INSIGHT INTO THE ROLE OF PERIODONTAL LIGAMENT ASSOCIATED PROTEIN-1/ASPORIN IN THE MAINTENANCE OF THE PERIODONTAL LIGAMENT USING A RAT ANKYLOSIS MODEL

Doctor of Clinical Dentistry (Orthodontics)
Thesis

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Glossary of Abbreviated Items

General

ABC	avidin-biotin complex
BMP	bone morphogenetic protein
B-SA	biotin-streptavidin
Cbfa-1	Core binding factor a1
EDTA	ethylenediaminetetraacetic acid
EGF	epithelial growth factor
FGF	fibroblast growth factor
HEBP	1-hydroxyethylidene-1, 1-bisphosphonate
IGF	insulin like growth factor
IL	interleukin
IMVS	Institute of Medical & Veterinary Science
KV	kilovolts
LRR	leucine rich repeats
LTB_4	leukotriene B_4
mRNA	messenger ribonucleic acid
PAP	peroxidase anti-peroxidase
PBS	phosphate buffered saline
PDGF	platelet derived growth factor
PDL	periodontal ligament
PG	prostaglandin
PLAP-1	periodontal ligament associated protein-1
PTH	parathyroid hormone
PTHrP	parathyroid hormone related protein
Runx2	Runt-related transcription factor-2
TGF	transforming growth factor
TNF	tumour necrosis factor
TNFR	tumour necrosis factor receptor

Measure of Length

mm	millimetre
µm	micrometre

Measure of Volume

ml	millilitre

Measure of Weight

mg	milligram
g	gram
kg	kilogram
mw	molecular weight

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SUMMARY

The cells of the periodontal ligament have been shown to be osteogenic but under normal conditions, the PDL space remains patent without the occurrence of ankylosis. Periodontal Ligament Associated Protein-1 (PLAP-1)/Asporin is a recently discovered protein that has been suggested to play a significant role in suppressing the osteogenic tendency of the periodontal ligament and maintaining the fibrous ligamentous nature of the periodontal ligament. Furthermore, PLAP-1/Asporin has also been shown to be associated with the differentiation and mineralisation of dental pulp stem cells.

In this study, the expression of PLAP-1 was investigated using a reversible ankylosis model induced by hypothermal insult. In paper 1, the principal aim was to determine the normal distribution of PLAP-1 reactivity in a normal rat maxilla and to analyse the pattern of PLAP-1 reactivity in association with the formation of ankylosis. In addition, another study (paper 2) was performed with the aim of investigating the distribution pattern of PLAP-1 within a normal rat molar pulp as well as its changes following freezing trauma.

The results from the first paper showed that PLAP-1 was expressed in the PDL, dental pulp, blood vessel walls and the nasal cartilage. Not all sections obtained ankylosis. Sections which did not obtain ankylosis demonstrated no significant PLAP-1 expression differences between control and experimental sides. Sections that did obtain ankylosis yielded a tendency towards increased PLAP-1 reactivity especially near the cementum. However, it was difficult to deduce whether the relationship of PLAP-1 to the ankylotic union was associated with bone formation or resorptive activities.

The results from paper two showed that PLAP-1/Asporin was expressed exclusively within the pulp under normal conditions and appeared to be associated with the odontoblastic and cell rich zone. Following trauma, PLAP-1/Asporin expression

decreased marginally (not statistically significant) alongside the dentine but increased significantly in the central pulpal region along with disruption and breakdown of the cellular structures.

From the results derived, it can be concluded that PLAP-1/Asporin is indeed expressed in several tissue/cell types and regions including the dental pulp and is not exclusively associated with the periodontal ligament. In addition, PLAP-1 appears to have a direct association with ankylosis although it is uncertain whether PLAP-1 aids in bone mineralisation or resorption. The second null hypothesis was also rejected although the change in expression of PLAP-1 within the pulp is more morphological than physiological. Results from the study also suggest that PLAP-1/Asporin does not appear to play a direct role in the formation of the tertiary dentine.

Further research is required to elucidate the true role of PLAP-1 within the periodontal ligament as well as the pulp. Additional investigations are also required to gain further insight into the maintenance of the periodontal ligament.