GGM

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PERIODONTAL PATHOGENS IN BLOOD OF PATIENTS WITH AND WITHOUT CARDIOVASCULAR DISEASE

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It has been hypothesized that oral pathogen microorganisms can migrate from the mouth to the artery plaques, through the blood, exacerbating atherosclerosis. To compare the oral pathogen microorganisms present in peripheral blood of individuals with and without coronary artery disease. RNA sequences were downloaded from the GEO database (accession number: GSE58150) and were obtained from blood of 8 individuals with (cases=8) and without (controls=8) arterial calcification. The controls had a coronary artery calcium (CAC) score of cero and cases had a CAC>514. After quality controls, the sequences were aligned to the hg38 reference genome using Hisat2. The unmapped sequences were fed into Kraken to determinate bacterial *taxa*. The ecological indices were calculated using Vegan. The Shannon diversity index range from 3.8 to 4.8 in cases and from 3.3 to 4.7 in controls. The species richness was between 817.8 to 1414.7 in cases and between 313.9 to 826.0 in controls. The mean number of species in cases and controls was 1437 and 1297, respectively. The periodonto pathogens *P. gingivalis*, *T. forsythia* y *A. actinomycetemcomitans* were identified in cases and controls in similar quantity. Some microorganisms were found exclusively in cases or controls. This study identified oral microorganisms in blood of patients with and without coronary artery disease using RNA sequencing data from a public database instead of the traditional methods. This study has generated new knowledge to deep in the relationship between this cardiovascular disease and periodontitis.

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ANALYSIS OF GENE PATHWAYS REGULATED BY *HOXB2* GENE IN GLIOBLASTOMA

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HOX genes are a subgroup of the Homeobox family characterized by a high degree of conservation among eukaryotes. In mammals, there are 39 HOX genes distributed in four clusters: HOXA, HOXB, HOXC, and HOXD, located on chromosomes 2, 7, 17 and 12, respectively. HOX genes are transcription factors that act during embryonic development, regulating fundamental biological processes such as proliferation, differentiation, migration and angiogenesis. Recent studies have indicated a tissue-specific expression profile of HOX genes in different tumor types, suggesting an important role in tumorigenesis. Previous results carried out by our group demonstrated that 85% of the HOX genes are over expressed in glioblastoma (GBM), and that the high expression of *HOXB2* is correlated with low GBM survival. In this sense, our main objective is to evaluate the functional role of the HOXB2 gene in GBM, and for this, the following techniques have been used: Cell culture, Short-hairpin RNA gene silencing, RNA extraction, RT-qPCR gene expression analysis, *in vitro* functional assays (clonogenic, cell proliferation, apoptosis, senescence and cell cycle), analysis of HOXB2 gene targets by Chromatin Immuno precipitation Sequencing and transcriptome analysis by RNA-Seq. Up to now, our results have demonstrated that HOXB2 regulates proliferation, apoptosis, senescence and cell cycle, in two GBM cell lines. With the completion of next steps, our study will provide a robust characterization of the functional role of the HOXB2 gene in glioblastoma, through the identification of its targets.