RESIDENT MEMORY T CELLS IN PERIODONTAL TISSUES: A POSSIBLE EXPLANATION FOR PERIODONTAL DISEASE SUSCEPTIBILITY AND RECURRENCE

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Introduction:

Recent studies demonstrated that the majority of T-cells remain as memory resident cells (TRM) within epithelial barrier tissues affected by chronic infection. TRM cells provide a first response against infections reencountered at body surfaces, where they accelerate pathogen clearance.
As gingival tissues share some characteristics with other barrier tissues constantly exposed to microflora, TRM cells may promote accelerated and higher inflammatory response accounting for larger and faster tissue damage in periodontal patients.

Aims:

Therefore, the aim of this study was to investigate the presence and distribution of TRM cells in periodontal tissues in relation to the periodontal damage and bacterial exposure.

Methods:

Immunohistochemical detection of CD103 was performed in 34 patients: 17 chronic periodontitis patients (test) requiring osseous resective surgery (ORS) and 17 healthy patients (control) with mucogingival problems requiring free gingival graft or laterally sliding flap procedure. In the test group 17 tissue samples were harvested from the secondary flap removed during ORS and in the control group 17 tissue samples were harvested each from the primary flap and from the palatal graft during mucogingival surgery. Intraepithelial and stromal CD103-positive cells per high power field were counted in areas with highest expression.

Results:

CD103 expression was significantly enhanced in the epithelial and connective tissue from patients with periodontitis compared to healthy controls ($p < 0.05$). In the control group epithelial and stromal CD103 expression was absent in 2 and 4 palatal mucosa tissue specimens, respectively, and in the connective tissue from 6 mucogingival primary flaps.

Conclusions:

The role of TRM cells in periodontal sites is still unclear. It can be hypothesized that the excision of gingival tissue harboring these cells could modulate the host immune response to plaque accumulation, leading to a slower and milder reactivation of periodontal disease.