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CLAST CELL ACTIVITY IN A MODEL OF ASEPTIC ROOT RESORPTION

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ABSTRACT

Bone resorption by osteoclasts facilitates orthodontic tooth movement. However, an adverse sequela of tooth movement is the possibility of inflammatory root resorption. In order to examine aseptic hard tissue resorption an investigative rat model was developed using *in situ* freezing of the upper right molar by the application of solid carbon dioxide to the tooth crown. Variations in duration and frequency of application permitted the investigation of the cellular reactions responsible for the progression and repair of resorption and periodontal healing using tartrate-resistant acid phosphatase and succinic dehydrogenase enzyme histochemistry and a concert of antibodies directed at specific osteoclast antigens in control and osteoprotegerin-affected animals. In addition, an electron microscopic investigation was undertaken to assess variations in the morphological features of the resorptive cells in the experimental model systems.

The development of the model revealed a pattern of root resorption dissimilar to previously reported patterns caused by orthodontic means. Hard tissue resorption was generated by short-term freezing of molar crowns while ankylosis was a feature of an intense, prolonged insult as well as in all osteoprotegerin-affected rats. TRAP activity was found to be to be a useful clast cell marker but not in areas of ankylosis where ED1 (a macrophage lysosomal marker) proved to be more valuable. ED1 label indicated that macrophages or, at least, clast cell precursors are more involved in hard tissue resorption than previously considered, suggesting that odontoclasts are likely derived from the macrophage lineage of clast cell differentiation.

While osteoprotegerin inhibited osteoclastogenesis and resorptive activity in the skeletal bones and periodontium of unfrozen control molars in rats, it did not appear to inhibit ultrastructural signs of resorption in the frozen rat molars. In addition, TRAP activity was present but diminished while ED1 activity was still evident in the resorptive areas. This study concluded that osteoclasts and odontoclasts, while appearing to be similar cells, are possibly not the only cells responsible for hard tissue resorption. Furthermore, the observation that osteoprotegerin failed to prevent inflammatory resorption in the frozen tooth model suggests that there are possibly other extraclastic mechanisms or factors in this experimental model that influence resorption. Hence, the usefulness of OPG as a potential inhibitor of hard tissue resorption and, in particular, orthodontic root resorption remains to be established.