OP21 EFFECTS OF THE PARTIALLY COX-2-SELECTIVE NON-STEROIDAL ANTI-INFLAMMATORY DRUG MELOXICAM DURING TOOTH MOVEMENT IN THE ABSENCE/PRESENCE OF PERIODONTITIS

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AIM: Orthodontists are increasingly confronted with patients with chronic periodontitis or who regularly take non-steroidal anti-inflammatory drugs (NSAID) to alleviate pain, including orthodontic pain. Whereas the effects of unselective NSAID and coxibes during orthodontic therapy have been studied, the consequences of a partially selective inhibition of cyclooxygenase 2 (COX-2) over COX-1 (11:1), as induced by the NSAID meloxicam, remain unclear. Thus the adverse side and beneficial effects of meloxicam in orthodontic therapy and whether meloxicam may be useful for preventing periodontal bone loss associated with recurring active inflammation during tooth movement in periodontally challenged individuals was investigated.

MATERIALS AND METHOD: One hundred and five male inbred Fischer344-rats were randomly assigned to five experimental groups: (1) control, (2) orthodontic tooth movement (OTM) of the upper left first/second molars (NiTi coil-spring; 0.25 N), (3) OTM with 3 mg/kg meloxicam p.o., (4) OTM with experimental periodontitis (cervical silk ligature), (5) OTM with periodontitis and meloxicam. After 14 (28) days of OTM, periodontal bone loss and OTM as well as C-reactive-protein serum concentration, gastric toxicity, orthodontically-induced inflammatory root resorption (OIIRR), osteoclast activity and dental-periodontal gene expression of known inflammatory/osteoclast markers was determined. *In vitro*, human periodontal ligament fibroblasts (hPDL) were stimulated with orthodontic pressure (2 g/cm^2) and combined with toxins of Aggregatibacter actinomycetemcomitans with/without meloxicam (10 µmol) and gene/protein expression of inflammatory/osteoclast markers as well as osteoclastogenesis in co-culture with RAW264.7 cells were determined.

RESULTS: Meloxicam significantly reduced tooth movement velocity and OIIRR by 40-50 per cent, osteoclast activity and relative expression of inflammatory/osteoclast marker genes within the dental-periodontal tissue, while presenting a good gastric tolerance profile. *In vitro*, a corresponding significant decrease of PG-E_2/IL-6/RANKL(-OPG) expression and hPDL-mediated osteoclastogenesis was observed. Meloxicam prevented the orthodontically triggered exponentiation of periodontal bone loss during acute periodontitis as well as *in vivo*/*vitro* the periodontitis-induced exponentiation of tooth movement velocity, OIIRR and the associated pseudo-inflammation/osteoclastogenesis.

CONCLUSIONS: Meloxicam seems to downregulate hPDL-mediated inflammation, RANKL-induced osteoclastogenesis and consequently tooth movement velocity, limiting its suitability for orthodontic analgesia. Since tooth movement is not completely inhibited, but only decelerated, its gastric tolerability and protective effects regarding OIIRR and periodontal bone loss (in acute phases of chronic periodontitis) suggest the possibility of a future prophylactic application.