bacterial endocarditis) or gradually (subacute bacterial endocarditis) – Prophylactic antibiotics may be recommended before certain dental or surgical procedure. It is increasingly recognized that in addition to causing specific oral diseases such as dental caries and periodontitis, oral dysbiosis is associated with inflammatory bowel diseases such as Crohn's disease and various immune disorders such as rheumatoid arthritis and diabetes mellitus [5]. This finding provides the proof of concept that bacterial characteristics may contribute to the occurrence of IE in patients with *S. aureus* bacteremia. Bacterial endocarditis, commonly referred to as infective endocarditis, is an infection of the inner lining of the heart chambers and valves (the endocardium). Recently, it has been highlighted that stability defects in the bacterial flora (dysbiosis) undermine the interaction between commensal bacteria and host immunity and consequently causes a variety of local and systemic diseases [4]. Knowledge of the interrelationship between oral commensal bacteria and oral mucosal immunity is considered essential for understanding the pathogenesis and pathophysiology of various systemic diseases. Previous researchers have analyzed neutrophils in the bone marrow and peripheral blood of germ-free (GF) mice that have never been exposed to bacteria after birth and found fewer neutrophils in comparison with normal convention mice [[8], [9], [10]]. *S. aureus* isolates causing community-acquired, definite native-valve IE (n = 72) and bacteremia (n = 54) were collected prospectively as part of a French multicenter cohort. However, the multivariate statistical tool DAPC, applied on microarray data, segregated IE and bacteremia isolates: IE isolates were correctly reassigned as such in 80.6% of the cases (C-statistic 0.83, P0.001). Periodontitis is inflammation of the oral mucosa caused by microbial dysbiosis that accelerates the differentiation of CD4+ T cells into IL-17-producing proinflammatory T cells (Th17) [12]. – Risk Factors: Individuals with pre-existing heart conditions (like congenital heart defects, prosthetic heart valves), those with a history of rheumatic fever, intravenous drug users, and those having certain medical procedures are at higher risk. Short-chain fatty acids produced by intestinal microflora are important for the maintenance of colonic epithelial cells and modulation of regulatory T cells [4]. Neutrophils, the most abundant innate immune cells, are known to be responsible for the termination of invading bacteria at the oral mucosal barrier. In this study, we compared germ-free (GF) mice with conventional (CNV) mice to clarify the role of oral commensal bacteria in relation to oral immune cells. In parallel, the genotypic profiles of all isolates, obtained by microarray, were analyzed by discriminant analysis of principal components (DAPC). (2). Infective endocarditis (IE) (1) is a severe condition complicating 10–25% of *Staphylococcus aureus* bacteremia. ). 2