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Healthy and Inflamed Gingival Fibroblasts' Role in Osteogenesis and Osteoclastogenesis

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Objectives Due to chronic inflammation in periodontitis, gingival fibroblasts (GFs) retrieved from inflamed sites have been exposed to bacterial products, activated leukocytes and pro-inflammatory cytokines and might be primed differently to modulate bone biology compared to GFs from healthy sites.
Aim: to explore the biological characteristics of GFs obtained from biopsies from inflamed and non-inflamed (i.e. healthy) periodontal tissues from periodontitis patients.
Methods After 21 days of co-culture of GFs with leukocytes, osteoclasts were counted, inflammatory cytokines were measured and osteoclastogenesis and osteogenesis-related gene expression was quantified.
Results GFs from inflamed and healthy tissues exhibited a similar osteoclast inducing capacity, similar levels of TRAcP enzyme activity and secretion of the pro-inflammatory cytokines TNF-α and IL-1β. Expression of osteoclastogenesis-related genes (IL-1β, TNF-α, RANKL and OPG) as well as the osteogenesis-related parameter alkaline phosphatase (ALP) activity did not differ between the two groups. Notably, the cellular responses displayed a time-dependency for cytokines and their gene expression; TNF-α reached its peak on day 6 but decreased by day 14, while TRAcP enzyme continued to be highly expressed until day 21. Gene expression of TRAcP, TNF-α, and RANKL progressively increased over time, while the expression of OPG decreased.
Conclusions Both inflamed and healthy GFs show an early response of inflammatory markers during osteoclast formation (as disclosed by the increased expression of TNF-α and TRAcP on day 6). There were no differences in the capacities of GFs from inflamed and healthy sites from periodontitis patients to modulate osteoclastogenesis and osteogenesis. This suggests that the in vitro cultured GFs have intrinsic capacities irrespective of inflammatory state and do not seem specifically primed by the in vivo inflammation. Further research is needed to investigate whether GFs from non-periodontitis patients have differential activities regarding osteoclastogenesis and osteogenesis than GFs from periodontitis patients.