S-02 : *In vitro* Dysbiotic Oral Biofilms Deregulate the Host Immune Response

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An imbalance in the periodontal microbiota and dysbiosis deregulate the host immune response, leading to chronic inflammation. Since little is known about the initiation of dysbiosis, it can be hypothesized that some commensal bacteria can suppress the outgrowth of pathobionts by H2O2 production. However, serum and blood components released during inflammation can neutralize this suppressive effect, leading to the initiation of dysbiosis. The aim of this study is to determine if the neutralizing effect of serum, hemoglobin and hemin on the inhibitory effect of the commensal bacteria on pathobiont growth is translated in a more pronounced immune response. Bacterial quantitative PCR, expression analysis of bacterial virulence and cellular inflammatory genes and cytokine quantification by ELISA were performed to quantify the pathobiont outgrowth and to detect possible differences in inflammatory response after exposure of cell cultures to homeostatic or dysbiotic biofilms. Peroxidases, serum and blood components neutralized the inhibitory effect of H2O2 by exogenous peroxidase activity, increasing the pathobiont outgrowth. Moreover, the addition of serum, peroxidase and blood compounds upregulated the main virulence genes of *P. gingivalis* and *P. intermedia* in multi-species biofilms. Exposure of epithelial and fibroblast cultures to these dysbiotic biofilms increased the expression of IL6, IL1β, TNFα and MMP8, but especially of IL8. Moreover, higher amounts of IL8 were produced after the challenge with dysbiotic biofilms. Conversely, homeostatic and commensal biofilms had a minor inflammatory response at expression and protein level. Overall, serum, peroxidases or blood compounds allowed the outgrowth of pathobionts and increased their virulence. Dysbiotic biofilms enriched in pathobionts and virulence factors significantly increased the inflammatory response compared to homeostatic and commensal biofilms.