Cyclooxygenase (COX) catalyses the conversion of arachidonic acid into prostanoids and related compounds which have been implicated in periodontal bone loss. Therefore, the aim of this study was to quantify COX-1 and COX-2 expression in gingival tissue derived from healthy/gingivitis and periodontitis sites. An immunoperoxidase technique was used to stain for COX-1 and COX-2 as well as CD4, CD8, CD14 and CD19 positive cells. The tissues in each group were each divided further into three subgroups according to the size of infiltrate (1+, small infiltrates; 2+, medium infiltrates; 3+, extensive infiltrates). The results showed that COX-1 and COX-2 expression were up-regulated with increasing inflammation. There was a significant increased expression of COX-1 in the 2+ periodontitis tissues in comparison with the 1+ periodontitis lesions (p<0.043). Similarly, COX-2 expression increased in the periodontitis 2+ lesions compared with the 1+ tissues (p<0.017) and there was a similar trend in the healthy/gingivitis tissues although the results were not significant (p<0.062). While there were no differences in COX-1 and COX-2 expression in each of the 1+, 2+ or 3+ groups in the healthy/gingivitis and periodontitis tissues, analysis of the percentages of COX-1 and COX-2 positive cells within each tissue section showed that there were higher numbers of infiltrating mononuclear cells which expressed COX-2 rather than COX-1 with increasing inflammation. COX-1 rather than COX-2 was expressed on keratinocytes and COX expression on these cells reduced in sections with larger infiltrates. Endothelial cells were generally negative for COX-1 with COX-2 cells located mainly in sections with small infiltrates. In conclusion the study has shown that COX-1 and COX-2 were both up-regulated on inflammatory mononuclear cells and that the percentage of COX-2 positive cells was increased in comparison with COX-1 with increasing inflammation.

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