

# Bisphosphonate Therapy of Osteoporosis Predisposes Epithelial Breakdown in Osteonecrosis of the Jaws, with Calcium Ion Constraint

Louis ZG Touyz\*

Department of Periodontics, Faculty of Dentistry, McGill University, Canada

Osteoporosis is a common bone disease prevalent in aging in humans deriving from all ethnic backgrounds. Bisphosphonates (BPs) influence the metabolism of soft tissues, including direct toxicity on oral mucosae. This contributes to mucosal ulceration and exposure of underlying bone [1]

The ulcerative necrosis is prevalent on sites when thin mucosa covers bony protuberances, prominences or exostoses. BONJ cases are often preceded by dento-alveolar therapy. Dental treatment procedures, like deep root-planing, extractions and surgical placement of osseointegrated implants are acknowledged as predisposing practices which precipitate BONJ. The increased demand for calcium ions necessary for bone regeneration and repair, exceeds the dysfunctional osseous reserve, and results in epithelial break down and localized osseous necrosis.

Oral biofilm is highly infectious, and without intact epithelium and resilient bone, BONJ develops. Consequently because oral surgical procedures are central in developing BONJ, not only should all dentists must be aware of this and be ultra-vigilant about all patients being treated with bisphosphonates, but all dental treatments should be concluded before starting the bisphosphonate therapy. When the normally very low (10<sup>-8</sup> M) intra-cellular calcium levels rise, the gap junctions undergo conformation changes that close the gap junctions channel, inhibiting transfer of other metabolites [2].

Increase of intra-cellular calcium forces closure of gap junctions, and subsequent disruption of flow of vitally needed cellular metabolites for normal cell function, growth and differentiation. Gap junctions, so necessary for cell adhesion and organ integrity in mucosae, are constituted mainly by proteins (desmocollins, and desmogleins) but cannot develop because of lack of essential supply of nutrients and consequently the epithelium breaks down. BP's interfere with the supply of Ca<sup>2+</sup>, the formation of inter-cellular connections and consequently the potential mucosal healing. Ca<sup>2+</sup> is retained intra-cellularly and obstructed from normal flow to where it is used for epithelial cell functioning, inter-cellular connection, desmosome and gap junction development [3].

## References

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**\*Corresponding author:** Louis ZG Touyz, Department of Periodontics, McGill University, Faculty of Dentistry, McGill University, Canada, Tel: 514-398-7203; E-mail: [louis.touyz@mcgill.ca](mailto:louis.touyz@mcgill.ca)

**Received** March 23, 2015; **Accepted** April 22, 2015; **Published** April 29, 2015

**Citation:** Touyz LZG (2015) Bisphosphonate Therapy of Osteoporosis Predisposes Epithelial Breakdown in Osteonecrosis of the Jaws, with Calcium Ion Constraint. *J Osteopor Phys Act* 3: 135. doi:[10.4172/2329-9509.1000135](https://doi.org/10.4172/2329-9509.1000135)

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